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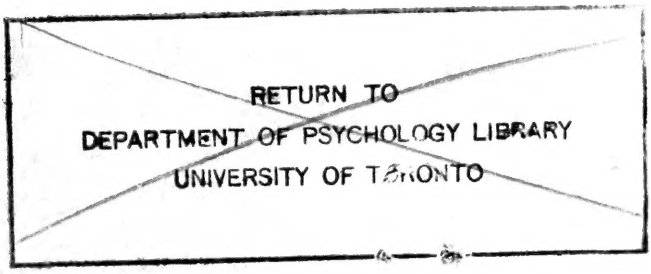

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STUDIES OF CEREBRAL FUNCTION IN LEARNING

VII. THE RELATION BETWEEN CEREBRAL MASS, LEARNING, AND RETENTION

K. S. LASHLEY
Department of Psychology of the University of Minnesota

SEVEN TEXT FIGURES AND EIGHT PLATES

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INTRODUCTION

Indications of a correlation between complexity of behavior and the quantity of functional nervous tissue have been given by various lines of evidence: comparative anatomy, cerebral pathology, and physiological experiments have each suggested such a relationship, but have provided no clue as to the manner in which cerebral mass contributes to complexity of function. The phylogenetic series offers the clearest evidence for the importance of neural mass in determining intelligence, yet even this is not unequivocal. It is difficult to determine how much of the neural tissue is concerned with purely vegetative or simple sensory-motor functions, increasing in weight with the bulk of the muscles and the surface area of the sense organs, and how much represents

a real advance in functional complexity. The work of Dubois and of Lapicque ('23) has done much to clear up this problem by defining the ratio of brain to body weights and Dubois' coefficient of cephalization presents a possible means of accurately correlating cerebral development with intelligence. But the lack of any quantitative determination of differences in the complexity of behavior of different species of animals makes it impossible as yet to evaluate this coefficient. The work of Szymanski ('12), Turner ('13), and von Frisch ('14) on learning[1] in insects shows them little inferior to lower mammals, and among mammals we cannot say that a relatively higher brain weight is a certain indication of superior intelligence, since we have no sure measure of the latter.

Within the single species the correlation is still less certain. Basset ('14) found that a strain of rats of less than average brain weight was inferior in learning ability to a normal strain, but his data, as Paterson ('17) has pointed out, are not statistically valid. Crude comparisons of the brain weights of superior, normal, and criminal men show the former groups as having slightly higher weights (Donaldson, '03), but are of doubtful significance because of the uncertainty of the evidence for a real difference in the complexity of the behavior of the groups (Goring, '13; Fernald, Hayes, and Dawley, '20).

For ganglion cell number, as indicated by surface area of the cerebrum or degree of fissurization, the relationship is even less clear. The primates show a relatively greater surface area than lower mammals, and the anthropoids the greatest surface area of the primates, but, as Monakow ('14) has pointed out, many of the ungulates show much greater fissurization of the cortex than carnivora with probably much higher intelligence, so that the exact significance of surface area

[1] The relation between learning ability and intelligence is by no means established, since we cannot define either function except in terms of specific instances. Our measures of learning in animals require both the solving of problems and retention of the solutions, and I use the term learning here in the broader sense as including at least some phases of intelligent behavior.

remains in doubt. It is probably, as Lapicque ('23) suggests, an adaptation for nutrition of the cortex and as such should depend more upon the absolute size of the brain than upon its complexity of organization.

More certain evidence of the significance of ganglion cell number appears in the cyto-architectural studies of Bolton ('14) and Southard ('14). They have found a general reduction in the number of functional ganglion cells in amentia and dementia, but the limit to which cell degeneration can occur without mental deterioration is not determined, nor is it certain that the symptoms in these disorders are due to the reduction in cell number, and not to invisible changes in the remaining functional cells.

Students of gross injuries to the cerebrum have been so occupied with problems of cerebral localization that the possibility of quantitative relationships has been largely overlooked. The theory of localization of faculties has worked fairly well for sensory projection areas and motor areas, but the clinical literature contains many so-called negative cases which fail to conform to any schema of localization. In particular the association areas have given difficulties. Reported effects of lesions in them are contradictory and in many cases questionable. Much of the difficulty here may be due to failure to take into consideration the extent of the lesions as well as their locus. Bianchi ('22) holds that marked deterioration occurs only after very extensive lesions in the frontal areas, and Monakow ('14), in discussion of aphasia, points out that severe and permanent symptoms occur only after extensive or diffuse lesions. Beyond the suggestion of this critical amount of injury for the production of severe symptoms, the clinical literature gives no consideration to the problem of cerebral mass.

Taken altogether, these lines of investigation point to a relationship between neural mass and complexity of behavior, but there is little suggestion of the reason for this relationship or of the real degree of correspondence. Attempts to localize intelligence in any particular part of the brain, as in

the frontal lobes,[2] have failed, and we can only say that the complexities of behavior which we term intelligence are somehow a function of the activities of the entire cerebrum. It is yet an open question as to whether the more complex types of behavior result from a mere multiplication of reflex and conditioned reflex paths or involve some fundamentally different mechanism from those with which the study of spinal reflexes has made us familiar. In many respects the reflex theory seems inadequate to account for the larger number of human and animal reactions, yet our knowledge of neural functions is as yet so slight as to make the formulation of any plausible alternative hypothesis impossible.

Many phenomena of behavior seem explicable only in terms of a dynamic function of the central nervous system for which the conceptions both of the conditioned reflex and of a mosaic arrangement of faculties are inadequate. Reactions to ratios between stimuli, to spacial and temporal relationships—in fact, most of the adaptive behavior of the organism—demand a mechanism in which dynamic as well as integrating functions may be exercised. Much of the recent direct evidence on cerebral mechanisms also points to a functional unity rather than a mosaic arrangement in activities within the larger functional divisions so that the problem of mass relationships in neural function takes on a more important aspect than is implied in either the theory of conditioned reflex arcs or in that of localized faculties.

It has seemed important, therefore, to carry through a series of experiments designed to test the relative significance of functional localization and of the factor of total mass in the performance of various types of activity. In an earlier

[2] Bianchi ('22) has claimed that the learning function is restricted to the frontal lobes in primates and has reported experimental evidence in support of his view. Mr. Carlyle Jacobsen and the writer are now repeating his experiments on rhesus monkeys with the addition of quantitative measures of the rate of learning. We have been unable to verify his findings on a single point. In fact, removal of the entire frontal and parietal association areas has not resulted in any measurable retardation in the rate of formation of complex sensory or motor habits.

study (Lashley, '20) I tested the effects of lesions ranging from 15 to 50 per cent of the total surface of the rat's cortex upon the rate of formation of a motor habit. Within these limits it was found that no lesion, of whatever location or extent, had any effect upon the rate of learning, although there was indication that lesions more extensive than those studied systematically (more than 50 per cent) did produce a marked retardation. The result was based upon a limited number of cases and required verification and extension to other types of learning. I have therefore undertaken a program which includes: 1) a study of the effects of very extensive lesions (more than 50 per cent) of the cerebrum upon the learning and retention of various types of habits in the rat; 2) measurement of the rate of formation and the retention of localized and non-localized habits after lesions of lesser extent and of various loci; 3) determination of the effects of lesions of various extents upon the function of a sensory projection area; 4) a test of the validity of the findings in the rat for the monkey and other higher forms; 5) a review of the clinical literature to determine the relative importance of locus and extent of lesion for the severity and duration of symptoms in man.

The present paper is a report of experiments upon the third of these problems: the influence of the extent of lesions within the visual area of the rat upon the formation and retention of habits of brightness discrimination. Some justification of this choice of material is perhaps necessary. Objections are obvious; the rat is low in the evolutionary scale and conclusions based upon it are not necessarily applicable to higher forms; the visual area shows a subordinate spacial arrangement within the total area and the production of scotoma may lead to ambiguous results; the habit of brightness discrimination is a simple one and perhaps does not involve any mechanisms comparable to those which function in the adaptive behavior of higher forms. The importance of these objections is admitted, but for a preliminary experiment the material has definite advantages. A large number

of animals is required (more than 150 have been trained and operated to obtain the present series) for a quantitative study, and for this reason rats are the best available material. The visual habit is the only one which has been localized with any certainty in the cerebral cortex of the rat, so that there was, in fact, little choice in the matter. The primitive character of vision in the rat (Lashley, '12) makes it impossible to generalize from it to man, but, on the other hand, the very simplicity of the function makes it more suitable for this work, since it eliminates in part the problem of subordinate localization within the visual field (v.i.).

Studies of neural function in lower mammals constitute at best only a preliminary survey, suggesting and defining problems and methods which may later serve as a starting-point for work with higher forms, rather than giving laws of universal applicability. We must always question whether principles of neural action derived from studies of the rat will hold true for primates and can answer only by repeating the experiments with the higher forms. Nevertheless, the agreement thus far obtained when comparable experiments have been performed (Lashley and Franz, '17; Franz, '07; Lashley, '21, '24 a) suggests a very close similarity between the higher and lower mammals in all fundamental mechanisms. Differences seem to be matters of relative differentiation of functions rather than radical changes in mechanism.

EXPERIMENTAL METHODS

Training

The rats were trained in a Yerkes' discrimination box to enter a compartment illuminated directly with a frosted 6-watt miniature lamp and to avoid a darkened compartment. In previous studies I have used this box only for qualitative work, to detect the presence or absence of the habit of reacting to brightness. In the present study quantitative results are sought; measures of the amount of practice necessary to establish the habit and of the amount of loss subsequent to

operation. The reliability of the method for quantitative work must therefore be considered.

The discrimination habit provides only two criteria of learning, the number of trials necessary to reach some standard of achievement and the number of errors made during this number of trials. Two methods of estimating the reliability of a measure have been employed. Hunter and Heron ('23) have computed the correlation in the scores of individual animals at different stages during training on the assumption that individual differences in learning ability will constantly influence the scores at different stages of learning. This method is inapplicable in the present case, since the discrimination method gives data only at the completion of learning. A second method employed by these writers compares the learning records of the same animals on different problems and determines the reliability of a method by the consistency of its results with those obtained by other methods. This is laborious and not altogether satisfactory, since we have as yet no certainty that individual differences in learning ability are constant for different types of problems. A third method of estimating reliability, fully as valuable as these, may be applied here. If individual variations in learning, as determined by a learning test, are found to correlate highly with any other variable which itself is not a function of the learning test, this correlation is evidence for the reliability of the test. A negative result is, of course, meaningless by this criterion, but a constant positive correlation, if statistically valid, is convincing evidence of the reliability of the method. Several quantitative studies have been made with the discrimination apparatus. That of Dodson ('17) is the most extensive. It is internally consistent and agrees in general with the results of other writers (Yerkes, Hogue and Stocking, and Cole),[3] so that it justifies the use of the measure, although it gives no indication of its fineness.

[3] For references see Dodson, '17.

A more certain test of the reliability of the discrimination method is offered in the present study. The animals of group B show a high correlation between retention tests and the extent of brain injury. The latter variable is in no way modified by the training method so that we must conclude that the method really does measure with some accuracy a function of the brain injury.

The animals were trained with punishment in the non-illuminated compartment and food in the illuminated one. Ten trials per day were given, the animals being allowed to reach the food in every trial. Training was continued until ten successive errorless trials were obtained on each of three successive days—thirty consecutive errorless trials in all. These thirty consecutive errorless trials constitute practically perfect learning, since errors are rarely made during continuous training after such a record has been established. Animals given 1200 trials overtraining after this standard was reached averaged only seven errors in the entire period of overtraining (Lashley, '21).

The same method was used in the retention tests, which were thus essentially retraining tests and resembled the 'savings' method rather than the 'Treffer' method in human studies. Any trial in which the animal entered the darkened alley of the apparatus one or more times was counted as one error. Of the two criteria, total trials required for learning and total errors made during practice, the latter is probably the more reliable, since one error due to chance distraction made after twenty-nine errorless trials may increase the total number of trials out of all proportion to the seriousness of the mistake or the increase in total errors. The two criteria correlate highly ($p = 0.820$ for group A), so that it probably makes little difference which is employed.

Where lesions of different sizes were compared in the same group, the experiment was arranged so that both animals with slight and extensive lesions were trained on the same days and with identical methods. Similarly, the training of animals from both groups A and B (v.i.) was carried on at the

same time to eliminate seasonal variations and progressive changes in the experimenter's technique.

Since I knew the extent of the lesions roughly from the character of the operations, there is a chance that preconceived theories might modify the training of particular animals, even when the point where such a personal equation might enter could not be detected. The only control which I can offer for this is the observation that in five cases where I greatly misjudged the extent of the operation the behavior of the animals conformed to the actual extent of the lesion as determined at necropsy and not to my earlier expectations.

In the series of cases reported some are included which were trained in earlier experiments (Lashley, '20, '21 a, '22). These are marked with an asterisk in the tables. The initial training of cases 50, 53, 54, 55, 56, 65, 66, 87, 88, 94, 109, 110, 111, and 112 was done by Miss Dorothy Hunter and Mr. L. E. Wiley. I have computed constants both including and excluding the cases from these sources. Their exclusion does not significantly change the results, except to increase the probable errors.

The possibility of reaction to other than visual cues must be considered. To determine the effective stimulus the following procedures and criteria of visual reaction were used after completion of training or retention tests:

1. Trials with both lamps extinguished. Failure to advance beyond the discrimination compartment indicated that the previous reactions had been to visual cues.

2. Trials with both lamps lighted. Initial confusion with rapid development of a position habit.

3. Control of reaction to temperature. With one lamp lighted the animal was started into the discrimination compartment. When he entered the illuminated alley, the lights were reversed, leaving the lamp bulb heated but dark in the alley chosen, lighted but not yet hot in the other. Turning back when the light was extinguished and prompt entry into the newly lighted alley gave evidence that the reaction was not to temperature.

4. Control of reaction to odor. *a*) Food odors. Both alleys from the discrimination compartment communicated similarly by small doorways with compartments containing food. These were always closed by light swinging doors of mica which the animal must open to enter the food compartment. *b*) Ozone from the punishment grill. As the box was arranged the grills were not charged until after errors were made.

5. Control for reaction to sound. The box was adjusted before the animal was brought from the home cage in another room. Correct discrimination was evidence that the animal was not reacting to the noise of setting the box.

6. Accidental cues from experimenter. Trials given by another person without disturbing discrimination controlled such accidental cues.

This series of tests is time-consuming and tends to break down the discrimination habit, since any slight change in the total situation disturbs the animal. Tests 1 and 2 were therefore made only with every fifth animal, the entire series with about one in ten. In no case was evidence obtained that the animal was discriminating on the basis of any stimuli other than the visual ones. Whether the reaction was to the retinal image of the lamp or to the general illumination of the apparatus was not determined. From the behavior of the animals and from tests made earlier (Lashley, '12) it is probable that both elements enter into the reaction.

Surgical and histological methods

Operations were performed with thermocautery under ether anesthesia. The lesions were restricted to the occipital third of the cortex, both hemispheres being injured in every operation, sometimes symmetrically, sometimes not. The extent and form of the injury was left largely to chance, although an effort was made to destroy every possible part and combination of parts of the occipital cortex in one or another animal.

Recovery from brain injury in the rat is rapid, and with this type of operation all traces of shock and discomfort from the wound disappear in three to five days. When the lesion lies outside of the visual area and is not too extensive, perfect reactions in the discrimination box may be obtained within twelve hours after operation, sometimes immediately on recovery from anaesthesia. A seven-day interval between operation and retention tests was therefore judged sufficient to allow for recovery from the immediate effects of the operation. This was further controlled by a group of animals with a fourteen-day interval.

For recording and computing the extent of lesions the formal diagram reproduced in the plates was used. This was constructed from serial sections of normal brains cut in transverse, horizontal, and sagittal section. From these, internal structures were projected to the surface and indicated by dotted lines. The rat's cortex shows no superficial markings to which the position of a lesion can be referred, so that in reconstruction of lesions from serial sections it is necessary to use internal structures for identification of the level of the sections. The principal structures used for later identification of levels were the anterior margin of the striatum (level 6), and genu (8), anterior commissure (11), posterior margin of the chiasma (13), origin of the tractus thalamo-mamillaris (15), anterior descending margin of the hippocampus (18), anterior end of aqueduct (20), anterior margin of inferior colliculi (23), caudal pole of the hemisphere (30). There is considerable individual variation in the relative positions of these structures in various brains and the reconstructions are subject to error on this account. To avoid this as far as possible, the following methods were used.

At necropsy the brains were removed and the extent and position of the visible lesion measured and transferred with proportional dividers to a printed diagram. The brain was then hardened, cut in transverse section, and stained with carbothionin. Camera sketches were made at intervals of 250 μ throughout the lesion. The levels of these sections were

determined by reference to internal structures. When any marked departure from average proportions appeared, the three sections corresponding most closely to levels 18, 23, and 29 were selected and the position of the others determined by interpolation.

The extent of the lesion on each section was next determined under higher magnification and marked on the camera sketch. Measurements were made on the sketch and transferred to the corresponding level on the diagram with proportional dividers. The points so determined were checked with the original sketch of the lesion made at necropsy, the points connected by the best fitting line, and the enclosed areas inked in.

To estimate the extent of the lesion the following method was used. The areas included between the parallel lines on the diagrams were treated as rectangles. The total length of these included within the diagram of the lesion gave the area of the lesion in arbitrary units, and this divided by the total length of the rectangles included in the entire diagram of the cortex gave the percentage of the cortex destroyed. In the measurements the entire diagram of the dorsal aspect was included, but only those portions of the lateral aspects lying below the level of the corpus callosum. This allows roughly for the overlapping of the dorsal and lateral aspects.

This method gives a crude measure of the percentage of the neopallium destroyed by the operation. The absolute percentage is not accurate, since the effects of perspective in the diagram and the exact limits of the neopallium are disregarded, but the use of the same method for all brains makes the percentages comparable. Repeated measurements of the same brain indicate an accuracy of $\pm$ 5 per cent for individual determinations.

With the completion of the diagrams, the sections were reexamined for lesions to subcortical structures, especially the fornix, hippocampus, thalamus, and colliculi. Lesions to these structures were rated in four grades: 0, no injury; 1, slight injury (estimated 10 per cent or less); 2, medium

(estimated 10 to 25 per cent); 3, severe (estimated 25 per cent or more). Cases showing any lesion to the thalamus were excluded from the series.

EXPERIMENTAL DATA

Plan of experiments

The problem involved the quantitative determination of the effects of various lesions to the visual areas upon the rate of formation of visual habits and upon the retention of visual habits formed before the cerebral insult, with controls for shock, diaschisis, and the like. The organization of the experiment is summarized in table 1. Three groups of animals

TABLE 1

Plan of experiments

GROUP	SERIAL NOS.	PROCEDURE	RETENTION TESTS	NUMBER OF CASES
A	1 to 48	Trained after operation	None	48
B	50 to 98	Trained before, tested after operation	After 7 days	49
C	103 to 112	Trained before, tested after operation	After 14 days	10
D	49, 99 to 102	Thalamic lesions		5

were trained, giving data upon separate points, but also serving as controls for each other. These were:

Group A. Male rats about 120 days of age were subjected to lesion in the occipital region. Seven days after operation, training was begun in the discrimination box and carried to completion with ten trials per day. The training records of the animals were compared with those of the following group (trained before operation) to determine the average effects of the lesions upon initial learning. The effects of lesions of different size were also tested by correlation with training records.

Group B. Normal male rats about 120 days of age were trained to perfect discrimination. They were then allowed to rest for seven days, after which retention was tested (pre-

liminary retention tests). Cerebral lesions were then produced and, seven days after operation, retention was again tested (postoperative retention tests). The first training records served as a control for group A. The preliminary retention tests measured the amount of loss to be expected from disuse in the period allowed for recovery from operation. The postoperative tests provided data for determination of the average effects of the lesions upon retention and for a comparison of the effects of lesions of different magnitudes by correlation with retraining records.

Group C. Since there was a possibility that in severe cases shock effects of operation might persist for more than seven days and so give a misleading appearance of greater loss in those cases, a further group was trained with longer intervals between training and the retention tests. After seven days' training, fourteen days after operation, all of the animals of group B were giving evidence of discrimination, although the retraining of some was not completed until later. This fact was taken as evidence that they were no longer suffering from shock or depression. The intrval of fourteen days was therefore adopted for group C, as adequate to allow for recovery from shock, and the preliminary and postoperative rest periods were both made of this length. The procedure with this group was otherwise as with group B.

Group D. At necropsy it was found that the thalamus had been injured in some cases. These were excluded from the major groups, but since the results with them have some bearing on the problem they are given separately.

Summary of data

Since the three experiments provide controls for each other, the raw data will first be presented for all of the groups, then summarized in relation to the various problems raised. The large number of cases included in the study, 112 in all, precludes the publication of individual protocols, but the essential data are presented in the following tables and the extents

and loci of the lesions are shown in the appended plates. In the tables and plates the cases are numbered serially and arranged for each group in the order of the magnitude of the lesions.

Group A. Animals with training after injury in the occipital region, to determine the effects of injury upon the rate of initial learning. The cases, numbered from 1 to 48, inclusive, are summarized in table 2. The diagrams in figures 1 to 48 represent the cerebral lesions in the animals bearing the corresponding numbers. The total area covered by all the lesions is shown in text figure 1.

The lesions range in extent from 3.5 to 43.9 per cent of the total neopallium, with an average of 17.9 $\pm$ 0.9 per cent. For learning the animals of this group required an average of 121.9 $\pm$ 4.5 trials, with an average of 39.1 $\pm$ 1.6 errors made during training.

Group B. Animals with initial training before brain injury. Preliminary retention tests at an interval of seven days, followed immediately by operation. Postoperative retention tests seven days after operation. These cases are summarized in table 3, numbered from 50 to 98, inclusive. The diagrams of the lesions, bearing the corresponding numbers, are shown in figures 50 to 98, inclusive. The total range of the lesions is shown in text figure 2.

The lesions range in extent from 1.5 to 31.9 per cent of the total extent of the neopallium, with an average of 15.8 $\pm$ 0.7 per cent. For initial learning before operation these animals required an average of 128.2 $\pm$ 5.2 trials, with 41.8 $\pm$ 2.5 errors made during training. In the preliminary retention tests they made an average of 1.4 errors. For relearning after operation they required an average of 44.6 $\pm$ 3.4 trials, with 13.7 $\pm$ 1.2 errors, or about one-third as many as in initial learning.

Group C. Animals trained before operation with preliminary retention tests after fourteen days. Operation followed these tests immediately and postoperative tests fourteen days after operation. Data on these cases, numbered from 103 to

TABLE 2

Data for group A, with initial training after occipital lesions. Serial numbers, per cent of neopallium destroyed, number of trials required for learning, number of errors made during learning, and estimated destruction of the hippocampus (H), superior colliculus (S), and inferior colliculus (I) are given in successive columns. Cases included from earlier studies are marked with an asterisk

SERIAL NO.	PER CENT DESTRUCTION	TRIALS FOR LEARNING	ERRORS DURING TRAINING	H	S	I
1	3.5	130	34	0	0	0
2	5.2	190	78	0	0	0
3	5.6	200	80	0	0	0
4	6.8	80	32	0	0	0
5	7.3	50	18	0	0	0
6	7.7	190	53	0	0	0
7	7.7	70	21	0	0	0
8	8.3	90	24	0	0	0
9	8.8	190	58	0	1	0
10	9.1	20	10	0	0	0
11	9.6	70	30	1	0	0
12	10.2	120	59	0	0	0
13	10.3	200	65	0	0	0
14	10.5	100	22	0	0	0
15	10.8	110	24	1	0	0
16	11.3	60	17	0	0	0
17	11.4	130	28	1	3	0
18	11.4	50	25	1	0	0
19	11.5	110	36	0	0	0
20	11.8	120	33	0	0	1
21	12.2	130	51	1	0	0
22*	13.2	80	37	.....	.....	.....
23	15.8	130	36	1	0	0
24	16.8	90	32	0	0	0
25	16.8	130	33	2	0	0
26*	17.5	140	66	.....	.....	.....
27*	17.6	100	33	.....	.....	.....
28*	18.1	100	28	.....	.....	.....
29	20.7	110	36	2	0	0
30*	21.1	200	61	.....	.....	.....
31	22.4	110	37	1	0	0
32*	22.8	160	53	.....	.....	.....
33	23.9	110	32	3	0	0
34	24.5	90	30	2	0	0
35	25.8	160	46	2	1	0
36*	25.8	140	37	.....	.....	.....
37*	26.6	120	28	.....	.....	.....
38*	26.7	190	57	.....	.....	.....
39*	26.9	160	68	.....	.....	.....
40*	28.0	200	52	.....	.....	.....
41	28.9	120	52	3	1	0
42	29.1	80	23	3	0	1
43	29.4	50	15	2	0	0
44*	29.8	190	34	.....	.....	.....
45	30.1	70	32	2	0	0
46	31.5	110	38	2	0	0
47*	33.3	170	36	.....	.....	.....
48*	43.9	130	49	.....	.....	.....
Average	17.9	121.9	39.1			
St.dev.	9.2	46.4	16.4			
P.E.	0.9	4.5	1.6			

TABLE 3

Data for group B, trained before operation, with postoperative retention tests seven days after operation. Serial numbers, per cent of neopallium destroyed, number of trials required for learning, number of errors during learning, errors in preliminary retention tests, errors made in postoperative tests, estimated destruction of hippocampus (H), of superior colliculi (S), and of inferior *colliculi (I) are given in successive columns. Cases included from earlier studies are marked with an asterisk*

SERIAL NO.	PER CENT DESTRUCTION	TRIALS FOR LEARNING	ERRORS	PRELIMINARY TESTS	POSTOPERATIVE TESTS		H	S	I
					Trials	Errors			
50*	1.5				0	0	0	0	0
51	4.9	140	20	0	20	5	0	0	0
52	5.1	110	35	0	30	3	0	0	0
53	5.4	60	14	3	10	2	0	0	0
54	5.6	90	27	0	0	0	0	0	0
55	6.0	100	25	8	0	0	0	0	0
56	7.4	120	62	2	40	16	0	0	0
57	7.7	280	92	5	0	0	0	0	0
58	8.1	100	41	0	10	1	0	1	0
59	8.2	220	65	0	30	2	0	0	0
60	8.3	140	46	3	40	3	0	0	0
61	8.9	180	88	1	0	0	0	0	0
62	10.7	60	18	0	10	2	1	0	0
63	11.5	130	26	0	20	5	2	0	0
64	11.7	120	17	1	80	24	1	0	0
65	12.4	90	34	0	50	14	1	0	0
66	12.5	190	62	3	0	0	1	0	0
67	12.7	150	25	0	10	1	0	0	0
68	12.9	110	37	2	40	9	0	0	3
69	13.8	90	32	1	0	0	0	0	0
70	14.5	130	58	0	80	15	1	2	0
71	14.6	150	40	0	30	5	0	1	1
72	14.7	190	41	0	20	4	0	0	0
73	15.2	60	16	1	0	0	0	2	0
74	15.6	170	73	4	50	20	0	0	0
75	15.8	170	40	0	90	39	1	0	0
76	16.3	130	52	8	20	10	1	1	0
77	16.9	90	47	4	30	4	1	0	0
78	17.2	140	43	0	50	14	1	1	0
79*	17.6	60		1	100	27	0	0	0
80	17.8	100	33	0	20	7	1	0	0
81	18.0	170	44	7	30	8	1	0	0
82*	18.6	60		1	74	31	1	0	0
83	18.7	100	36	0	90	21	3	0	0
84	19.7	190	51	7	10	3	1	2	0
85	20.4	100	43	0	40	11	1	0	0
86	20.6	130	70	0	70	22	0	0	0
87	20.7	70	37	0	110	38	2	0	0
88	21.2	80	41	0	60	31	1	0	0
89	21.4	210	54	4	60	19	1	2	0
90	21.5	310	75	0	70	26	1	1	0
91*	22.4	150	65		90	38	0	0	0
92	24.5	60	19	0	70	25	2	1	0
93	27.7	110	38	0	60	20	3	1	0
94	28.1	130	38	0	80	20	2	0	0
95	28.3	210	92	1	70	21	0	0	0
96	28.8	80	27		110	40	0	0	0
97	29.1	100	29		110	40	2	0	0
98	31.9	150	79	0	100	24	2	0	0
Average	15.8	128.2	41.8	1.4	44.6	13.7			
St.dev.	7.2	61.4	25.4		34.8	12.7			
P.E.	0.7	5.2	2.5	0.23	3.4	1.2			

112, are summarized in table 4. The lesions are shown in plate 8, figures 103 to 112. The total range of the lesions is shown in text figure 3. The average extent of the lesions in these cases is 25.9 ± 1.5 per cent of the neopallium. For learning they required an average of 146.0 ± 8.8 trials with 41.7 ± 2.9 errors. In the preliminary retention tests they made an average of 4.8 errors. For relearning after operation they required an average of 93.0 ± 9.5 trials with 20.4 ± 2.1 errors.

TABLE 4

Data for group C, trained before operation with postoperative retention tests fourteen days after operation. Arranged as table 3

SERIAL NO.	PER CENT DESTRUCTION	TRIALS FOR LEARNING	ERRORS	PRELIMINARY TESTS	POSTOPERATIVE TESTS		H	S	I
					Trials	Errors			
103	15.7	140	35	11	20	5	1	0	0
104	19.3	230	52	0	40	6	2	1	0
105	20.5	120	17	4	90	30	1	0	0
106	21.5	190	56	1	120	22	3	0	1
107	24.2	100	33	1	180	29	0	0	0
108	25.8	190	58	4	80	29	1	0	0
109	26.0	100	48	3	90	22	3	0	2
110	33.0	140	53	10	120	34	3	0	0
111	33.6	120	21	8	130	15	3	0	0
112	39.8	130	44	6	60	12	3	0	0
Average	25.9	146.0	41.7	4.8	93.0	20.4			
P.E.	1.5	8.8	2.9	0.7	9.5	2.1			

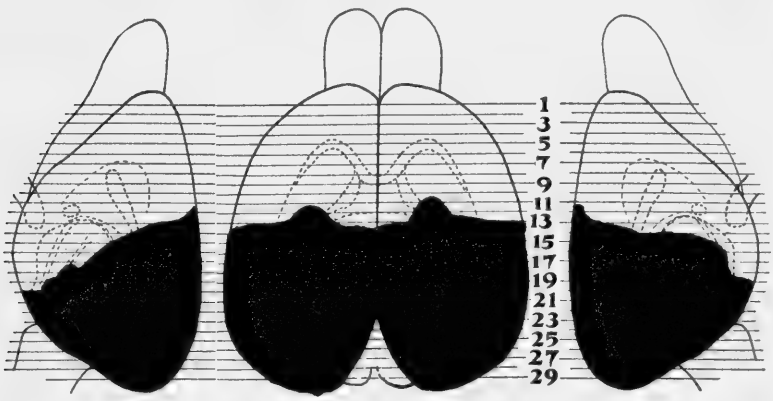
Group D. Cases with lesions to the thalamus. One case, no. 49 (fig. 49), was obtained with extensive lesions in the optic nuclei of the thalamus, followed by initial training. This animal required 280 trials for learning, with 132 errors. The percentage of the neopallium destroyed was 25.2, with practically all of the hippocampus degenerated and with some injury to the superior colliculi.

Four cases with injury to the thalamus after training were obtained. These were nos. 99, 100, 101, and 102. They required an average of 167.5 trials for learning, with 36.2 errors. In preliminary retention tests they averaged 1.7 errors. In postoperative tests they required 152.0 trials, with 54.2 errors.

THE EFFECT OF OCCIPITAL LESIONS UPON THE INITIAL FORMATION OF A VISUAL HABIT

The animals in group A were given initial training in visual discrimination after lesions in the occipital region varying in extent from 3.1 to 43.9 per cent of the neopallium. The average amount of destruction in the group was 17.9 ± 0.9 per cent. The total series (figs. 1 to 48) covers every part of the occipital third of the cortex. The total extent of injury in these cases is shown in text figure 1.

The training records of group A are summarized in table 2. The forty-eight animals required an average of 121.9 ± 4.5



Text fig. 1 Composite diagram made by superimposing the diagrams of the lesions for cases 1 to 48, group A, showing that every part of the occipital third of the cerebrum was destroyed in one or another of these animals.

trials for learning and made an average of 39.1 ± 1.6 errors during training. For comparison with these figures, the learning rates of unoperated animals trained under the same conditions are given in table 3, columns 3 and 4. These normal animals required an average of 128.2 ± 5.2 trials for learning and made an average of 41.8 ± 2.5 errors during training. The differences between the normal and operated groups are given below.

	Trials	*Errors*
Normal,	128.2 ± 5.2	41.8 ± 2.5
Operated,	121.9 ± 4.5	39.1 ± 1.6
Difference,	6.3 ± 6.9	2.7 ± 3.0

The slight differences in favor of the operated animals are less than their probable errors and are not significant.

In view of reported fluctuations in human behavior following brain lesions, it seemed possible that the operations might have produced a greater variability in the learning rates of these animals, even though the means were unaffected. To test this the standard coefficients of variation have been computed for the normal and operated animals and are given below.

	Trials	*Errors*
Normal animals,	0.479 ± 0.032	0.607 ± 0.041
Operated animals,	0.380 ± 0.026	0.418 ± 0.027
Difference,	0.099 ± 0.041	0.189 ± 0.049

The differences here are relatively larger than those between the means. There is a suggestion that variability was actually reduced by operation, but the differences are scarcely significant.

The lesions range in extent from 3.5 to 43.9 per cent of the neopallium. In order to determine whether or not there is a relation between the extent of injury and the rate of learning, the areas of the lesions have been correlated with the learning scores for all cases. Spearman's formula for rank order was used. The constants are given below.

For lesions with trials, $p = 0.132 \pm 0.142$
For lesions and errors, $p = 0.088 \pm 0.143$

The correlations are small and indicate that for the habit in question there is no significant relationship between the extent of brain injury and the ability to form the habit of reaction to brightness within the limits of the experiment.

From the data summarized in this section we may conclude that lesions to the occipital areas of the rat's cerebrum, whether slight or extensive, have no effect upon the ability of the animals to form habits of brightness discrimination. After complete destruction of the occipital third of the cortex, visual learning progresses as rapidly as in normal animals.

THE EFFECT OF OCCIPITAL LESIONS UPON THE RETENTION OF VISUAL HABITS FORMED BEFORE OPERATION

Mass relationships

Two groups, B and C, were used for tests of retention after operation. The data on them are summarized in tables 3 and 4. Group B constituted the chief experiment; C, the control for diaschisis effects. Group B will therefore be considered first. The constants for the group are given below.

	Trials for learning	*Errors during training*
Learning before operation,	128.2 ± 5.2	41.8 ± 2.5
Preliminary retention tests,		1.4 ± 0.23
Postoperative retention tests,	44.6 ± 3.4	13.7 ± 1.22

The preliminary retention tests give a measure of the loss to be expected from disuse during an interval of seven days, which was that later allowed for recovery from the shock of the operation. The small number of errors made following this seven-day rest period (1.4 ± 0.23) shows that the loss from disuse may be treated as negligible in considering the effects of operation.

The averages of 44.6 trials and 13.7 errors in the postoperative retention tests show that, on the average, a definite loss, equivalent to about one-third of the effect of initial practice, was produced by the operation. The lesions in this group (figs. 50 to 98) form a continuous series from 1.5 to 31.9 per cent of the neopallium, with an average of 15.8 ± 0.69 per cent. They cover almost the posterior half of the cortex, as shown in text figure 2. Some lesions within this range therefore reduce the retention of visual habits, although, as appeared in the preceding section, similar lesions have no effect upon subsequent initial learning.

Inspection of table 3 reveals the fact that many of the animals with slight lesions showed no disturbance of the habit following the operation, whereas those with extensive lesions often required as many trials for relearning as for the initial learning. This suggests a relationship between the extent of lesion and the amount of loss of the habit produced by it. It has been tested by computing correlations between the

extent of lesion and the scores made in postoperative retention tests by all the animals. Rank order and product-moment methods have both been employed. The results by the two methods are identical to the second decimal, so only the former are given below.

Extent of lesion with trials for relearning, $p = 0.712 \pm 0.047$
Extent of lesion with errors in retraining, $p = 0.721 \pm 0.046$

These coefficients are about sixteen times as great as their probable errors, and therefore clearly significant. They indicate a close relationship between the extent of injury and the amount of deterioration of the habit formed before operation.

The data in table 3 suggest that the relationship may not be rectilinear and, to test this, correlation ratios have been computed for trials and errors on per cent destruction. The constants obtained were the following:

Trials on per cent, $\eta = 0.828 \pm 0.030$
Errors on per cent, $\eta = 0.841 \pm 0.028$

The correlation ratios are considerably larger than the coefficients, but the difference is only 1.9 times its probable error (Blakeman's formula) and is not sufficient to establish the curvilinear character of the relationship.

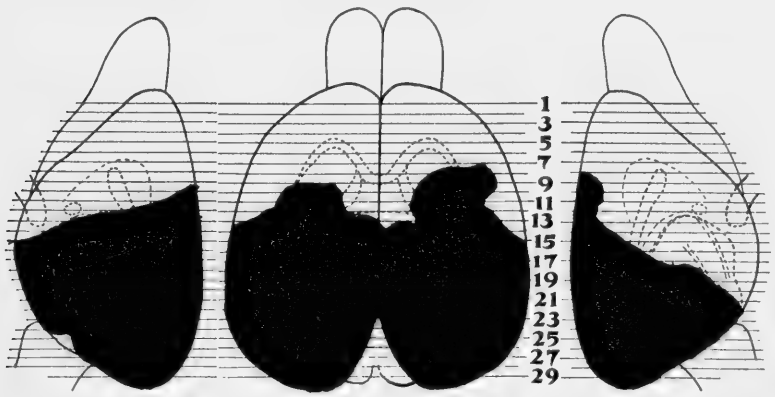
Possible causes of spurious correlation

Measured by either of these constants, the relationship between memory loss and extent of injury is surprisingly close. Is it due to the actual removal of cerebral tissue or only to some secondary effects of the operation, such as general shock or diaschisis? The possible secondary factors which might produce a spurious relationship are:

1. General shock from operation and irritation of the wound. In an earlier paper (Lashley, '21) I have shown that extensive lesions to the frontal lobes and corpus striatum may produce little disturbance of visual discrimination. Judged from its general effects, this is a very much more severe operation than even the extensive occipital ones, yet the effects were

no greater than those of the lesser operations in the present series (average 33.3 trials, 6.9 errors for relearning). It seems, then, that differences in the general shock effects of the operation are inadequate to account for the correlations found. A further argument against general shock may be derived from group A in which training was begun at the same interval after operation as in group B, without the slightest effect of the operation upon the rate of initial learning.

2. A diaschisis effect or specific depression of lower visual centers requiring time for spontaneous recovery proportional



Text fig. 2 Composite diagram made by superimposing the diagrams of the lesions in cases 50 to 98, group B, showing the total range of the injuries in this series.

to the extent of the injury might result in a failure of animals with extensive lesions to show retention, because the interval between insult and retention tests was insufficient to allow of recovery from the severer depression. To control this possibility, the experiment with group C was undertaken. The method with this group was precisely the same as that with group B, except that fourteen days were allowed to intervene between training and preliminary retention tests and between operation and postoperative tests. The data for group C are summarized in table 4 and the lesions are shown in figures 103 to 112. The average extent of lesion for the group was

25.9 ± 1.5, thus considerably greater than that in group B, since the object of the experiment was to test the effects of the longer interval on recovery from extensive injury. The total extent of the lesions is shown in text figure 3. Constants for the group are given below:

	Trials	*Errors*
Initial learning,	146.0 ± 8.8	41.7 ± 2.9
Preliminary retention tests,		4.8 ± 0.7
Postoperative retention tests,	93.0 ± 9.5	20.4 ± 2.1
Average per cent destruction,	25.9 ± 1.5	

The average loss in this group is very much greater than that in group B (63 per cent loss as against 33 per cent). Judged on this basis, the longer rest period following operation did not reduce the amount of practice necessary for relearning, but a better estimate of the effects of the rest period may be gained by comparing groups with equal amounts of destruction. For this purpose similar constants have been computed for the fifteen cases of group B having the greatest cerebral lesions (nos. 84 to 98, inclusive) and are given below.

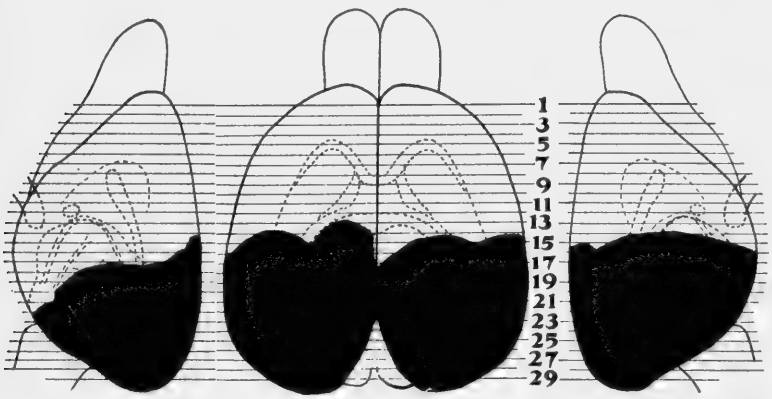
	Trials	*Errors*
Initial learning,	138.7 ± 11.4	50.5 ± 3.1
Preliminary retention tests,		0.8 ± 0.2
Postoperative retention tests,	74.0 ± 4.7	25.2 ± 5.7
Average per cent destruction,	23.8 ± 1.2	

The average extent of lesion in these cases from group B is very close to that of group C. In retention tests group C, with the longer rest period after operation, is inferior as judged by trials and superior as judged by errors, but the differences are in neither case significantly greater than their probable errors.

The loss from disuse shown by the preliminary retention tests is somewhat greater after the fourteen-day interval than after the seven-day, but is not sufficient to mask a recovery from operative shock, if this had occurred. The experiment proves that no better retention records are made after the fourteen- than after the seven-day rest period following operation. Since, as was pointed out above, all the animals of

group B had recovered from shock and were giving clear evidence of discrimination after fourteen days, the experiment seems to prove that neither general shock nor temporary diaschisis is an important factor in determining the inferior retention of the animals with more extensive lesions.

3. If small lesions produced no effect, whereas extensive lesions abolished the habit of brightness discrimination, a spurious correlation might arise through the inclusion of both types of cases. It is possible that function is determined by the presence of a critical amount of tissue, somewhat as the power of regeneration is limited in lower animals. To



Text fig. 3 Composite diagram made by superimposing the figures of the lesions in cases 103 to 112, group C, showing the total range of the lesions in these cases.

test this, the cases in table 3 were divided into three groups comprising the cases with the lesser (nos. 50 to 66), median (nos. 67 to 82) and greater (nos. 83 to 98) lesions and the correlations for extent of lesion with errors in relearning were computed for each of these subgroups. The constants obtained were the following:

For the lower third, $p = 0.28 \pm 0.15$
For the middle third, $p = 0.59 \pm 0.11$
For the higher third, $p = 0.59 \pm 0.11$

The correlation was also computed for group C, giving the constant $p = 0.30 \pm 0.19$.

For these subgroups the correlations are less than for the group as a whole,[4] as usually follows in any case when the range of one variable is decreased, but they are in all cases positive and fairly large. The analysis, therefore, does not bear out the postulate of a critical mass necessary for the performance of the habit, but rather indicates that deterioration from brain lesion is a continuous function of the extent of the injury.

4. Munk ('81), Luciani and Seppilli ('86), and other students of the visual area have held that there is a focal point for visual function in the occipital region, with surrounding areas of lesser importance, although still concerned in vision. It is possible that in this series of operations some lesions involved such a focal point, others missed it; that the larger the lesion, the better the chance of including the focal point, and that consequently a greater proportion of the larger lesions than of the smaller produced loss of the habit and so led to a spurious correlation. To test this, the loci of injury have been subjected to the following analysis.

Records of the animals which showed the greatest amount of deterioration, making thirty or more errors (75 per cent as many errors as made by them in initial learning) were selected and a composite diagram constructed to determine whether all involved the destruction of a common focal point. These were cases 75, 82, 87, 88, 91, 96, and 97. The composite diagram obtained is given in text figure 4. In it the parts which escaped destruction in some cases but were destroyed

[4] By the formula,

$$r'_{xy} = 1 - (1 - r_{xy}) \frac{\sigma^2_y}{\sigma'^2_y},$$

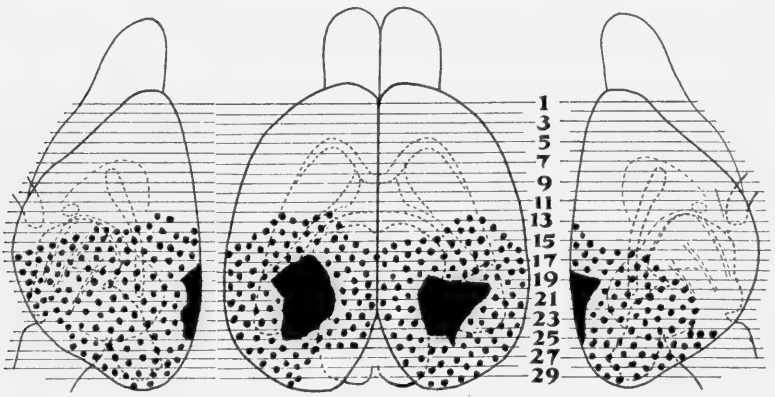
(Otis, A. S., Jour. Ed. Psychol., 1922, vol. 13, p. 293) the expected correlations from these data, due to reduction in range only, are:

For the lower third, $r = 0.04$
For the middle third, $r = 0.65$
For the higher third, $r = 0.58$

This indicates that the reduction in correlation actually found on splitting up the data is due solely to the reduction in range and that the correlation for the whole group ($r = 0.721$) represents a continuous function of the group rather than an artifact arising from the inclusion of heterogeneous samples.

in others are stippled. The areas which were destroyed in every case are marked in solid black. Only the dorsal convexity of the occipital region of both hemispheres was destroyed in all. Consequently, this must be the focal point implied in the foregoing hypothesis. But this area was also completely destroyed in cases 55, 58, 59, 61, 62, 69, and 73, none of which made more than two errors in retention tests. This dorsal area alone cannot, therefore, have any special significance for the visual function.

All cases which made not more than five errors in retention tests were selected and a composite diagram constructed of



Text fig. 4 Composite diagram made my superimposing the lesions in animals of group B which made more than thirty errors in retention tests. The areas in solid black were destroyed in all cases. The stippled areas in some, but not all.

the lesions in them, to determine if any possible visual area uniformly escaped destruction. These were nos. 50, 51, 52, 53, 54, 55, 57, 58, 59, 60, 61, 62, 63, 66, 67, 69, 71, 72, 73, 77, and 84. The total extent of the lesions in these cases is shown in text figure 5. The combined lesions cover the entire occipital third of the cortex with the exception of the extreme lateral pole of the right hemisphere. In this series no possible focal point could have escaped injury, and the fact that none of the animals showed deterioration of the habit demonstrates that no such point exists within the occipital region of the cortex.

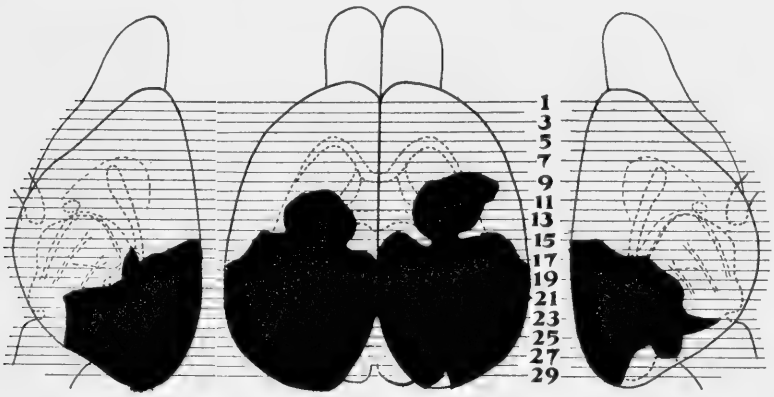
5. Since many of the cases showed lesions in subcortical structures, we must further inquire into the rôle of these in the production of the defects. Cases with thalamic lesions have been excluded from the series. Lesions to the hippocampi and colliculi are indicated in table 3. Inspection shows that there is no significant correspondence between the lesions in the colliculi and the severity of the symptoms. The extent of lesion to the hippocampi follows closely that to the cortex. With a larger number of cases, the calculation of partial coefficients might show the relative significance of lesions in the two regions for the production of the habit disturbances; but with so small a number of cases, these would be unreliable, and we can only judge from the anatomical relations of the hippocampus that it is probably not concerned in the production of the results.

Scotoma versus habit interference

These considerations seem to rule out the possibility that the correlation between amnesia and the extent of injury is a secondary effect of the operations, dependent upon shock or temporary depression of the lower visual centers. They show also that it is not due to an error in sampling a large area including a smaller hypothetical visual center, and that it is almost certainly not due to chance injury to lower visual centers. Consequently, we must ascribe the results to the actual reduction in the mass of cerebral tissue. Injury to the occipital area interferes with the habit of brightness discrimination in direct proportion to the extent of the lesion.

The establishment of the quantitative relationship still leaves the cerebral mechanism undetermined. The results presented for group B are capable of two different interpretations. The lesions may have produced a cortical blindness or they may have interfered with the habit organization without affecting the purely sensory mechanism. The former is the more in accord with the traditional view of occipital lobe function and will therefore be considered first.

In order that cortical blindness should produce the results obtained, the following conditions must be met: The lesions must produce areas of scotoma proportional in size to the extent of the injury. With such totally blind areas in the visual field the chances that the functional area would be stimulated by the light in any given trial might be inversely proportional to the size of the scotomatous areas. Animals with large injuries would tend to make more errors. Improvement during the retention tests would consist in the animal's learning to fixate the stimulus light with the intact parts of the retina.



Text fig. 5 Composite diagram made by superimposing the diagrams of the lesions in all cases of group B which made not more than five errors in retention tests. Every part of the occipital third of the cerebrum was destroyed in one or another of these cases without serious deterioration of the habit.

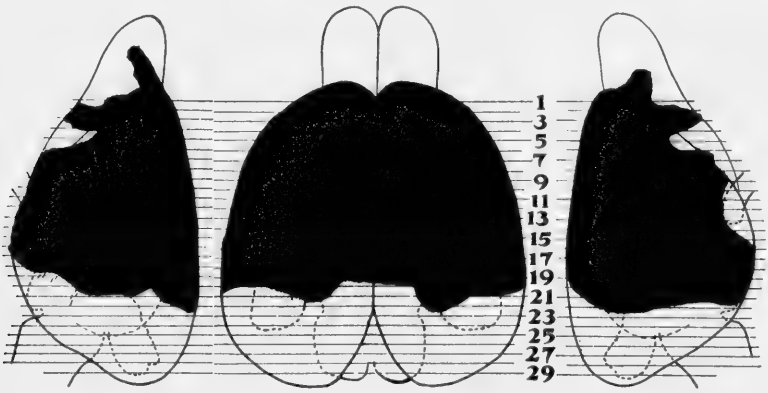
Several considerations oppose this view and, I believe, make it untenable: 1) No part of the behavior of the animals in retention tests suggests this adaptation in fixation. The rat's eye can be rotated only slightly and fixation involves orientation of the head, but no unusual postures were ever noted in the operated animals. 2) If the difficulty in discrimination had resulted from scotoma, it should have appeared in the learning of group A as well as in the retention tests of group B, since scotoma is equally a blindness to new and to familiar objects. No influence of the lesion was apparent,

however, in group A, so that it seems certain that cortical blindness could not have been responsible for the results with group B, the lesions in the two groups being practically identical. 3) Finally, from what we know of the conditions of vision in the rat and in man, the production of scotoma in the rat seems very improbable.

Poppelreuter ('23) has distinguished six levels of complexity in the organization of visual function in man, basing his conclusions chiefly on hemianopic cases. These are: *Level 1, amorphous quantitative sensitivity.* Differences in intensity of illumination are recognized, but without location or form in the hemianopic field. *Level 2, size perception without definite form or localization* within the visual field. *Level 3, amorphous form perception.* The general direction of single lines crossing the visual field can be distinguished, but any complication of lines or patterns appears amorphous. *Level 4, perception of discrete objects.* The number which can be distinguished within the hemianopic field is very limited and patterns are not identified. *Level 5, mild amblyopia.* True pattern vision is possible. *Level 6, normal vision.*

Vision in the rat is at a very primitive level. Color vision is absent (Watson and Watson, '13), true pattern vision is probably lacking, and the best evidence suggests that the animal can distinguish differences of brightness, differences of size, and gross differences in the direction of single lines within the visual field (Waugh, '10; Lashley, '12). Thus the limit of visual sensitivity in the rat corresponds rather closely with Poppelreuter's third level. In the present study we are dealing only with the most primitive level, that of reaction to great differences in brightness (level 1), and this Poppelreuter finds to be retained in practically every case of hemianopsia. There is, then, no reason from analogy with man to believe that cerebral lesions will produce scotoma in the vision of the rat for brightness, and the dissimilarity of the results for groups A and B gives conclusive evidence against scotoma as an explanation of the quantitative results found.

The same objection holds for the hypothesis that the quantitative results are due to a total amblyopia proportional in severity to the extent of the lesions. These should likewise have affected equally the learning and retention tests. Thus, as I have pointed out in an earlier paper (Lashley, '20), the rapid formation of visual habits in the rat after destruction of the occipital region of the cortex shows that the sensitivity of the animals to visual stimuli is unimpaired; only the retention of habits formed before the injury is affected. The present experiment seems to establish this fact beyond question. It is the mechanism which maintains the organization of the habit, the engram in Semon's terminology, and not the mechanism of visual sensitivity which is destroyed.



Text fig. 6 Composite diagram, showing the total extent of lesions in the frontal, temporal, and parietal regions which produced no significant effect upon the habit of brightness discrimination.

The relation of habit deterioration to the locus of the injury

The location of the visual area in the rat's brain can be determined only by inference from the effects of cerebral injuries. As I have shown in earlier papers (Lashley, '20, '21), the habit of brightness discrimination survives the destruction, singly, of the frontal, temporal, and parietal areas over the region illustrated in text figure 6. Experiments reported earlier and group B of the present series demonstrate that extensive destructions in the occipital third of

the cortex abolish the visual habit. We must conclude, therefore, that the cerebral mechanism of the visual habit is largely confined to this occipital region.

Within this region the experiments show that the various parts are equipotential for the performance of the habit. As was brought out in the preceding section, animals making thirty or more errors in retention tests showed a distribution of lesions such that only a small dorsal region (text fig. 4) was common to all. The range of destruction in these animals is from 15.8 to 29.1 per cent of the neopallium. The average destruction was 22.4 per cent. Thus, except for the small dorsal region, every possible part of the visual area escaped destruction in one or another animal, which nevertheless showed serious disturbance of the habit. In contrast to this, the combinations of lesions in animals which showed little disturbance covers every part of the occipital region (text fig. 5). Thus, the habit of brightness discrimination survives the destruction of any part of the occipital region, provided that the lesion is small, whereas it is abolished by larger lesions irrespective of their location within the occipital areas. This can only mean that the cerebral mechanism of the habit, whatever its physiological character may be, is diffused throughout the occipital region. Any part of the mechanism can perform the functions of the whole, in the absence of other parts, provided only that a sufficient quantity of tissue remains intact. The evidence opposed to the view that this is the result of scotoma seems conclusive and leaves only the hypothesis that the lesions produce an amnesia, as consistent with all the results of the experiment. The quantitative data point to the conclusion that the efficiency of the memory trace is proportional to the amount of functional tissue, irrespective of its locus, and this in turn suggests that the function of the memory trace must in some way be additive, efficiency increasing as a simple function of the mass irrespective of the neural patterns involved.

THE EFFECT OF THALAMIC LESIONS

One case, no. 49, with injury to the thalamus was found in the series with training after operation. The number of trials required by this animal was 280, with 132 errors. The average for the group with which he was trained was 121.9 trials with 39.1 errors. The greatest amount of practice required by any member of the group was 200 trials, with 80 errors. This suggests that the thalamic lesion was effective in retarding the learning, although one case is far from proving the point. The extent of cortical lesion in this case is shown in figure 49. The surface lesion is not greater than that of animals which showed no retardation of learning. In addition to the cortical lesion, there were severe injuries to the optic radiations and to the pulvinar on both sides, complete degeneration of the fornix in both hemispheres, and extensive injury to the left superior colliculus.

Among the animals with training before operation and retention tests after, four were found with injuries to the thalamus. These were nos. 99, 100, 101, and 102. The surface lesions are shown in figures 99 to 102. The training records are given below.

NO.	LEARNING		PRELIMINARY RETENTION TESTS	POSTOPERATIVE RETENTION TESTS		PER CENT DESTRUCTION
	Trials	Errors	Errors	Trials	Errors	
99	80	33	0	80	18	24.0
100	220[1]	68	0	130	51	28.1
101	70	25	3	250	65	30.3
102	300[1]	109	4	150	83	31.6

[1] The extensive initial training of these animals is due to the fact that they were given the first 100 trials without punishment for errors.

The thalamic lesions in these cases were the following:

No. 99. In the right hemisphere there was a very slight injury in the pulvinar, scarcely penetrating the surface. The optic radiations and lateral geniculate body were uninjured. On the left side there was extensive injury to the anterior

thalamic nucleus, some injury to the optic radiations, but scarcely any in the optic nuclei. Real injury to the optic paths and nuclei of the thalamus was questionable.

No. 100. There were slight injuries in the region of the nucleus habenulae on each side. On the left there were slight injuries to the optic radiations, but other visual structures were uninjured. The right lateral ventricle was much enlarged, with almost total destruction of the whole external capsule of the right hemisphere. An old cyst indicated that much of the destruction was due to infection. This is the only case reported in which there was evidence of infection of the cerebral substance.

No. 101. On the right there were extensive lesions in the pulvinar and lateral geniculate nucleus, extending caudad to include part of the superior colliculus. The optic radiations were almost completely destroyed. On the left the thalamus was not injured.

No. 102. The left pulvinar and optic radiations were severely injured. The right thalamus was untouched.

There are no data on the exact limits of the visual nuclei in the rat's thalamus, so that only a rough estimate of the extent of the lesion can be made. In nos. 101 and 102 the injuries were restricted to one side, but there involved a great part of the optic path. In other cases the injuries were slight.

The mean number of trials in the retention tests of the group with which these animals were trained was 44.6, with 13.7 errors. They all, therefore, exceeded the average of the group. The mean trials required by cases with equal cortical lesions (25 per cent or more) was 88.3, with 27.5 errors. The maximum was 110 trials, with 40 errors. All except no. 99, therefore, required more practice for relearning than did cases of similar cortical injury without lesions to the thalamus.

We cannot draw conclusions from so few cases, but there is here a suggestion that thalamic lesions disturb retention to a greater extent than do cortical injuries and to a degree proportional to the extent of the lesion.

INTERPRETATION OF RESULTS

The data presented in the foregoing sections seem clean-cut and adequate to establish the lack of any influence of injury to the occipital cortex upon the learning of brightness discrimination and the quantitative relation between the extent of injury and the amount of practice necessary for recovery from the resultant amnesia. The data tell nothing, however, of the way in which these effects are brought about so that their interpretation with reference to the broader problems of cerebral function must be largely speculative. Neither the failure of animals with occipital lesions to show retardation in learning nor the quantitative effect of lesions on retention conforms to expectation from current neurological theory, so that we must be doubly cautious in drawing conclusions. If the results stood alone in contradiction to the classical views of cerebral function, I should hesitate to draw any inferences from them, but they come only as a further step in a series of observations, all of which emphasize the dynamic function of cerebral tissue and the lack of any absolute localization of so-called mental faculties. The accumulated evidence seems to demand some revision of our theories of cerebral mechanism, so that, even though we can form but a vague notion of the modifications which the theories must undergo, it seems worth while to point out some of the implications of the data, both for the sake of setting further problems and for the light which the more general, if speculative, conclusions can throw on some of the obscure questions of the cerebral mechanism of thought.

Learning as a non-localized function

The fact that the ability to form habits of brightness discrimination is not in the least affected by complete destruction of the occipital third of the cortex is difficult to reconcile with any current theory of cortical function. The loss of visual habits after destruction of this occipital region clearly establishes it as a visual area in the usual meaning of the term. The postoperative retention tests in no case required

a significantly greater number of trials for relearning of the visual habit than are required in the original learning (except perhaps in cases of thalamic lesion). The operative destruction, therefore, did not interfere with the capacity to relearn any more than it did with the capacity for original learning. Only the after-effects of previous training were reduced or abolished.

This result is in general accord with previous findings for the motor habits of the 'inclined plane' and 'double-platform' boxes (Lashley and Franz, '17; Lashley, '20) in the rat, where it was shown that habits destroyed by injury to the frontal regions are reacquired with normal facility; with Franz's findings ('07) for motor habits in cats and monkeys when the frontal lobes are destroyed, and for monkeys after lesions in the visual areas (Franz, '11). Data on aphasia (Franz, '24) indicate that in man also relearning of habits lost through cerebral insult may progress at normal rate. It is true that this rapid recovery in man is not invariable, but, as Monakow states, the unimprovable cases are those of extensive diffuse lesions. They therefore probably correspond to the cases of very extensive lesion in the rat (Lashley, '20) in which learning is definitely retarded.

If, as seems clearly established, these smaller lesions do not lessen the animal's ability to learn, we must conclude that no part of the cerebral cortex is better adapted for the formation of any particular habit than is any other. Any anatomically continuous cerebral area may serve the learning function, provided it presents a sufficient mass. This must mean that in a problem situation the effects of stimulation irradiate to all parts of the cortex. As the habit is established there comes into being a definite structural modification having topographical position and capable of destruction by brain injury. The learning process is independent of locus, whereas the mnemonic trace or engram has a definite localization.[5]

[5] The problem of localization is perhaps even more complicated than these data indicate. I have records of two animals in which practically all of the cortex

We have taken it for granted that the location of the visual function in the occipital region is due to the massing in this area of projection fibers from the optic nuclei of the thalamus, and this view is doubtless correct, but there remains to be explained the fact that these fibers are of no especial importance for the learning of visual habits. Since the habit can be established equally readily in the absence of the occipital projection fibers and yet normally has cerebral representation there, it seems as though these must normally restrict the habit mechanism to the occipital region, not only by conducting impulses to this part of the cortex, but also by exerting some inhibitory action upon those parts which take over the habit function when the occipital areas are destroyed.

A similar example is offered by the observation of Goldstein ('23) that a pseudofovea develops only in patients with complete hemianopsia, and not in hemiamblyopia. Here the functioning of part of the visual area of the injured hemisphere seems to prevent the reorganization of the visual mechanisms, whereas the complete destruction of the area permits the shift of fovea. The cases are not parallel, for in the human patients there is only a reorganization within the intact field, and not, as in the rat, a vicarious function for the hemianopic field, yet both conditions suggest that functional areas somehow actively restrict similar functions to themselves.

Mutual facilitation of cerebral areas

We do not know what is lost when a habit deteriorates—whether there is a dropping out of some essential elements

in front of the occipital region (the anterior two-thirds) was destroyed after training in brightness discrimination. The whole occipital region with its thalamic connections remained intact. These animals lost the habit as a result of the operation and relearned it in no more trials than were required for their original learning, thus showing that the loss was no more due to a general deterioration than it is in the case of occipital lesions. More work must be done before the significance of these cases will be clear, but they suggest that an extensive lesion in one part of the cortex may have the same effect as a more limited one in another part—a further indication of the dynamic relations of the cortex as a whole.

with retention of others or a general weakening of the whole mechanism. In some instances habits are possibly aggregates of relatively independent activities linked together by simple associative bonds, as in the case of reproduction of lists of words, where the forgetting of one link may block reproduction of the whole series, or in the maze habit which has been held to consist of a simple somaesthetic-motor chain. The habit of visual discrimination seems, on the contrary, to be a unitary act. Although it requires the coordination of many acts in response to at least two stimuli, the actual discrimination cannot be analyzed into independent parts, but seems to function either in its entirety or not at all. A similar type of learning is presented by the conditioned reflex, in which a single reaction is associated with the conditioned stimulus. During training the reaction sometimes follows the conditioned stimulus, sometimes fails to do so, but whenever the reaction does appear, it is as perfect as any later amount of practice can make it. The improvement can be stated only in terms of the proportion of trials in which the conditioned reflex is elicited. So with the discrimination reaction, the loss can be stated only in terms of the percentage of trials in which discrimination does not occur.

The behavior of the subjects, both in conditioned reflex and discrimination experiments, suggests that the incompletely formed habit is subject to inhibition by distracting stimuli (Bechterew, '13) which gradually lose their effectiveness as the habit is more firmly established. With the conditioned reflex the distracting stimuli are easily observed. With the discrimination habit we cannot always detect the source of the interference. The condition rather resembles the fluctuations of function described by Franz ('24) for aphasia, where the subject may at times inexplicably have command of a sizable vocabulary, at other times be speechless. Yet, although we cannot detect the interfering agent, we may not assume that the engram is now present, now absent. We must rather take it for granted either that irrelevant neural processes sometimes inhibit the reaction or that, in a condition

of lowered tonus, the habit mechanism is incapable of providing a sufficient mass of excitation to activate the motor centers except under special conditions of reinforcement.

Forgetting might then be stated as a general weakening of the habit mechanism in the sense either that its power to dominate all other cerebral processes in the problem situation is lowered or that it can excite motor centers only when they are in a condition of hyperexcitability or tonic activity. The results of these experiments may thus be translated from terms of the number of trials required for relearning to those of efficiency of the engram in dominating the cerebral field or in overcoming resistance of relatively inexcitable motor centers, and we are justified in speaking of gradations in the strength of the engram.

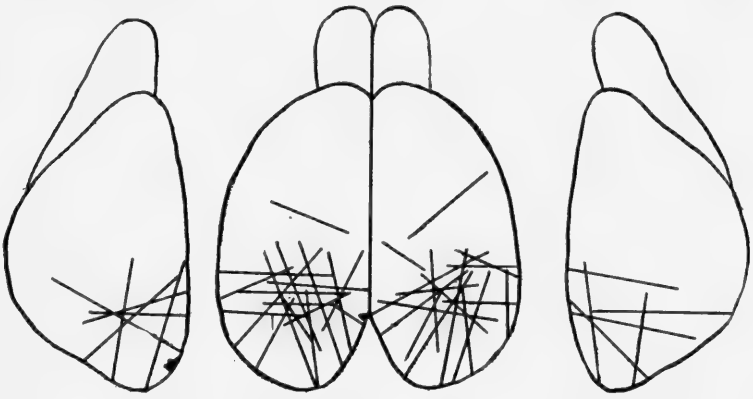
This leads to the conception of a general weakening of a unitary habit mechanism following injury in the visual area, which parallels such conditions as nominal aphasia, where there is not a loss of a larger or smaller number of words, but a greater or lesser difficulty in recalling all words of a given type. The correlations between extent of lesion and loss of the habit show that the degree of weakening of the engram is proportional to the extent of the lesion, irrespective of its position. As a corollary of this, the larger the mass of intact tissue (or the greater the number of neurons) within the functional area, the greater the efficiency of the habit mechanism, no matter what particular neurons within the system are preserved or destroyed. This can only mean that the equipotential parts of a habit system exert some sort of mutual reinforcement under normal conditions; that there is a summational effect in the production of the habitual acts. The conditioned reflex arcs or postulated elements of the habit system are therefore not independent, but closely organized in this dynamic relationship.

The lesions described in this study almost without exception penetrated the cortex and destroyed the underlying substantia alba throughout their greater extent. They thus cut any association fibers which underlay the injured cortex and

hence severed any direct connections which may have existed between areas on opposite sides of the lesions. Text figure 7 is a composite diagram showing by straight lines the long axes of the lesions in those animals which made not more than five errors in retention tests. In every case included in the diagram tissue probably intact and functional in the visual habit lay on each side of the lesion, so that the lines represent planes of section between functional areas involved in the habit mechanism. In the entire series the occipital cortex was divided in a great variety of planes and, in one case or another, almost every possible part of the area was isolated from other parts. Yet in these cases the retention of the habit was but little affected. Thus it appears that the cerebral mechanisms of the habit can function normally and can exert their mutual reinforcement in spite of the destruction of any particular group of cortical association fibers within the area, provided only that the divided portions of the area retain some connection with the remainder of the cortex or with subcortical structures. This means either that the facilitation is exerted solely upon centers centrifugal to the occipital area through a common termination of discrete paths from that area or that the cerebral mechanism is of such a character that facilitating impulses between neighboring areas may traverse any anatomical bridge which happens to remain intact.

In previous discussions of equipotentiality of areas within the cortex I have assumed reduplication of parts as the simplest hypothesis to account for it. That is, the muscular contractions involved in the performance of the habit might be initiated by impulses coming over reflex paths which are scattered uniformly throughout the functional area and impinge on the same final common path. A small lesion might then destroy a part of these paths, but leave enough intact to carry out the function. Mutual facilitation of such reduplicated paths might furnish a mechanism for the mass action of the visual area, but certain aspects of the present data throw some doubt upon this and suggest a different interpretation of the facts.

Brightness discrimination is not a simple reaction which can be stated readily in terms comparable to the descriptions of spinal or of conditioned reflexes. The stimulus is complex. The simultaneous or successive applications of at least two optical stimuli is necessary to activate the habit. The reaction is not merely an advance to light or retreat from darkness. If both alleys of the discrimination box are darkened the trained animal will not enter either, as is to be expected, since he is trained to avoid the darkened alley. But if both alleys are illuminated the animal also frequently refuses to advance. His response is thus not merely tropistic to a lighted



Text fig. 7 Composite diagram, showing by lines the long axes of lesions which separated parts of the occipital area in animals which made not more than five errors in retention tests.

alley, but is conditioned by the presence of both stimuli. In addition to this, the habit is conditioned by the tactile and olfactory stimuli of the problem box and by hunger. It thus involves a complexity of integration which seems unlikely to be carried out without the activity of transcortical association paths.

The relative importance of the cerebral cortex and thalamus in the performance of such acts of discrimination is as yet an open question. Discrimination habits have been established in animals with a very primitive cerebrum (e.g., White, '19) and even in invertebrates (e.g., von Frisch, '14) so that

we cannot say that the cerebral cortex is necessary for their performance. Nevertheless, attempts to set up such habits in decerebrate birds and mammals have not as yet been successful. In these forms the cortex seems to have usurped these functions or to have acquired some dynamic relation to the thalamus which prevents the latter from functioning in the absence of cortical facilitation.

The evidence at hand is inadequate to decide whether the activity in question involves facilitation within the cortical area or merely conduction over isolated paths through the cortex. If the former, then there must be facilitation without determinate association paths; if the latter, the cortex is left without any significant integrating function. Neither conclusion conforms with our notions of cerebral activity, yet there seems no third alternative. We can only deal in probabilities here; yet in view of the fact that the direction taken by further investigation, both experimental and clinical, will in some measure be determined by our preconceptions of cerebral function in such cases as this, it seems worth while to balance the probabilities and to formulate the problem more definitely.

Although we have abandoned the doctrine that single memories are stored in single brain cells, we still cling to a somewhat similar belief in the theory that the capacity for each particular response is maintained by a condition of low synaptic resistance between certain definite neurons arranged in a more or less intricate pattern. This has proved to be a useful conception, but that it fully describes all neural organization is open to question. Data on equipotentiality of cerebral areas suggest that to some extent the habit mechanism is independent of particular neurons. The possibility of facilitation between cerebral areas in spite of the section of any particular group of association fibers may indicate a still further lack of dependence upon determinate neuron connections. Once we grant this as a possibility, a host of facts suggest themselves which seem explicable in no other way than by a mass action of nervous tissue independent of

specific conduction paths or predetermined localization. In general, these are instances where the reaction is determined not by the stimulation of particular nerve endings, but by the excitation of any receptors so long as a constant pattern or ratio among them is maintained; or where the reaction consists not in the activation of a given group of muscles, but in the movement of any effector in a certain relation to the orientation of the body. I need only cite a few examples in order to show the common occurrence of such reactions.

Martin ('22) has pointed out that the character of the vasomotor reflex is determined by "the total impulse-stream generated within the afferent portion of the nerve trunk in a unit of time," irrespective of the particular fibers stimulated. In the field of vision we have such familiar facts as that two objects of unequal brightness or size may produce a constant differential reaction no matter upon what part of the retina their images are projected, or what angle they subtend. Thus I found for the rat (Lashley, '12) that when trained to choose the larger of two visual patterns, he would continue to choose the larger, even when both exceeded in area the larger with which he had been trained. Köhler ('21) has reported a number of similar observations for the chimpanzee. In the cutaneous field Weber's circles form another familiar example of the same principle. On the motor side we see many instances of functional equivalence in the employment of diverse muscle groups. I have reported cases of this sort in the transfer of training to limbs paralyzed during training (Lashley, '24 a). Marina's experiments ('15) on interchange of eye muscles are of a similar type, and a familiar example is the common ability to trace script of any size with either hand or foot.

Among the more complex integrations of man this principle of independence of particulate neurons is even more clearly indicated. Head's analysis of the semantic type of aphasia (Head, '20) is the best recent example. The patients lose the ability to distinguish or think certain relationships, although the words which express these relationships are still retained.

Examples ranging from cerebral mass function in the rat's discrimination of brightness to the very complex conditions of aphasia in polyglots, who lose merely the 'spirit' of a language, all point to the same conclusion. The thing which is localized is, in the majority of cases, the ability to relate any reactions in certain specific ways, rather than a relation between specific reactions. That is, modes of thinking or reacting rather than specific thoughts or reactions are eliminated by cerebral lesions. A sensory pattern projected on the cortex must shift from place to place with movements of the stimulus over the sensory surface and yet may retain its capacity to elicit a constant reaction; it must therefore act as a whole and not by isolated connections of single cortical cells with lower motor levels. Once a cortical system of integration is established for one sensory-motor coordination, other sensory-motor combinations seem capable of fitting spontaneously into the schema, even though different afferent and efferent neurons are involved (e.g., the conformation of new word combinations to the habitual grammatical form). The situation seems best described by saying that when a final common path is sensitized to a given cortical pattern, it will respond to that pattern no matter in what part of the cortex it occurs. The significant feature of the cortical pattern is the ratio between its parts, and not the particular neurons excited.

There are many points in common between the behavior data cited above and the direct physiological data obtained in the present experiment. The former point to a cerebral mechanism which, although spacially extended, behaves as a unit and within which dynamic relations or stresses are more important than particulate neuron connections. The physiological work indicates a cerebral area which behaves as a unit whose parts are equivalent in function, capable of summated action and independent of particulate cortical association paths. The analogy is so close as to suggest that in the operative experiments we have really isolated a mechanism which underlies the more complex adaptive reactions: that

the mass action shown in these experiments is a general principle of neural activity.

Current doctrines of cerebral function, in so far as they attempt to specify the mechanism of integration and are not content with vague ascriptions of mental traits to anatomical areas, are based upon the principle of reflex organization. In its present form this conception implies the dependence of reaction upon the connections of particular neurons. It of course permits of innumerable complications through inhibition or facilitation exerted between reflex arcs, but the principle remains that of absolute dependence of the reaction upon the particular neurons which are activated. Direct adaptive association between afferent and efferent paths can only occur when low synaptic resistances already exist between them, established either by growth or learning processes. New coordinations can be established only by 'random' activity and 'selection.'

The facts of mass action do not readily fit into such a schema and in conjunction with the facts of temporal variation, equipotentiality, and the functional equivalence of final common paths form a consistent body of physiological evidence opposed to the reflex hypothesis. This, with behavior problems of the type cited above, seems to demand a plasticity of neural function which cannot be deduced from any explanatory system dependent upon the connections of particulate neurons. As Herrick says, "the concept of the reflex is not a general master key competent to unlock all of the secrets of brain and mind," and the inadequacy of the reflex theory seems to demand the formulation of some additional or alternative hypotheses. As yet, too little is known of the non-conformable cases to permit of any detailed statement. It must suffice at present to point out that the problem raised for neural function has many points in common with the problems of morphogenesis which arise in experimental studies of embryology and regeneration. In both cases it is the pattern or total relationship of parts as well as particulate structures which determines the final product. The best-

fitting theory of the mechanism of development is that which appeals to physiological stresses, as elaborated by Child ('23) in his discussions of physiological gradients, and it seems probable that further development of theories of neural function must proceed along somewhat similar lines.

SUMMARY

To test the influence of the extent of brain injury upon the learning and retention of reaction to brightness, rats were trained before and after cerebral lesions of various extents and loci. Enough cases were obtained to permit of statistical treatment of the results. The results indicate that:

1. Injuries to the occipital region inflicted before training and including every possible part of the occipital third of the cerebrum have no effect upon the ability of the animals to form the habit of brightness discrimination. Total destruction of the 'visual' area does not reduce the speed with which a simple visual habit may be formed.

2. Injuries in the same region produced after the habit was established resulted in a weakening or total loss of the habit.

3. The loss, as measured by the amount of practice necessary to reestablish the habit, was closely proportional to the extent of the injury and independent of its locus within the occipital third of the cortex.

4. Evidence is given to show that the loss of the habit is not the result of operative shock or of the production of scotoma. The degree of retention is a direct function of the amount of nervous tissue intact.

5. Lesions in the optic radiations and optic nuclei of the thalamus seem to retard learning of visual habits.

6. The relation between cerebral mass and the efficiency of retention is interpreted as indicating a summation of the activities of different parts of the visual area. This summation takes place in spite of the cutting of any particular group of association fibers.

7. From this it is argued that the theory which makes the conditioned-reflex arc the unit of cerebral organization is

inadequate and that an additional cerebral mechanism permitting greater plasticity of action and resembling in its action the syncytium of lower invertebrates must be postulated.

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PLATES

EXPLANATION OF PLATES

In the diagrams shown on the following plates a uniform arrangement has been followed. The dorsal and lateral aspects of the brains are represented with internal structures projected as dotted lines on the surface. The extent of the lesions is represented by the blackened areas. The numeral on the left is the serial number of the animal, corresponding to the data given in tables 2, 3, and 4, The figures on the right give the percentage of the neopallium destroyed by the operation.

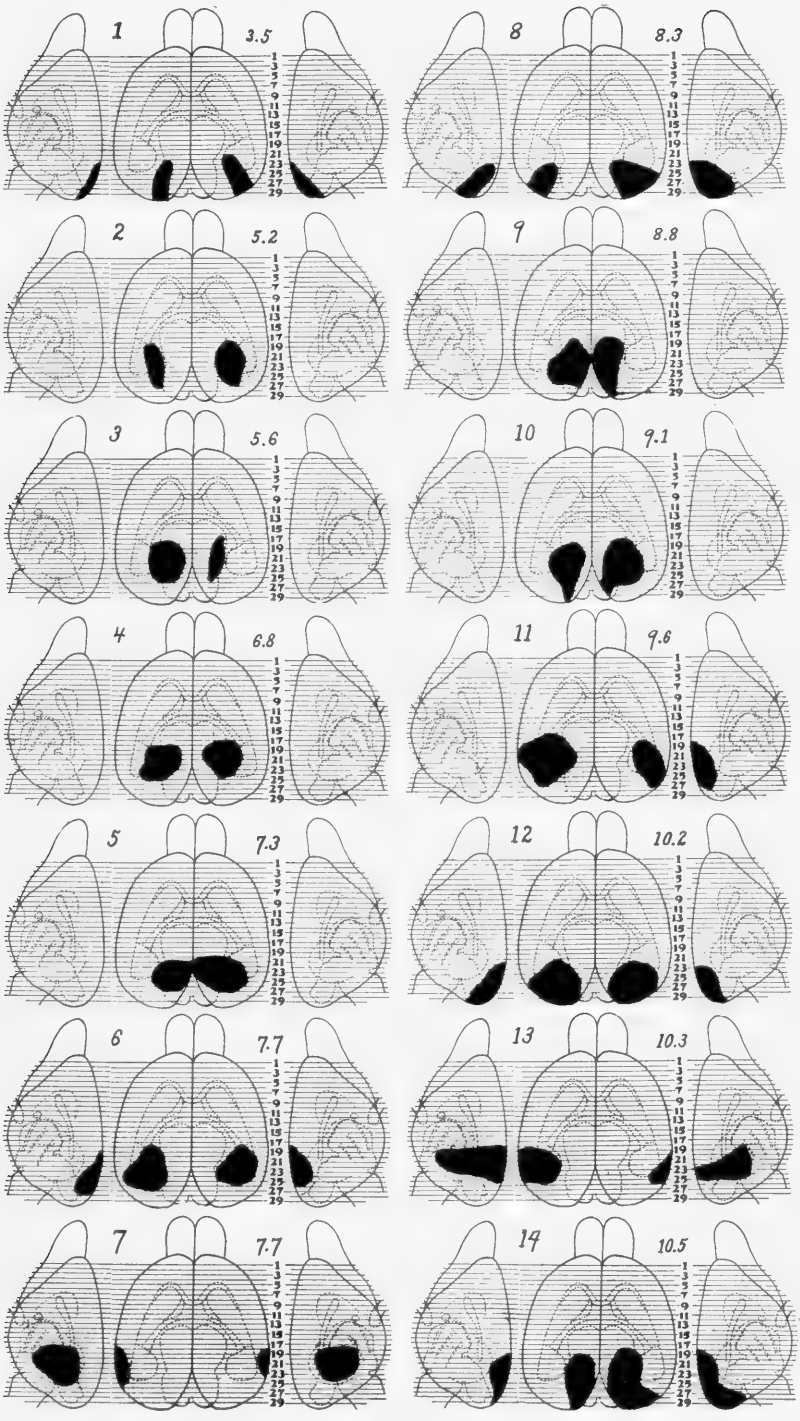
Figures 1 to 48, inclusive (pls. 1, 2, 3, and 4). Cases in group I, trained after operation, arranged in order of the magnitude of the lesions.

Figure 49 (pl. 4). Case with thalamic lesion trained after operation.

Figures 50 to 98, inclusive (pls. 4, 5, 6, 7, and 8). Cases in group B trained before operation, with retention tested after seven days. Arranged in the order of magnitude of the lesions.

Figures 99 to 102, inclusive (pl. 8). Cases trained before operation. Retention tested after thalamic lesions.

Figures 103 to 112, inclusive (pl. 8). Cases in group C trained before operation, with retention tested after fourteen days.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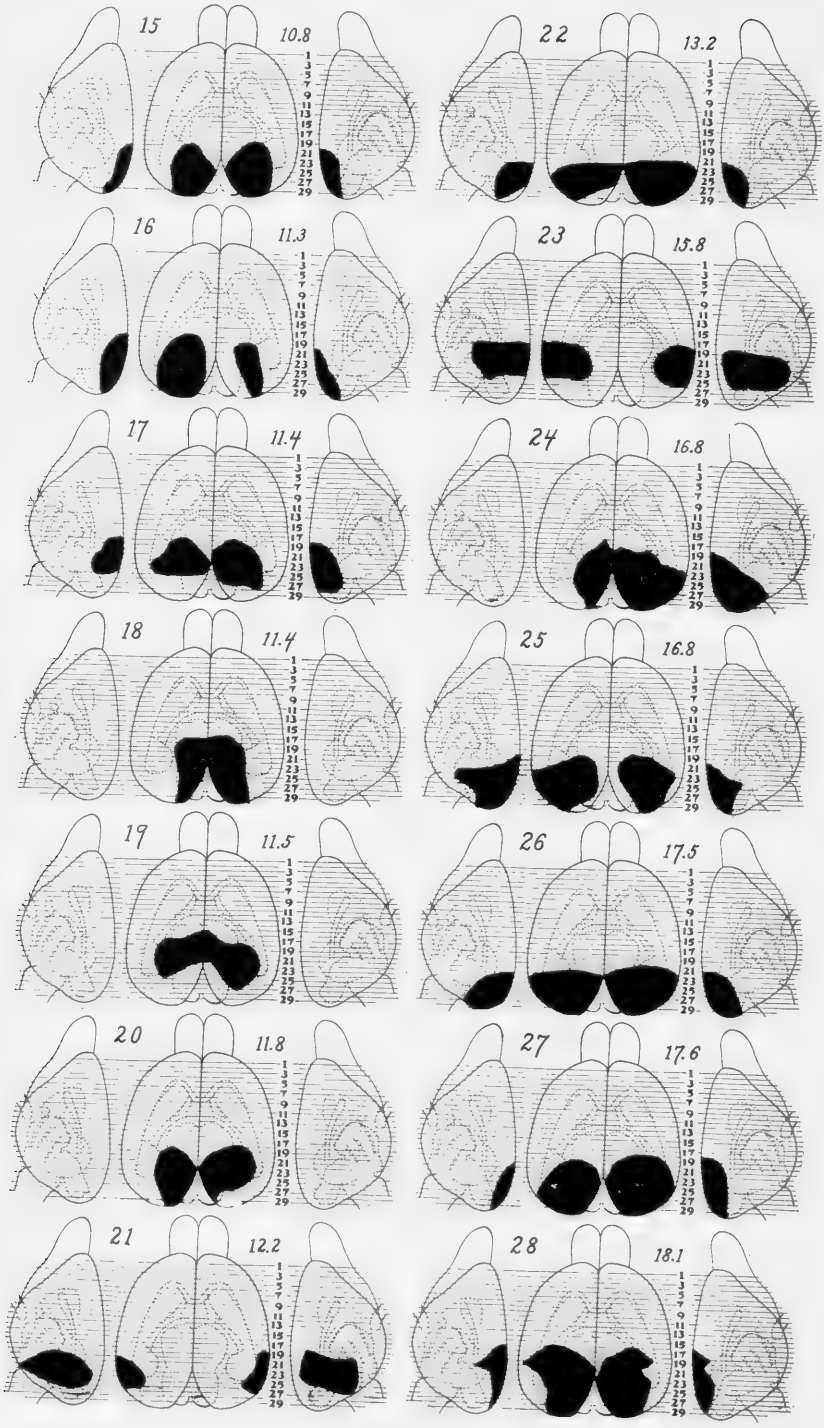
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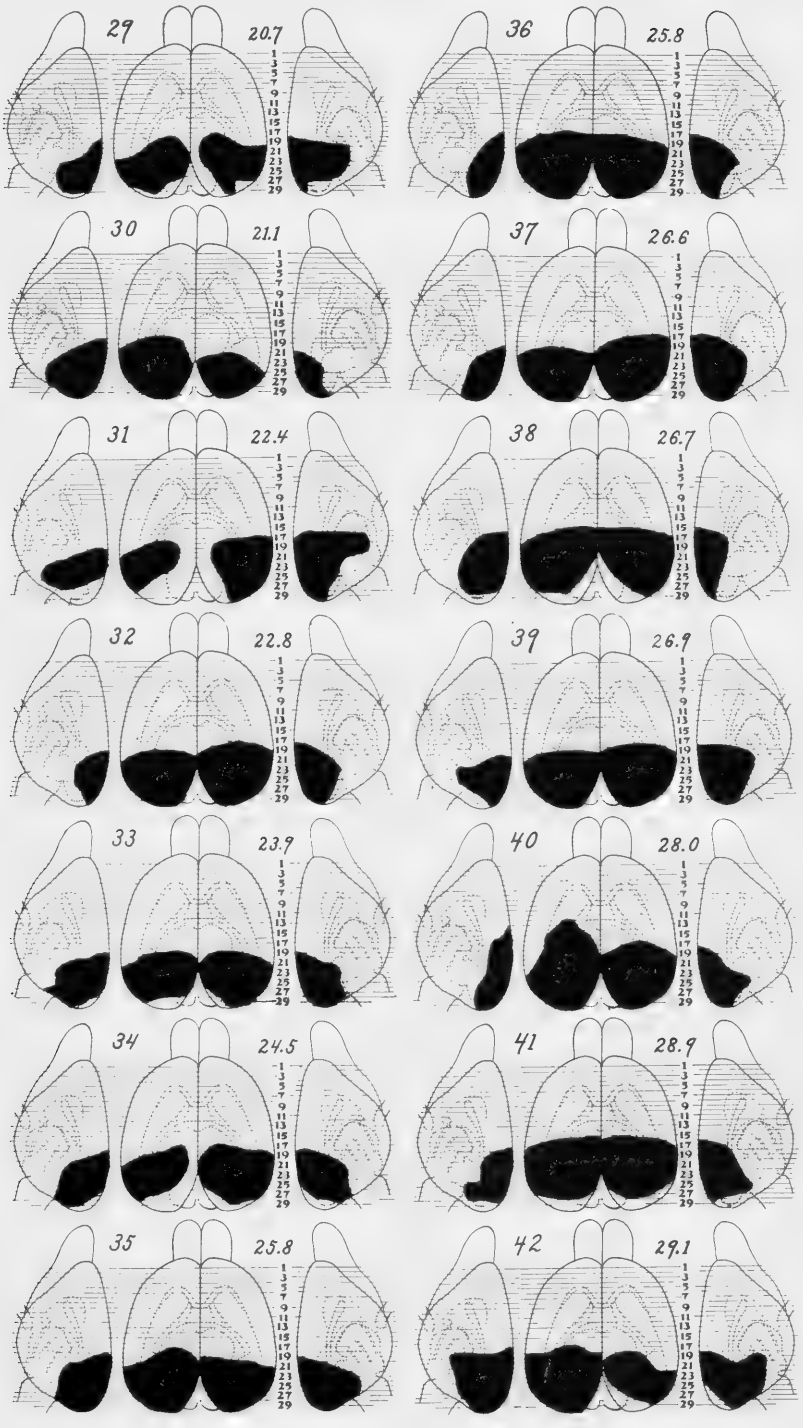
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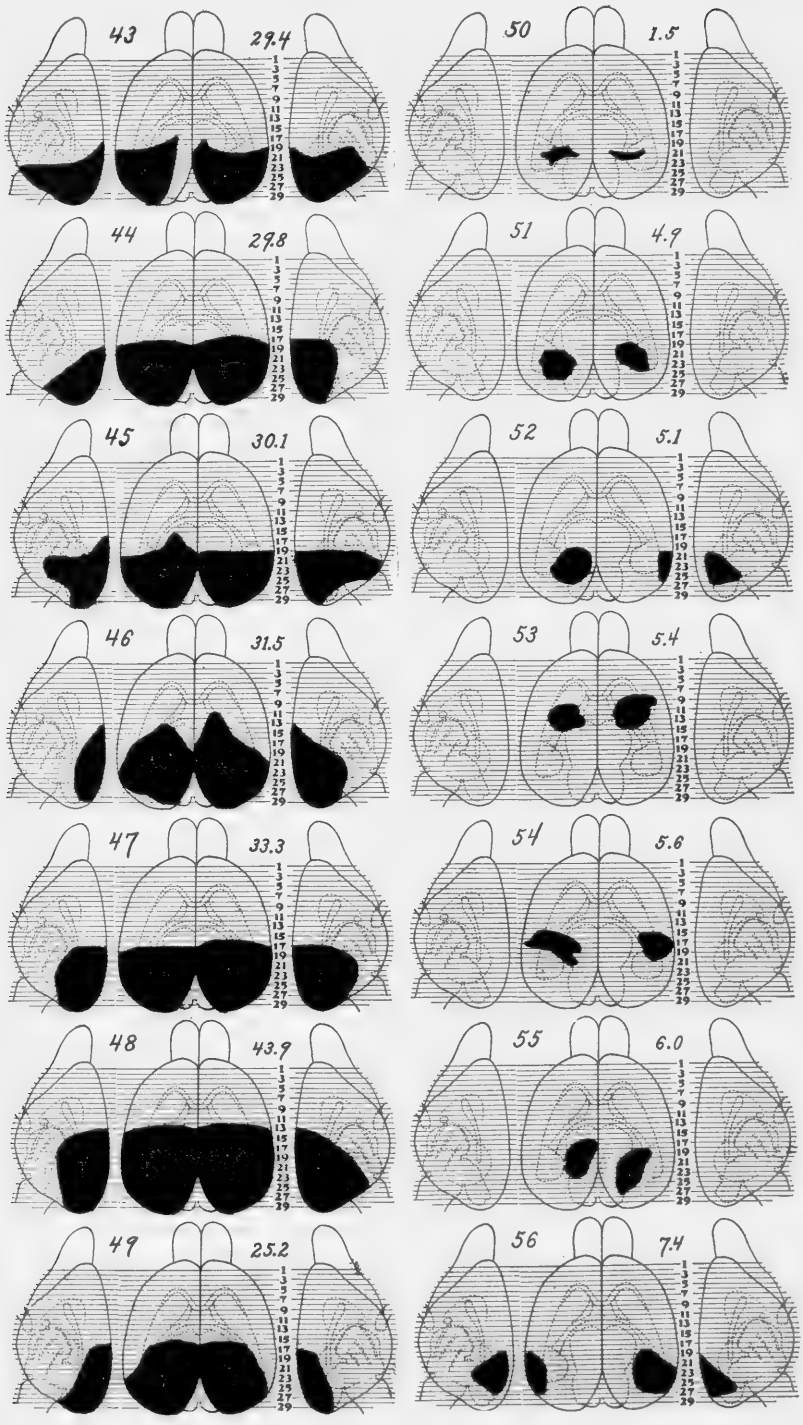
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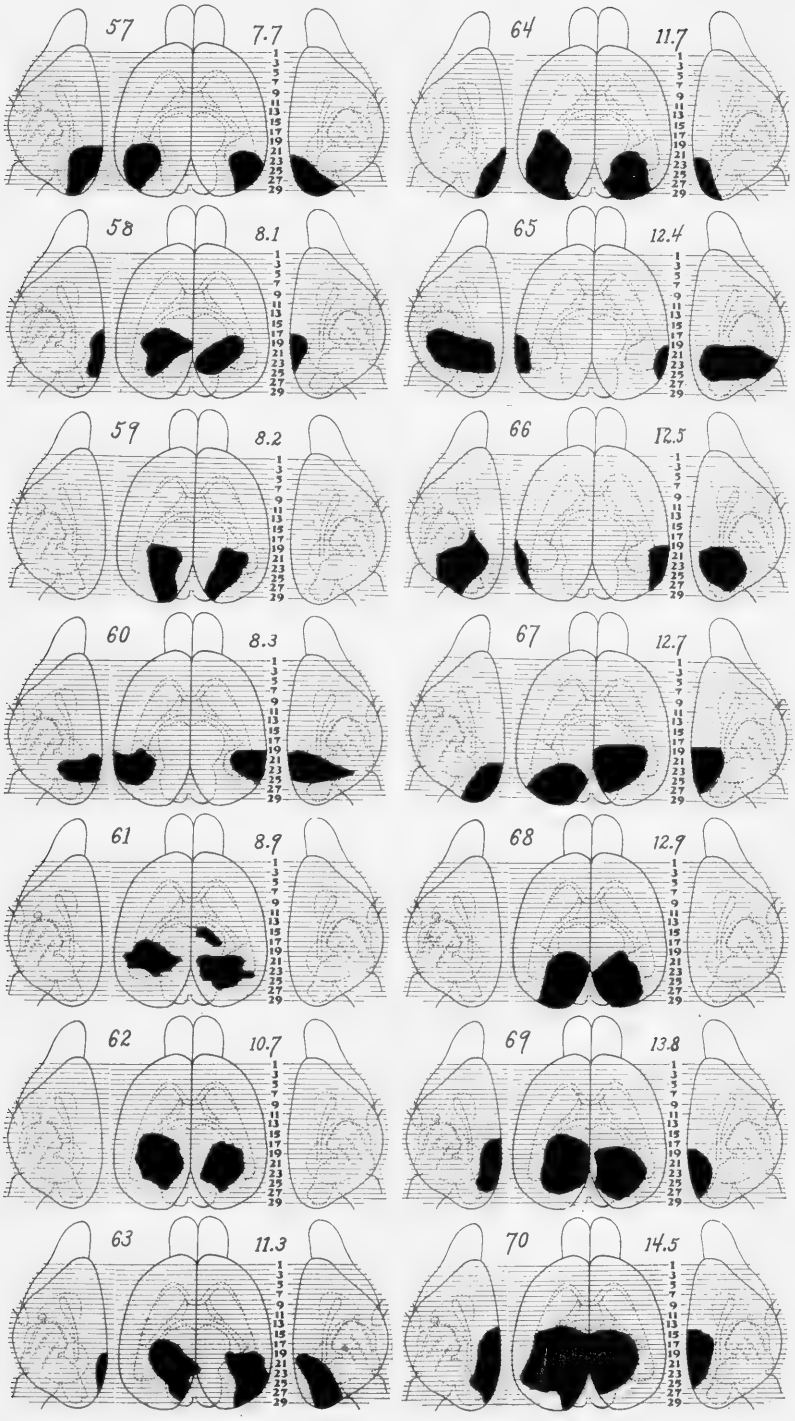
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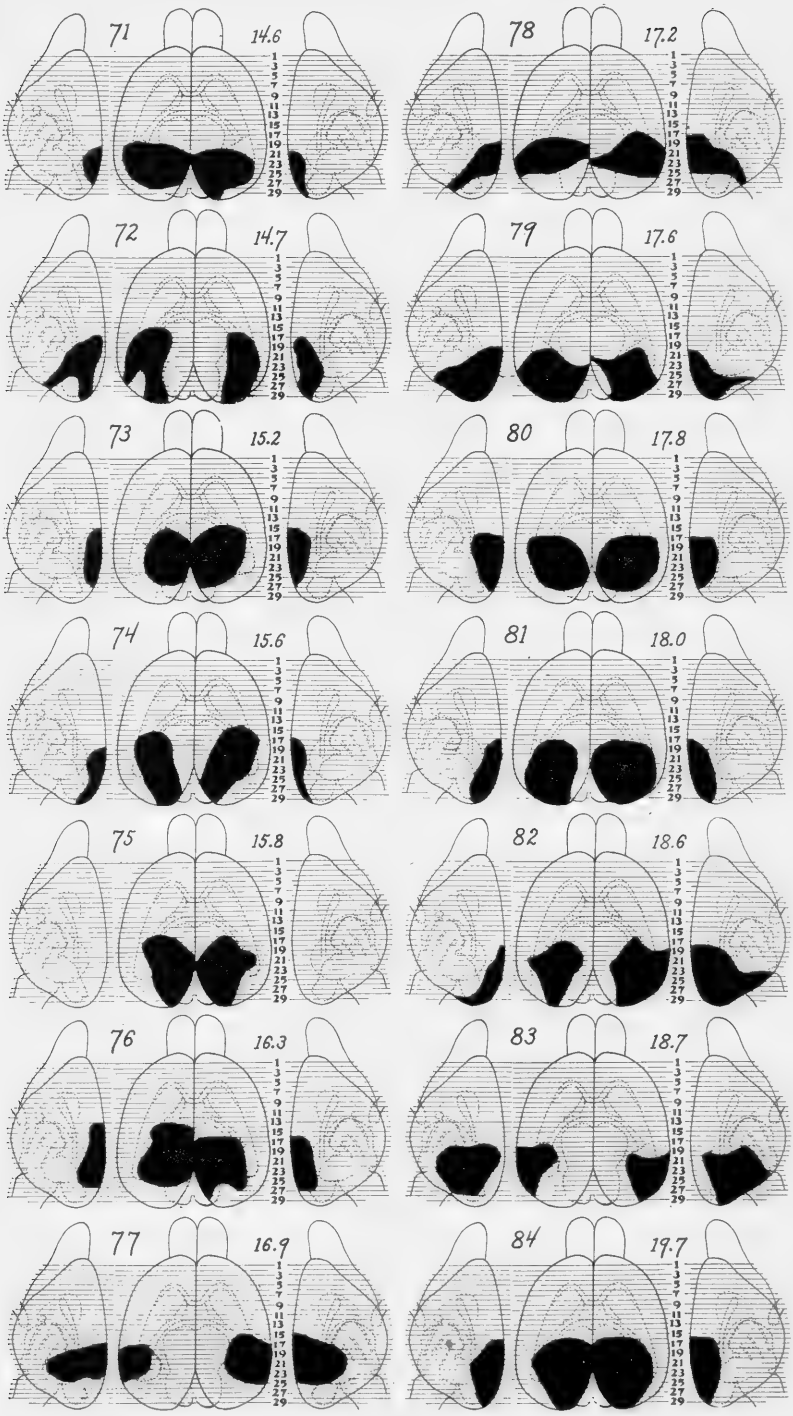
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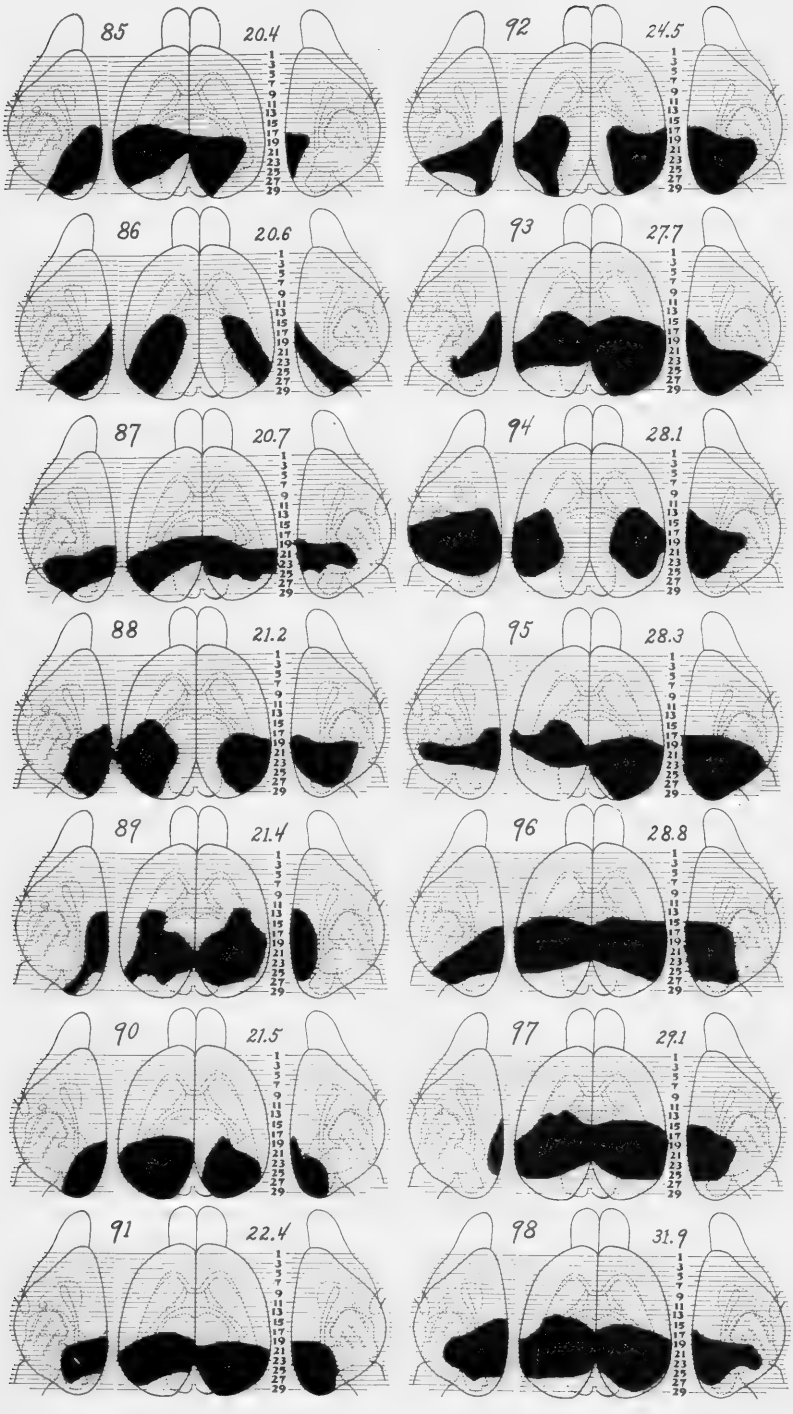
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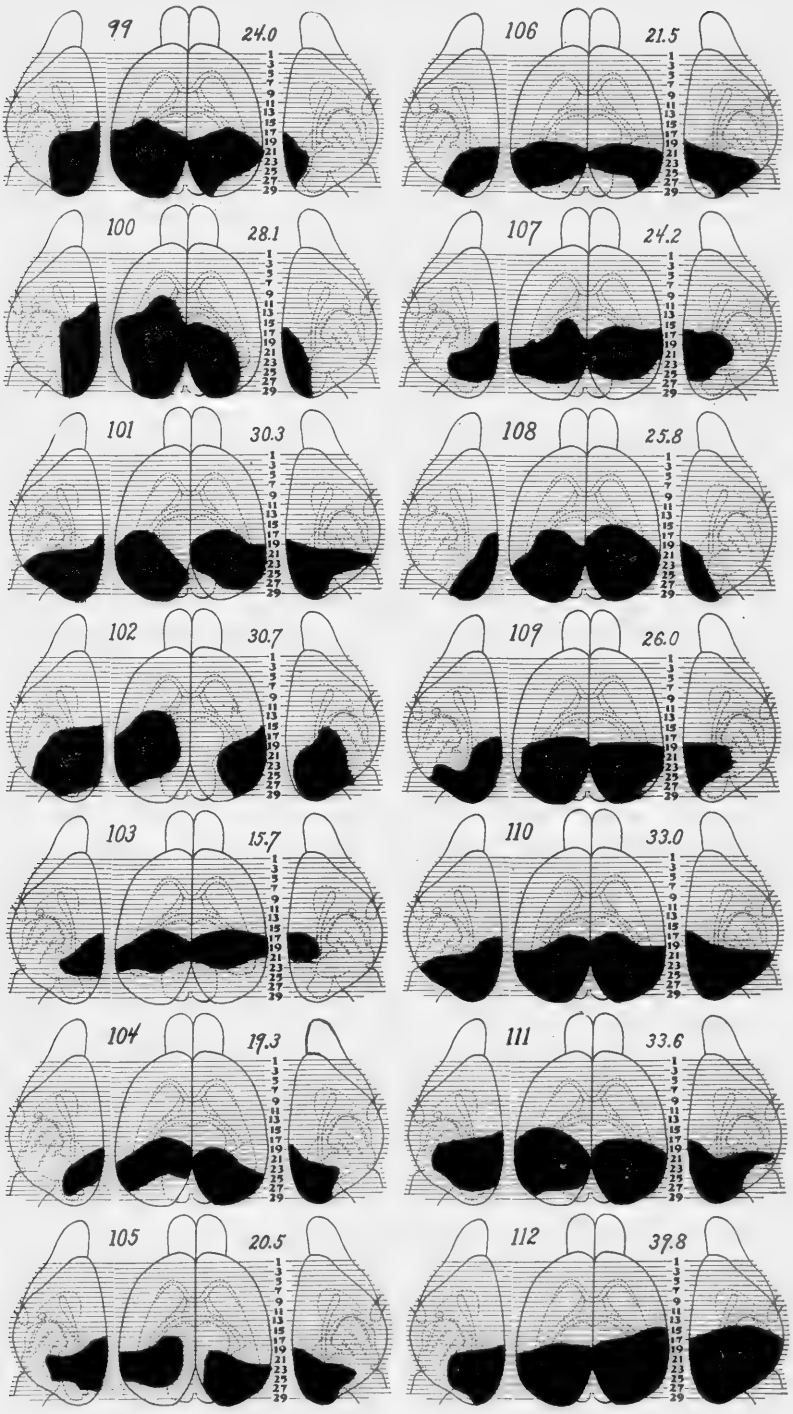
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