The Effect of Cerebral Destruction on the Performance of the White Rat in Various Maze Situations

by

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Abstract

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Shepard found that all rats which could master his unit-alike (u-a) maze (a series of standard units followed by a final unit, like the standard units except that the reaction necessary to avoid the blind is in the opposite direction from that necessary at the choice points of the standard units) can also learn his unique-unit (u-u) maze (layouts at all choice points alike; layouts of alleys from each choice point to the next, all uniquely different; about the same number of right and left reactions, distributed irregularly, required at the choice points). But he found a considerable number of rats which can learn the u-u maze but cannot learn the u-a maze.

The first question asked in this study was whether any locus or amount of cortical lesion could change rats able to learn the u-a maze into rats without this ability but still able to learn the u-u maze.

Seventeen rats learned the u-a maze after an average of 80 trials and 62 errors. Since Shepard so constructed his u-a maze that all but floor cues were controlled, and since he believes that the floor cue is one of resonance, probably auditory in nature, the lesions were largely focused on Fortuyn's area p, although almost all the cortex was covered at one time or another. The average lesion was 40% of the neocortex. With an average of 32 trials and 26 errors, all these rats were able to relearn the maze. This represented far less deterioration than Lashley found after comparable or smaller lesions in his seemingly simpler alternation maze III which was smaller and more readily learned preoperatively.

To check on this apparent discrepancy, the second question asked in this study was whether the same rats with the same lesions would show similarly

discrepant post-operative effects in the two mazes. Eighteen rats with locus and amount of lesion similar to the above again showed similar results in the u-a maze but much greater deterioration in the Lashley maze (56 trials, 63 errors before operation and 72 trials, 227 errors post-operatively).

Another question raised was whether the amount of neocortex removed was related to the post-operative deterioration in either maze. No relationship was found between amount of lesion and retention or relearning of the u-a maze, but a high correlation was found between amount of lesion and errors in relearning the Lashley maze.

Within the limits of this study, the question as to whether locus of lesion was related to postoperative disturbance in either maze could not be answered except that area p is not essential for the retention or relearning of the u-a maze, nor is the amount of area p left intact related to its degree of retention.

Some other rats, in addition to those reported above, were, for various reasons, rendered unable to relearn the u-a maze, but neither could they learn the u-u maze; in no case was a rat rendered unable to learn the u-a maze while retaining the ability to learn the u-u maze.

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HISTORICAL SURVEY

Historical Survey

As early as 1901, Franz began to publish his studies on the function of the cerebrum. Most of his early work was concerned with the formation and retention of associations, and simple sensori-motor habits. Here he rebels against drawing conclusions as to the nature of the function of the cerebrum by simple observation of animals following extirpation of parts of the cortex. In his effort to attain exactness of description he developed some of the training methods devised by Thorndike. "By the use of this method we may have animals, which are to be operated upon, form certain associations, the presence or absence of which can later be determined by the experimenter in a few moments." From these studies Franz concludes that the frontal operations severed the connections which made up the recently formed habits, but that these habits could be reformed in some other portion of the brain in the same time as that required for normal animals in the same situations.

In 1911, Franz made an attempt to determine the function of the visuo-psychic area,"believed by histologists to be closely connected with the calcarine area. Subsequent developments directed the inquiry into other channels and eventually it became imperative to reinvestigate the whole occipital lobe." His work up through the publication of this article indicated that "the histological differentiation had not the physiological import which the histologists imputed to it." This 1911 article deals largely with the relations of the lateral aspect of the occipital lobes in the monkey to certain sensory and perceptual states. He readily trained eight monkeys

to decline certain colored foods soaked in a solution of quinine, and to take only those foods which they had consistently experienced as not bitter(in some cases soaked in saccharine). The food consisted of cubes of bread that had been dipped into thoroughly saturated watery solutions of red, green, blue and yellow. He reports that the cubes were fairly homonymous, with no smoothness of surface. He is convinced that the reaction was to color, though his control of brightness is by no means convincing.

After relatively small lesions in the lateral parts of the occipital lobes, he finds"no interference in the color discrimination, nor any disturbance of a true visual character; but the operation is followed by disturbances in the coordination of movements which are based on sensations from the eye and its appendages(the eye muscles). These disturbances in coordination are not due to lack or loss of the true visual element, but to lack or loss of the afferent motor elements(intrinsic or extrinsic eye muscles)".

In 1915, Franz reported a study of the stimulable cortex of five monkeys. He found that the motor areas differ in size for different monkeys, and for the same monkey in the two hemishperes. He also found differences in the relative distribution of these areas for different monkeys and for the same monkey in the two hemispheres. He restricted his maps to the arm and leg segments and found that the arm segment proved to be the largest area, and the foot area the smallest, with the other segments between these two extremes in varying proportional sizes.

He believes that his findings, that the same motor cell can discharge in different manners, argues against the cut and dried chain reflex theory.

Franz and Lashley together embarked on a study of rats from the point of view of cerebral function. They wished to determine whether or not the rats could acquire habits after the removal of certain small or large parts of the cerebrum, but not after removal of other parts; or if they could retain but not acquire habits after certain cerebral destruction. They used as apparatus a simple one-choice maze, and in July of 1917 they published the results obtained from this maze for rats with lesions in the frontal lobes. Rats which were not overtrained required 29% less time for the first 15 trials after destruction of the frontal lobes, and made 53% fewer errors than they required for learning the maze before operation. Considering the fact that retention was tested in less than 24 hours after operation, there seems little loss that was not the result of the distracting effects of head bandages and shock. The overtrained rats, for whom training was continued for 200 trials after learning, required 87% less time for the first 15 trials after operation and made 90% fewer errors than they made in initial learning. "As a whole, therefore, the experiments show that in the white rat the removal of large parts of the frontal portions of the brain does not greatly interfere with a learned reaction. This is the more remarkable since it seems probable that the so-called motor area is in that region and that in most, if not all, of the cases

there was a destruction or abolition of the motor connections.The results also suggest that the habit reaction is not necessarily cortical in these animals.

In September of the same year, Lashley and Franz published another study of cerebral function in the rat. They used two sorts of apparatus: the above mentioned one-choice maze and the incline plane problem box. The simple maze habit was found to survive the complete distruction of the frontal third of the cortex. On the other hand, complete destruction of the frontal poles caused loss of the problem box habit, though the temporal and parietal areas were destroyed without injuring the habit. Some considerable portion of the poles must be preserved, but it may be any given portion. The habit was reacquired by some animals after almost complete destruction of the frontal poles. In the case of the simple maze, where they find the frontal poles are not necessary for the learning or survival of a habit learned before operation, they conclude that either the frontal lobe is not used, or it very soon ceases to function and something else takes over the habit. They find no marked disturbances after complete destruction of the stimulable areas of the cortex, but, in certain cases, marked hemiparesis seemed to result from destruction of the corpus striatum.

In 1920, Lashley published the results of further studies along the same lines. Again he used two sorts of apparatus. Instead of the incline plane box, he substituted

for it the double platform box, which seemed to be a fairer test for the comparison of operated with normal rats. The other piece of apparatus used was the Yerkes Discrimination box, which involves the simple visual discrimination of choosing a lighted compartment instead of a darkened one. He performed this experiment for the purpose of answering several questions, chief among which was the question: "Is any part of the cerebrum especially efficient in learning, irrespective of the type of sensori-motor coordination involved in the habit? No single part of the cerebral cortex seemed any more important for learning the double platform box than any other part. Some part, but not any specific part of the frontal cortex probably functions most normally in this type of habit, according to these results, but other regions may substitute if the frontal region is destroyed.The visual discrimination habit after its formation was not injured by destruction of either frontal or parietal cortex, but it was destroyed by the occipital operation; however, it could be reacquired in normal time.

Thus Lashley concludes that the occipital lobes normally function in the visual habit, but they are not necessary for the visual habit. As in the case of the double platform box habit, the operated animals prove to be slightly superior to the normal controls in learning ability. "Then learning at normal rate of either kinesthetic- or visuo-motor habits is possible after the destruction of any given part of the cortex.Destruction of cerebral nuclei

also has no effect on the rate of learning. Within limits from 100 to 50 per cent of the cortex there is no relation between the amount of cerebral material functional and the rate of formation of complex habits. Extensive lesions to one corpus striatum are accompanied by paralytic symptoms and it is probable that the stimulable cortex and the corpora striata have alternative motor functions."

It was in connection with the work on the double platform box that Lashley first suggested his theory of the equipotentiality of the cortex, for no single part of the cerebrum seemed necessary for the learning of the habit, and no single part more efficient in its learning than any other part. In his reply to Hunter's criticism of his interpretations, he defines equipotentiality as the condition in which lesion in any part of a cerebral area produced no measurable disturbance of function, whereas destruction of the entire area was followed by conspicuous loss of function. But, he asks, what becomes of the concept of sensory and motor projection areas if the entire cerebral cortex of the rat is equipotential in learning? In the case of the double platform box habit, he was not certain as to the kinesthetic projection area, nor of the exact function of the kinesthetic impulses in the behavior of the rat. Therefore he wished to obtain data on learning involving some stimuli which could be experimentally controlled, in order to determine whether there are definite areas corresponding to the sensory projection areas of higher forms, and to try to determine the relation of these to the habit formation.

For this purpose he selected the visual discrimination habit. This is also related to a question raised by the work of Franz. We have already mentioned his experiments on the frontal lobe. Here he was inclined to take the view that the frontal lobes acted as an association area, that they were functional in learning because it was learning, and not merely because they are the normal projection areas for some group of afferent or motor impulses. Lashley describes the experiments of Franz as dealing only with various kinds of latch boxes, all of which probably had a kinesthetic foundation. He feels that if Franz's view is correct, the destruction of the frontal lobes should result in the abolition of any complex, recently formed habit. But if the frontal lobes simply serve as projection areas, some sensory habit should be found which would survive frontal lobe destruction, and be eliminated by injury to other parts of the cortex. This, as we have said, he found to be the case for the visual discrimination habit. "The entire cortical mechanism of the perfected visual habit is included within the occipital pole of the cortex." In this particular publication, the data is not conclusive as to whether he removed all of the occipital cortex, when the habit was relearned. The study is merely generally indicative and does not settle the question. He also brings out the fact here that he finds paresis dependent on lesion to both stimulable cortex and corpus striatum.

In 1921, Lashley published two additional studies on cerebral function. The first concerns the effect of long continued practice on cerebral localization. He trained the

rats in the brightness discrimination habit, then added 1200 trials after learning was completed. They still lost the habit after occipital operation, proving to him that the habit did not drop to lower centers.

The other study was concerned with the motor areas. Here he finds that the caudate nucleus and motor area are not necessary for motor habits. For this experiment he used the brightness discrimination habit and found that the response does not go down by this path - the stimulable area and the caudate nucleus are not functional in the visual motor habit. A unilateral lesion of the motor cortex and caudate nucleus caused rotation toward the injured side. Lashley describes this difficulty as being not in voluntary movements, but rather in the formation of new patterns of tonic contraction. Lesions of the motor area produced some small degree of paralysis immediately after operation, but this soon disappeared. But where the caudate nucleus was concerned, the effect was quite permanent. Lashley says this was not really a paralysis but a motor incoordination. If both nuclei were removed, it showed not as motor disturbance but as sluggishness.

In 1922, Lashley published a fourth study on cerebral function in learning. This study is concerned with vicarious functioning after destruction of the visual areas. Here again he used the visual discrimination between light and darkness. The rats were trained after the visual area was removed. The combined lesions covered the back half of the hemispheres in the first operation; in the second operation, some other portion of the brain was removed. The combined extent of the second lesions covered the front two

outlet from the cortex for completely integrated movements, or movements acquired as a result of training(voluntary or conditioned reflexes). 2) In the intact animal the Betz cells are the principle centrifugal paths, but some neural impulses of like function may descend by extra-pyramidal tracts and these tracts may assume vicariously all the functions of the motor area. 3) The motor area is part of the mechansim functioning in the regulation of tonus and posture and not directly connected with the conditioned reflex act. The first theory Lashley says is ruled out by the evidence for habit formation after destruction of the areas. As a test of the other two theories, if after total destruction of the precentral areas the animal shows undiminished ability to carry out activities of all degrees of complexity acquired before operation, this proves that the lesion did not destroy any part of the conditioned reflex arcs involved and thus that these arcs do not traverse the motor areas. If the habits are lost but can be retrained, then the second view is true.

Lashley says that such a test has been made for the rat; he cites his 1921 study as unmistakable evidence against the participation of the motor areas in the activities of the simple maze and the visual discrimination box. He adds that since the publication of that study, these results were confirmed for a complex maze and the double platform box. As the rat shows no paralysis after lesion to the stimulable areas alone, he does not think it possible to generalize from the rat to higher forms which do develop a paralysis in such a case. Therefore, using monkeys, he destroyed the greater part of the precentral

gyrus of both hemispheres. He had previously trained the monkeys in habits of manipulation and visual discrimination. When the animals revived and recovered from paralysis, they showed perfect retention of the habits. He concludes that the so-called motor areas are not directly concerned with the performance of complex learned activities. The motor impulses of conditioned reflexes must descend from other areas of the cerebral cortex than the precentral gyri, which cannot be regarded as the source of impulses to voluntary movements. He adds that recovery from paralysis was not due to vicarious functioning of the corpus striatum. Lashley considers the precentral gyri as part of the kinesthetic mechanism for reflex control of spinal posture and maintainance of excitability of the lower motor centers.

His second study published in 1924 was an investigation into the theory that synnaptic resistance is reduced by the passage of nerve impulses. He blind-folded the left eye of the rat and trained it to avoid the brighter of two lights. Then he tested the same rat with the right eye blind-folded and the rat still showed perfect discrimination. He repeated the experiment after removal of the occipital cortex, so that no correlation between the eyes could be made, with the same results and concludes that there is no final common path for the two eyes.

On the motor side he refers to the results from the experiment on monkeys where the precentral gyrus was removed, with resulting paralysis of the left arm and leg. The monkey learns to open a lock with his right arm, but when the left arm recovers, it can be used to open the lock

almost perfectly.

In 1926, Lashley published a seventh study of cerebral function in learning. Here he was concerned chiefly with the relation between cerebral mass, learning and retention; and here he claims he found his first evidence experimentally of a relation between cerebral mass and efficiency, indicating that the cerebral lesion always produces some deterioration of function of the injured area, leading to the notion of mass action. This he defines as the theory that, within any given functional area of the cortex, the subordinate parts can individually perform any of the functions of the whole, though at a lower level of efficiency(applicable to certain functions only). This study he considers carefully controlled and as the most significant study bearing on the subject.

To test the influence of the extent of brain lesion on the learning and retention of reaction to brightness, over 100 rats were trained before and after cerebral lesions of varying extents and loci, and over 100 controls were used. The facts stressed in this experiment were:

1. There is a well defined visual sensory projection area on the occipital cortex of the rat(Fortuyn's w)

2. Lesions to the cortex which do not involve this area or its projection fibres have no influence on the formation of the visual habit or its performance.

3. Previous partial(including every possible part of the occipital third of the cortex) or complete destruction of the area does not influence the rate of initial formation of the habit.

4. Destruction of the whole field w, after training, results in total loss of the visual habit.

5. The habit can be reacquired as by normal untrained animals.

6. Incomplete destruction of area w results in errors in the post-operative retention test, and the amount of training necessary to bring the animal back to errorless discrimination is proportional to the extent of injury in the general region of area w, independent of the locus within the occipital third of the cortex.For retention of this habit, the different parts of field w are equipotential.

He further gives evidence to show that the loss is not due to shock, general or specific, nor to scotoma. If shock affects relearning, it should also affect learning, which it does not. As a partial check against the factor of diaschisis, he points out that the results are the same whether the wait after operation be 7 or 14 days. If the factor of scotoma interfered with vision in a trained animal, it should also interfere to a measurable extent with the vision of an untrained one, but does not. He thinks the loss is not due to a greater effect for large than small lesions as a large frontal lesion does not affect retention for the habit.Nor does he think the loss is due to a critical extent of lesion, less than which there is no effect, as the correlation between extent and loss holds in the case of small, medium and large sized lesions. He thinks the loss is not due to destruction of a focal point essential for the habit as he grouped the rats who showed good and poor retention and found that the combined extent of their lesions covered the same area, so that each would be as likely to hit the focal point. He thinks it

likely that the habit mechanism may be sub-cortical, but he thinks that this would leave his problem unchanged, for the final functioning in the normal animal would still be conditioned by the quantity of intact tissue in area w, irrespective of the locus of the tissue within the field. It was to explain these facts that he postulated the notion of cerebral mass action. Also in this article, he finds a suggestion that thalamic lesions disturb retention of the habit to a greater extent than do cortical injuries and to a degree proportional to the extent of the lesion.

He interprets the relation between cerebral mass and efficiency of retention as indicating a summation of the activities of different parts of the visual area, summation taking place in spite of the cutting of any particular group of association fibres. He is convinced that the theory which makes the conditioned reflex arc the unit of cerebral organization is inadequate, and an additional cerebral mechanism postulating much greater plasticity of action must be substituted.

In 1926, Lashley and McCarthy published an article which represented an attempt to test the theory that the proprioceptive system is very important in complex habits such as the maze. They used here for the first time the 8 cul de sac maze, observing the effect on this habit of cerebellar lesions. The lesions produced marked motor and postural disturbances, far in excess of anything resulting from cerebral lesion. They felt that the postural abnormalities must have greatly altered the pattern of kinesthetic excitation. Using "green" rats, they found an average of 59 errors required for initial learning, 3.8 errors for

pre-operative retention, and 6.3 errors for retention after cerebellar lesions. Also, after training two normal rats in the maze, their eyes were removed; they showed distinct retention. Then Lashley inflicted a cerebellar lesion and they found still a distinct retention. Even where the complete cerebellum was removed, although the rat needed more trials to bring his score up to criterion, he still learned the maze. Such rats cannot stand on their feet, but role and twist through the maze perfectly well.

In 1927, Liggett and Liggett found that rats with the olfactory lobe removed are not inferior to normals in learning the visual discrimination habit.The operated rats were more active than the normal, and destruction of frontal portions of the cerebrum slightly accelerated the formation of the visual discrimination h abit(confirmed by Lashley).

In 1928, Cameron published a study on cerebral destruction in relation to maze learning.His mazes were larger and of more complicated geometric pattern than the mazes previously used by Lashley, but the main difference was that Cameron's mazes were composed of unique units, whereas Lashley's most complex maze was simply a swing series. Cameron found that rats with relatively little destruction of frontal cortex were able to learn new problems in the maze, showed a certain amount of retention of old habits, and were able to adapt to simple modifications of already formed maze habits. But in all three activities the operated animals proved inferior to their normal controls. The beginning of the curves for both groups was strictly comparrable in contour, but the latter parts of the same curves was very different. The normal animals

in their curves rapidly reached the base line; the curves of the operated group continued to zig-zag on for some distance. This was marked for all three maze activities. He concludes that the frontal operation had a decided effect on the learning, retention, and adaptation to changes of the mazes. The effect seemed to him to be due not simply to removal of specific cortical components. He regarded the lesions as having disequilibriated an exceedingly complex dynamic system so that future adjustments could not be made with the same ease as was the case for normal cortical equilibrium.

In 1929, Lashley and Ball published a study in which they had trained 11 rats in the maze with 8 culs de sac, then subjected them to partial section of the spinal cord in the upper cervical region. A series of cases with complete interruption of the dorsal or lateral funiculi and one animal with partial section of the ventral funiculus were obtained. The ability of the animals to traverse the maze was then tested. In spite of serious sensory disturbances, some animals with each type of lesion traversed the maze without significant errors."It is not certain that all afferent paths were interrupted, but it is certain that these animals showed more pronounced defects of sensitivity to movement and to posture than has ever been observed after any cerebral lesion in any species". For Lashley, the most surprising result of these experiments was the capacity to orient in the maze after section of the descending tracts. He concludes that the motor impulses essential to the performance are not restricted to any of

the definite tracts, but descend either diffusely or over alternative paths in the different funiculi. Likewise, no particular afferent path of the cervical cord was essential for the performance of the maze habit. "After such lesions, the animals ran the maze accurately in the darkness when all other directive sensory cues seemed ruled out." In the light of Shepard's experiments in the maze, it is obvious that Lashley did not have adequate controls of all other directive sensory cues. Lashley concludes that the learning of the maze is essentially the establishment of an engram entirely central in nature, probably formed in the cortex, and not all over the body as the Behaviorists would have it. From this central connection there are any number of paths in and out. "The maze habit cannot be interpreted as a series of kinesthetic-motor reflexes but must be referred to some intraneural mechanism capable of producing an integrated sequence of movements in the absence of directive sensory cues.

Late in 1929, Lashley published his book, "Brain Mechanisms and Intelligence." Here he asks, "Can we find in animals a constellation of activities, correlating among themselves, having some of the attributes of human intelligence, and capable of modification through experimental control of nervous functions? If so, there is a chance for insight into the nature of the variable which is responsible for the correlations, for the neurological account of intelligence." In an effort to answer this question, he trained a group of rats in a variety of problems either before or after operation, with retention tests to determine the influence of lesions on previously formed habits or upon the

retention of habits formed after operation. Lashley thinks that the 8 culs de sac maze and the Yerkes discrimination box especially provide an adequate measure of some function which is affected by brain injuries. He feels that they give no indication of the fineness of this measure, but that it is a defect of all methods of evaluating tests. The rats were trained successively in the various problems of which maze III (the 8 culs de sac maze) was the first; thus the performance in this maze is a record of "green rats".

22 operated rats, whose combined lesions involved every part of the neopallium, completed the series of problems as well as 22 normal controls. For maze III, the operated rats required 6½ times as many trials as the normals, and not all rats on which this average is based completed the problem. On the other hand, the lesions produced no effect on the ability to form the visual habit in the same rats; in fact, the operated were slightly superior in this habit to the normal rats. As an additional check, 13 rats which had been used to test the influence of lesions upon the previously formed maze habit, were initially trained in the brightness habit showing a score slightly better than normal on the average, although, on the post-operative retention test for the maze they needed five times as many trials as the normal controls, showing distinct retardation.

From these results Lashley concludes:

1. The operated rats are significantly inferior to the normal controls trained under similar conditions in the learning and retention of a variety of mazes.

2. There is no evidence of any inferiority in the formation or retention of the habit of brightness discrimination. For Lashley these results justify the conclusion that cerebral lesions may produce a marked retardation in the ability to learn and remember some problems and at the same time leave the capacity with respect to other problems entirely unaffected. Again he offers what he feels is reliable evidence that the deterioration is not due to temporary shock or specific diaschisis. Chief among his evidence to this effect is the fact that rats trained initially in maze III after operation, when tested for retention after several other habits intervened, required ten times as much practice as did the normal controls.

Now Lashley considers the influence of the locus of lesion on retardation in learning. He has only two cases restricted to area p, the so-called auditory area, both of which are very small lesions, with an average of 6.5%. The rate of learning for these rats was not significantly higher than that of normals. "They are not adequate for a test of the function of area p, but they serve as control groups for areas which slightly overlap area p." Lesions in the so-called somesthetic region required more than four times the normal amount of practice. Lesions in the visual area required 6½ times the normal amount of practice. Those cases with lesions in both visual and somesthetic area required six times as much practice as the normal controls. Lesions in the motor area required 4½ times as much practice as did the normal controls. Thus lesion anywhere within these four

areas may produce marked retardation in learning maze III. Although Lashley recognizes the problem of the relative effects of lesions in different areas is complicated by the difference in size of the different fields and by the possible influence of the size of the lesion, and although he realizes that the number of cases upon which he bases his conclusions is small, he concludes after correction of the results that equal injuries in the different cortical areas produce equal amounts of retardation .

In the brightness habit, for all areas the results approximate closely the normal average."Results from the brightness discrimination habit must be distinguished from those that would result from pattern vision. The former probably deals with a more primitive or generalized mechanism. For the latter, something like a point to point correspondance between the retina and the cortex seems essential whereas nothing of the kind is required for brightness discrimination. The learning of the latter is independent of any part of the cortex, but when the habit is formed by the normal animal, it shows definite cortical localization" As we have said, the double platform box habit and the brightness discrimination habit are similarly affected by lesion in that both show definite cortical localization when learned by the normal animal.

For most of this series of problems Lashley finds some relationship between extent of cerebral injury and the amount of practice necessary for learning, which he does not think is due to spurious factors; the correlation is

best seen in maze III. Approximately equal correlations within the single fields further show that the retardation depends solely upon the extent of destruction, irrespective of the locus within the cortex. No such correlation appears for the brightness discrimination habit. Here Lashley says he thinks the difference lies in the complexity of the two habits. Herrick feels that the operated rats are superior to the normal controls in this habit because "the presence of uninjured cerebral cortex may actually retard the learning of a very simple habit by reason of the complicated processes through intercurrent cortical associations from other sensory fields." For normal animals, an increase in the number of culs de sac resulted in no disproportional increase in the difficulty of the problem for the rat; for the operated rat, increase in the number of culs de sac resulted in disproportional increase in difficulty.

Now Lashley investigates the relation between the extent of injury, the complexity of the problem, and the degree of retardation. He finds that the simpler problem offers difficulties which are not much greater for the operated than for the normal animals, and the difficulty does not greatly increase with increasing magnitude of the operation. But the more complex problem is more difficult for the operated than for the normal animals, and as the size of the lesion increases, the difficulty of the problem becomes progressively greater. This statement seems born out by the results of Cameron. But for the brightness discrimination and the double platform box habit, which

the normal animals required many more trials to learn than they did for the maze, and which on the face of things were seemingly much more complicated, there was no such deterioration. Lashley thinks this may be due to differences in sensory and motor components of the two habits, or that perhaps such habits are simpler than the maze in spite of the difference in the number of trials required for original learning.

"The inferiority of the operated animals in retention of maze III represents a real reduction that cannot be ascribed to defect in the learning mechanism." He thought it possible that the operated and normal rats forgot the same amount, but that the operated rats had a harder time regaining what they had lost. However, he finds that this is not the case, that there is a real reduction in retention as well as in learning ability, for the relative amounts of practice for relearning do not correspond to those for learning, and the operated rats start at a lower level of efficiency than they did in original learning.

From his curves of learning Lashley feels he can draw but one conclusion: that the course of learning is qualitatively the same for normal animals and animals with lesions of all varying extents; the rate of formation alone is affected, and this is somewhat proportional to the size of the lesion. Even those rats with the greatest cerebral destruction in the cortex are capable of continuous improvement through practice, though the tendency to stereotyped acts stops their progress at a level of efficiency

less than that of normal animals. This refers to retention of habits formed initially after operation.

In 1927, Lashley had found that there was a fundamental difference for the brightness discrimination habit between the effects of the operation, depending on whether the lesion was inflicted before or after training. If the animal is trained, then operated upon, the habit is abolished by any large lesion in the occipital third of the cortex, and the amount of retardation is proportional to the extent of injury in the occipital area (in terms of practice to relearn).But the practice then required never exceeds the upper limit of initial learning for normal animals. No other lesion affected the retention. If the rat is first operated upon, then trained, no effect on the learning of the brightness habit results even if the entire occipital third of the cortex is removed; the same applies for the frontal lobe with the double platform and the inclined plane box habits. Lashley finds a positive correlation between size of lesion and degree of amnesia irrespective of locus within the occipital region, for the brightness discrimination habit.

In the case of the maze, the initial learning has proven retarded by lesion anywhere; Lashley now tries to determine whether retention is also a function of the entire cortex.His conclusion for the brightness habit that within a functional area, the efficiency of performance is conditioned by the amount of nervous tissue available, independent of any particular area or association tracts, was tentative, and he hesitated to accept this radical conclu-

sion without a test which could decide conclusively between the possibilities of the results being due to sensory defects or mass function in the visual discrimination habit. He felt that the best way to settle this question would be repetition of the experiment with a different habit - a conditioned reaction to an auditory or olfactory stimulus, since there is little evidence for any spacially distributed anesthesias following lesions within cortical areas for these receptors, which could influence tests as might scotoma. But no satisfactory training technique was available for these habits at the time. He considered a kinesthetic-motor habit next suitable;"even though the somesthetic system has cortical spacial projection, the spacial arangement is segmental and there is no possibility that larger or smaller areas of anesthesia will proportionally interfere with the retention of habits involving somesthesis. Accordingly he used maze III. He trained the rats to ten successive errorless runs, then gave them retention tests after a ten day interval; then he operated upon the rats; ten days after the operation retraining was begun and continued until the rats made ten errorless successive runs or until they had made 150 trials. He had 59 cases with lesions from 4.5% to 50.9% of the cortex, covering in their combined extent all of the neopallium except some of the lower temporal region.

To test the visual factors in the maze, 12 other rats were trained; 6 were blinded after training, then tested for retention. The other 6 were blinded before operation or training; then they were trained, then subjected to

destruction of the visual areas of the cortex. Here he finds that lesions which in otherwise normal animals would seem to cause loss of the habit through production of cortical blindness actually produce the same effects in animals which were totally blind during the formation of the habit. This pertains to his study of the effects of sense privation upon maze learning, which we will consider later in greater detail.

The average amount of practice required for initial learning of maze III by the group of 59 rats was 54.5 errors and 24.8 trials. In preliminary retention they required 3.8 errors and 3.5 trials. In post-operative retention they required 16 trials on an average and 247.4 errors. The average per cent of destruction for the group was 17.7. Serious disturbance of the habit was rare in any case with less than 15% destruction. Lashley feels that his evidence on the whole is opposed to localization of the habit in any part or parts of the cortex."In both initial learning, then, and post-operative retention, any lesion over 15%, irrespective of its locus, may produce loss of the maze habit. In both cases the evidence is inadequate in respect to area p."

He finds a significant relationship for the maze between the amount of destruction and the severity of amnesia, directly referable to the lesion. "It is less certain, though probable, that all parts of the cortex participate equally in performance of the maze habit, and lesions of equal size produce equal loss of the habit, irrespective of the locus".

And now Lashley analyzes the sensory and motor requirements of the maze and the general behavior of the operated animals for clues as to the relative importance of sensory and higher level processes in production of retardation. "Failure to find a constant dependence of the habit on any locus or field points to a lack of dependence of the habit on any single sensory function. If, for example, maze learning is not reduced by total blindness, we should not expect it to be disturbed by injuries within the cortical visual area unless this area has some other than simple visual function." We have seen that the brightness discrimination habit was lost after occipital lesion to rats blind during original learning, and lost to the same degree as for rats with normal vision. Also we have seen that a habit involving a specific receptor can be formed at normal rate after destruction of the cortical field for that receptor. Here Lashley summarizes his evidence on the effects of sense-privation and finds that for vision, olfaction, proprioception and motor defects, destruction of the sensory projection area produces a disturbance of function from 9 to 60 times as great as did destruction of the corresponding peripheral mechanism."And this lack of effect of sense-privation is not due to shift to the use of other sensory directive cues." However, he did not control the other directive possibilities sufficiently well to justify such a statement. He concludes that retardation of the formation and retention of the maze habit is not due to sensory or motor defects but to disorganization at a higher level of integration.

Notes which he kept on the behavior of the rats in the maze reveal to Lashley no clear indication of specific sensory defects or loss of specific elements in the habit."An occasional animal seemed to have difficulty with the right-left alternation in the maze, though they had definite general orientation to the food. With others the trouble was more with the general orientation, but neither case occurred consistantly, nor was related to locus of lesion. Four types of post-operative behavior were observed: (this concerns post-operative retention)

a) The animal behaves very much as does a normal one in initial learning. He moves rather slowly, explores all parts of the maze thoroughly with few repetitions of errors, and gains a general orientation quickly, so that he back-tracks only infrequently after the first two or three trials.

b) The animal seems to wander aimlessly in the maze. His movements are slow, or not faster than normal. He does not enter any cul de sac repeatedly but moves back and forth through rather large sections of the maze. He acquires general orientation slowly, tending to back-track after many trials.

c) The animal tends to repeat the same error time and again. He usually runs more rapidly than the normal and usually dashes back and forth in one alley many times before passing through the doorway to the next. This is not due to sensory defect and failure to be stimulated by the doorway, for many of the animals pause and thrust their heads through the openings each time they pass, yet they

continue the perseveration in one alley; others continually retrace a complicated path through several alleys.

d) The animal is disoriented and behaves very much as those in group b, but runs at high speed. He does not repeat the same error as do those in group c."

He found no correlation between these types of behavior and locus of lesion, but he did find that perseveration and rapid running usually went with the more severe deterioration, as measured in trials for relearning. The impression he received from almost all the seriously degenerated cases was one of more or less general disorganization involving both specific reactions to all parts of the maze and general orientation to the food. The behavior of the animals with initial learning after operation was the same as the behavior of the post-operative retention cases. Animals which had learned the maze completely after operation showed greater loss after 40 days than did normal animals, but no such difference was evident for the brightness discrimination habit. He concludes that it cannot be a question of sensory deficiency.

He noticed peculiar eating habits in the cases of the rats with extensive lesions. They did not grab the food in the food box, nor were they aggressive in competition with other rats for food."The whole picture was one of deterioration rather than of a loss specific for the maze. It seems clear that the problem is to explain the lack of retardation in other situations rather than the retardation in the maze."

In this connection he mentions several experiments

in progress at the time, the results of which are later reported in his reply to Hunter. These results indicate that with increasing complexity of the discrimination problem, a retardation appears which was not evident in the simpler discrimination problem:

1) After complete destruction of area w, the ability to distinguish between two lights of unequal intensity is retained, but the threshold is usually higher and the rate of formation of habits involving discrimination of slight differences is slower than for the normal.

2) Habits based upon the discrimination of small differences of intensity seem to be abolished by lesions which have little effect on the light-darkness habit.

3) Complete destruction of area w abolishes permanently the capacity of the animals to distinguish visual patterns, but leaves almost unaffected the capacity to distinguish position and relative brightness of objects.

Lashley concluded, after analysis of the available data, that the more complex the problem to be learned, the greater the retardation produced by any given extent of lesion. Additional evidence is presented which indicates to Lashley that the interruption of association or projection paths produces little disturbance of behavior, so long as the cortical areas supplied by them remain in some functional connection with the rest of the nervous system.

Lashley draws the following inferences from the facts presented in this book:

1. The learning process and the retention of habits are not dependent upon any finely localized structural changes within the cerebral cortex. The results

are incompatible with theories of learning by changes in synaptic structure, or with any theories which assume that particular neural integrations are dependent upon definite anatomical paths specialized for them. Integration cannot be expressed in terms of connections between specific neurons.

2. The contribution of the different parts of a specialized area or of the whole cortex, in the case of non-localized functions, is qualitatively the same. There is not a summation of diverse functions, but a non-specialized dynamic function of the tissue as a whole.

3. Analysis of the maze habit indicates that its formation involves processes which are characteristic of intelligent behavior. Hence the results for the rat are generalized for cerebral function in intelligence. Data on dementia in man are suggestive of conditions similar to those found after cerebral injury in the rat.

4. The mechanisms of integration are to be sought in the dynamic relations among the parts of the nervous system rather than in details of structural differentiation.

Hunter has criticized Lashley's interpretation of his data severely in that the mass action theory was derived almost solely from studies of the maze. He goes on to say that the maze is learned in terms of stimuli from many sense fields and the elimination of one or many of these reduces the animal's capacity to learn the maze proportionately. Hunter thinks the lesions affect the learning ability by reducing the functioning of one or

more sensory projection areas.

In reply to this criticism Lashley points out that the first experimental evidence of a relation between mass and efficiency he obtained from the brightness discrimination data, the 1926 study, and this he still considers as the most significant experiment bearing on the problem. But because he feared some spurious correlation inherent in the Yerkes discrimination problem box, he cast about for another method which might differ enough to render a common source of error unlikely. "e reluctantly adopted the maze as the only other technique available,"with full realization of its shortcomings". Here he found a quantitative relationship between extent of lesion and efficiency both in learning and post-operative retention. He adds that Hunter seemed to have overlooked his tests of the possibility that lesions produce sensory disturbance in proportion to their size, so he reviews tham since his conclusions as to mass action in the maze is based on these more than on any other consideration:

1. A group of normal animals was trained in a maze, then blinded and tested for retention of the maze. They made an average of 6 errors in relearning. Animals with destruction of the visual projection area made an average of 353 errors in relearning.

2. The somesthetic paths of the spinal cord were transected in the second cervical segment. These animals made an average of 16 errors in post-operative retention. Animals with lesions in the somesthetic areas of the cortex made 145 errors in relearning.

3. Animals were subjected to cerebellar lesions which produced marked postural disturbances, far in excess of anything resulting from cerebral lesion. Their postural abnormalities must have greatly altered the pattern of kinesthetic excitation as well. They made an average of 6 errors in post-operative retention tests. The dorsal funiculus, including the pyramidal tract, was severed in four animals in the second cervical region. They made an average of 6 errors in post-operative retention tests. Animals with lesions involving the motor areas, with some invasion of the somesthetic areas, made an average of 188 errors in post-operative retention tests.

He offers three alternative interpretations of these facts:first that the greater defect after cerebral lesion may be due to involvement of more than one sensory field in every case; second that the sensory loss from cerebral lesion is greater than that resulting from sense-privation; third that the loss of the habit after lesion is due in large part to disturbance of some function other than cortical sensory reception, the unknown function which he calls mass action. The first two interpretations he rejects on the basis of what seemed to him adequate evidence. As evidence for the third he cites the cases of the animals blinded, trained in the maze, then operated upon, showing fully as great a loss of the maze habit as the animals trained with normal vision, with similar lesion. He also adds the following data:

1. Linear lesions which interrupt a large proportion of the optic radiations have an effect upon the

habit of light-darkness discrimination which seems proportional only to the amount of cortex destroyed and not to the area deprived of sub-cortical connections. This is not true for pattern vision, but this fact only complicates the problem without altering the question of the mass relation in brightness vision. The same relatively small effect of linear lesions interrupting large numbers of fibres appeared in the maze study but is as yet less well substantiated. I do not consider this minor effect of linear lesion as an established fact, and I am continuing the study, but the present evidence for it and its possible fundamental bearing on the neural organization cannot be ignored.

2. With the Miles elevated maze, in the learning of which vision probably plays an active part, the learning of animals with occipital lesions was more greatly retarded than that of animals with lesions in other fields. Since no such difference appeared between the same animals in learning the enclosed maze, I accepted this as in accord with the assumption that visual defect after lesion in area w is not the primary cause of retardation in the learning of the enclosed maze.

3. Though all sense modes probably contribute to meze learning in the normal animal, there can be little question but that some are more important than others. But when correction was made for the different extents of lesion in the different cortical fields studied, the amount of retardation in maze learning was the same for each of the sensory fields, within the limits of accuracy of the experiments.

4. Throughout his discussion, Hunter stresses the role of sensory elements in learning the maze. In my discussion I have dealt with efficiency in performance after learning chiefly, and intentionally, for it is here that the crucial test must be made. It is obvious that animals do make use of sensory cues in learning the maze, and that they depend on cues for much of their running. But that these sensory cues are the sole determining factor in the maze running is less obvious, and the data on the rate of initial learning are not relevant to the question. Lashley cites Lindley's work here, which Hunter offered as a consideration opposed to Lashley's theory. "Lindley used a different maze from mine, but one which was almost certainly more difficult than my maze III, whose simplicity Hunter emphasizes. Lindley discontinued training after 30 trials, and his blind-anosmic group largely failed to finish by this time, but we may compare the excess of errors made by this group in 30 trials over his normal controls with a similar record of the first 30 trials of my group with lesions in visual and olfactory centers. The animals with occipital lesions were certainly neither totally blind nor anosmic, yet their learning was nearly twice as greatly retarded as that of the totally blind and anosmic rats.

So wherever we have data for a comparison of the effects of sense-privation and destruction of corresponding sensory projection areas, the latter proves by far the more serious disturbance in the maze habit. I have, I think, ruled out adequately the factor of severity of shock or

of diaschisis proportional to the size of lesion, and of the existance of a small association area more likely to be invaded by large than small lesions, as well as invasion of sub-cortical centers. It might still be argued that all my cases with cerebral lesions suffered from defects senses other than those whose projection fields were directly involved. Such a condition might arise if there were a general cortical depression(shock or diaschisis) or if all senses were to some extent represented in all fields. The former I have ruled out; the latter involves a view of sensory projection areas too radical and substantiated by no anatomical or physiological evidence."

In 1931, Jacobson published a study that represented an effort to test the influence of removal of the frontal and prefrontal areas in the monkey on learning and retention. He trained monkeys before and after operation in a variety of problem boxes and, during the interval between preliminary and post-operative retention tests, trained them in a simple pattern discrimination problem. The forms, pieces of apple, were placed under glass to eliminate possible olfactory factors; quinine sulphate was spread on the under side of the forms to be rejected. He obtained the following results:

1. Injury to the frontal and prefrontal areas has no effect upon retention of simple problem-box habits nor upon the retention of pattern discrimination habits. Injury to these areas did, however, result in impairment of those problem box habits which involved integration of several simple latches in one box. Such failures are

not peculiar to frontal lesions, however, since destruction of the parietal areas produces a similar loss .

2. Injury to the frontal and prefrontal areas did not impair learning ability for the simple or combination boxes. There was no impairment of the ability to learn the pattern discrimination problem.

3. In so far as these problems measure, among other factors, the animals' intelligence, it is argued that the frontal lobes are not the organ of intelligence. The data support the interpretation that intelligent behavior is a product of the equilibriated action of the entire nervous mechanism.

4. The localization of a premotor area, anterior to the precentral sulcus, which functions in the acquisition and retention of skilled acts, is questioned.

5. No sensory disturbance accompanied frontal injuries. Increased restlessness and general activity were noted after frontal destruction. No permanent emotional changes were found.

In 1931, Loucks published a study on the efficacy of the motor cortex of the white rat in delayed alternation. Two classes of rats were trained in a 15 second delayed alternation series of ten choices, in which they fed for the first five seconds of each delay: animals trained prior to extirpation of the frontal motor cortex, and subsequently retrained; animals receiving their initial training after extirpation of this area. The alternation box was so devised that successive choices, separated by delays, could be obtained without handling the animal at any time during a run.

"Extirpation of the cortex in the frontal motor region prior to training resulted in an initial learning time significantly greater than the original learning time of normal rats. Eleven rats, from a group of twenty-five receiving their initial training subsequent to operation, had lesions ranging from 17 to 36 per cent of the neocortex, and failed to show any indication of ultimate mastery within a training period three times the median learning time of normal rats. The effect of frontal destruction is still more marked in rats receiving their initial training prior to operation. Eighteen of twenty-three animals trained prior to extirpation subsequently relearned the problem, but with a significantly longer time than in initial learning. Their lesions ranged from 9 to 30 per cent of the total neocortex. This inferiority is also borne out in a comparison with rats given their initial training period subsequent to operation, which have significantly greater lesions, and yet take no more time in their initial learning than these rats require for relearning. Five of the animals trained prior to operation had lesions ranging from 16 to 42 per cent and failed to show indications of ultimate mastery, within limits of the experiment. With regard to the general level of performance it seems better never to have learned than to have learned and lost.

Rats trained prior to extirpation gave post-operative performance that correlated with size of lesion to the extent of .54. Learning scores of rats receiving their initial training subsequent to operation gave neg-

ligible correlation with extent of destruction. Clustering of cases makes this last result ambiguous. It appears that cortical destruction, within the limits present in this study, does not directly produce a stereotyped response. The degree of asymmetry found in this study bears no significant relation to measures of performance.

Comparisons of observed performance and that predicted upon a chance basis indicate that rats have a marked innate capacity for delayed alternation. Frontal extirpation seems to reduce rather than entirely wipe out a significant innate tendency to alternate in those animals receiving their initial training subsequent to operation. The effect of extirpation upon rats trained prior to operation is to markedly increase the number of zero alternations or stereotyped responses. Performance curves indicate a positively accelerated rate of change.

Gross bodily orientation is definitely ruled out as the explanation of the delay apparatus in the present situation. That implicit muscle patterns play a fundamental role is also somewhat improbable in view of the fact that a considerable degree of muscular relaxation, induced by a general anesthetic, did not disrupt the alternation habit. By the method of residues it is deduced that the basis of the habit must be a neural trace - probably 'central'. The present study gives several indications that the limits of the rat's capacity in previous studies are a product of the particular setting utilized.

Destruction of the frontal motor region in rats produces a significant inferiority of performance when the

problem is adequate."

In his discussion, Loucks asks what constitutes an adequate indicator of cortical activity. "Where extirpation brings positive results, the problem is simplified. The proof of the validity lies in the results. Though we may not be able to ascertain whether the disturbance registered by the test is direct or merely secondary, yet we would then have indubitably established the fact that one aspect of the indicator mechanism was cortical."

In 1931, Lashley published another study on cerebral function in pattern vision in the white rat. This study is concerned with the ability to apreciate and discriminate position, distance, and a variety of visual patterns."Destruction of the entire anatomical visual cortex abolishes the capacity to react to differences in visual pattern, but leaves the ability to distinguish the position and relative brightness of two objects, and apparently leaves some discrimination of distance. Destruction of a small area in the lateral margin of the visual cortex produces a similar disability. Destruction of other parts of the visual cortex produces slight and inconsistant disturbances of pattern vision. Destruction of the optic radiations abolishes all reactions to visual objects but leaves the capacity to distinguish light from darkness. Destruction of the motor, somesthetic or auditory cortex or of the supposed visual association area does not appreciably affect capacity or the rate of formation of visual habits. The results are consistant with the assumptions that discrimination of light intensity is is a sub-

cortical function, that identification of the position of gross objects may be mediated by projection fibres scattered to all parts of the cortex, that vision for patterns or figures is conditioned by a small cortical area which probably represents the cerebral projection of the retinal fixation point, and that habits of reacting to complex visual stimuli may be formed without transcortical association with other sensory or motor fields.

In 1932, Lashley and Frank offer a correction of Lashley's 1931 statement that the capacity to distinguish simple visual patterns might be retained after destruction of large parts of the striate area in both hemispheres, so long as the small lateral area described remained intact. They now present evidence that the apparent importance of these lateral areas was due to the interruption of the optic radiations and not to destruction of cortical tissue.

"We cannot yet interpret these data with assurance. It is possible that limited destruction within the striate areas of the rat produces limited areas of scotoma, with normal vision in the remaining parts of the field. Or it may be that the condition resembles that described by Minkowski for the dog, where destruction of more than half of the striate area seemed necessary to produce permanent scotoma."

Earlier in 1932, Lashley had found evidence that there is a physiologically dominant fixation point in the temporal retina of the rat. "If definite scotomas result from partial destruction of the striate area, we should expect to find changes in fixation, correlating with the position of the lesion, with especially noticeable symp-

toms following destruction of the cortical projection field for the temporal retina. We have been unable to demonstrate any changes in posture of head or eyes during visual tests, indicative of fixation with any other part of the retina than the normal temporal region in these animals. This suggests an even more diffuse cortical representation of the retina in the rat than in the dog.

In 1931, Buytendijk published the results of operations on 20 animals in the motor centers, central and frontal. He observed no paralysis under normal conditions, but when he forced the rats to walk on a narrow piece of wood, incoordination became evident. He attributes this to destruction of the unity of the cortex which governs configurations as a whole. The perceptions and movements were no longer coordinated; the movements were slower and more rigid."This shows up more if the rat is placed in a more complicated situation. Food was placed in a small glass tube; the operated rats were unable to get to it." Also he tested the rats on an elevated three piece path. "The normal rats grasped the situation and acted stably, whereas the operated rats had stronger inhibitions, were confused, slower, and took much longer to reach the food." Then he set up a situation where three jumps were required, and found that the operated rat hesitates before each jump, and again takes longer than the normal rat. This apparatus was set up in front of a window. When he changed the direction of the jumps so that the light came from the other side, the operated rats had much greater difficulty in adapting to the change.

He also set up an elevated T-form, with food placed at the end of the right choice. He found an abrupt fall in the normal curve and concludes that the habit is not the result of a progressively formed association, but is a matter of sensori-motor intelligence. He finds also that the normal rat can readily adapt to changes in the direction of the correct choice. The operated animals he found could learn the T-form habit but could not adapt to changes in the direction of the correct choice. He finds individual differences not based upon the extent of lesion.

He set up still another apparatus, the 'double road' experiment, which he elevated so that the rat could use vision to orient himself. Both roads led to the food, but one was much longer than the other path. He forced the rat to traverse the longer path by blocking the shorter; then for one or two trips he blocked the longer and thus forced the rat to take the shorter path. Then he placed the rats at the starting point, with both paths open. He found that the normal rat chose the most direct route, but that the operated rats, especially those with the larger lesions, chose the longer path, which they had experienced originally and more often.

In 1932, Maier published the results of a study of cerebral destruction in the white rat. He postulated two types of behavior integrations: first, those made up of isolated experiences, and those made up of continuous experiences, R and L respectively. He interpreted R to be the ability which makes possible organization of past experiences in terms of a goal or end. The ability L

he interpreted to be the ability to organize past experiences in the order in which they were experienced. "It is thus reproductive in nature and makes for stereotyped behavior; ability R makes for new types of behavior." Tests involving these two types were devised. He also tested R minus L and R plus L. He found both R and L to be realities in 20 normal and 34 operated animals, and he found R, reasoning, to be qualitatively different from L, learning. By obtaining scores for R minus L and R plus L, he calculated values for R, which calculated values corresponded very closely with the experimental value for R (obtained from an entirely different set of data) in the cases of the groups of rats which made better than chance scores. The lesions were confined to the front half of the cortex, ranging from 7 to 41 per cent of the cortex, the average per cent of lesion being 22 per cent. "The calculated value for R as well as the experimental value decreased with an increase in the extent of the cortical destruction, showing that ability R depends on the amount of intact cortical tissue. The calculated value for L, however, remained constant and was not dependent upon the intact cortical tissue within the limits studied. The scores on tests involving R as well as the calculated value for R showed a sudden drop when the lesions exceeded 18 per cent of the cortical surface, indicating that there was a definite amount of cortical tissue which was necessary for the functioning of ability R. Operated rats showed a much greater tendency

to repeat errors than did normal rats. Small lesions involving less than 10 per cent of the cortex reduce the number of successful solutions but do not eliminate abilities R and L."

Later in 1932, Maier published a study of cortical destruction of the posterior part of the brain and its effect on the reasoning ability in rats. This study confirmed the results of the study of the function of cortex in the anterior part of the brain. He found the following results: "Small lesions to any part of the cortex have little effect on reasoning ability. Lesions larger than a certain critical mass completely eliminate the ability to pass the tests. Mass rather than locus determines what the effect of a lesion will be, but there is no evidence of a continuous function in the sense that lesions of increasing size show proportionately greater effect. Rather the effect of the lesion seems to be more nearly of an 'all-or-nothing' character. In this respect the relationship between cortical mass and reasoning ability differs from that between cortical mass and maze-learning ability. None of the lesions destroyed the ability to learn mazes, but all of the lesions increased the number of trials necessary for learning. Further evidence of a qualitative distinction between reasoning and learning ability is thus brought out. Lesions in the motor areas produced slight motor incoordination which became obvious when the rat turned corners on an elevated pathway. A comparison of the effects of cortical lesions in the anterior and posterior parts of the brain indicates

that for reasoning these two parts of the brain are qualitatively equipotential but quantitatively unequal in potentiality."

In 1932, Wiley published a study of the function of the brain in audition. He used a T-shaped apparatus in which the rat was trained to turn back when the buzzer was sounded and to go on if it was not sounded. Wiley had been unable to train the rats to go toward the sound. He draws the following conclusions:

1. The cortex of the rat plays a part in the formation of a simple auditory habit.

2. The function is not determined by the entire cortex but is limited to the postero-lateral part of the cerebrum.

3. The region involved in audition is closely delimited by Fortuyn's area p.

4. Large destruction in the auditory region seems to cause complete loss of the habit, but the habit can be reacquired.

The data show that there is a relationship between extent of lesion and degree of post-operative amnesia. It remains to be seen what will be the effect on the formation of the auditory habit if the auditory cortex (?) is removed before training. Within the limits of his experiment he finds that area p is related to his auditory habit as Lashley finds area w to be related to the habit of brightness discrimination in the rat.

"It is possible that the cerebral cortex outside of area p may assume this function. Pavlov in 1927

said he believed that the temporal region of the dog's brain is the acoustic analyzer, but that other cortical regions, and probably all of the cortical tissue, receive auditory impulses. If such is the case, then the remaining cortical tissue may carry out the auditory functions. It is just as possible that subcortical centers can carry out this simple function. Herrick ('26) in his discussion of Lashley's work on the function of the cortex in the habit of brightness discrimination, is inclined to believe that the habit, after loss of the cortex, is thalamic rather than due to a vicarious functioning of the cortical tissue. Our evidence shows only that if the cortex in area p is present, it plays a part in the formation and retention of the auditory habit.

"Area p seems to be a region for the reception and transmission of those auditory impulses to which the rat is capable of responding. Also, this region seems to act as a facilitating mechanism in the formation of the habit, as is suggested from the fact that two animals with large destructions in area p, required more trials to relearn the habit after the operation than any animal required to learn it originally. Lashley found that none of the animals in which the visual cortex was destroyed required more trials to relearn the habit of brightness discrimination than any normal animal required in learning. This difference suggests that the subcortical auditory mechanisms are, for the functions studied, more dependent upon the cortex than are the subcortical mechanisms which function in brightness discrimination. For pattern vision, on the contrary, Lashley

has found that the cortical mechanisms are absolutely essential. It seems possible, therefore, that the subcortical mechanisms are more dependent on the cortex in some habits than in others.

"The correlations found show a relationship between the amount of tissue destroyed and the ability to relearn the habit when the destruction of the tissue occurs in or near area p. The highest correlation between the extent of the injury and any measure of postoperative retention is o.63."

In December of 1932, Lashley, McDonald and Peters published the results of a study testing the effects of dilatation of the lateral and third ventricles on the rats' ability to learn the maze. Artificial enlargement of the ventricles to as much as 60 times their normal volume by injection of oil was followed by only slight retardation in learning, which is scarcely greater than would be expected from the damage to the cortex by the injecting needle. The records of 22 animals with spontaneous hydrocephalus were not significantly different from those of others having equal amounts of cortical destruction and no hydrocephalus. The evidence rules out trophic disturbance arising from mechanical distortion of tissue or increased intracranial pressure as factors in producing correlation between extent of lesion and efficiency in learning.

In 1933, Lashley and Wiley published a study of mass action in relation to the number of elements in the problem to be learned. "Measurements of the influence of extent of cerebral lesion upon efficiency in various

functions have given quite different results, according to the functions studied. In some cases there is clearly an all-or-nothing relation between some functional area and the capacity for performance; in others, a close relationship between surface extent of injury and degree of lowering of efficiency." This bimodality of distribution is consistent for all the studies pertinent but because all of the constants were determined from relatively small numbers of cases Lashley felt that they did little more than attest the importance of the mass relationship, giving little further information concerning it. For this reason Lashley and Wiley embarked on a study to collect a larger mass of data which could be analyzed with some assurance of reliability.

Accordingly, 127 rats with cerebral lesions and 60 normal controls were divided into four equal groups, each of which was trained on one of four mazes with four, eight, twelve, and sixteen culs de sac. Subsequently, all the 127 rats were trained in the Lashley maze III. The former group of mazes were of the general plan of the Lashley maze III, "which plan has the advantage that the number of blind alleys can be increased indefinitely without introducing any fundamental change in the general plan".

The normal and operated groups were compared with respect to the relative difficulty of the four mazes. The operated animals were markedly retarded in all mazes, but the relative difficulty of the simple and complex mazes was the same for them as for the normal controls. Thus he corrects his conclusions as to the factor of complexity

made in his book, "Brain Mechanisms and Intelligence", which conclusions he considers based on results statistically unreliable. "The problem of relative difficulty where qualitative differences are introduced remains unsettled. We should expect to find that a battery of tests of qualitatively different tasks graded in difficulty and making up an apparently continuous series for normal individuals, would show disproportionate difficulty at the higher levels for demented individuals in accordance with the severity of the dementia. Comparison of the results with the qualitatively different tasks used by Cameron, Lashley and Maier suggest that this is actually the case for animals with cerebral lesions. But extensive experiments will be necessary to establish the point and to reveal the nature of the effective qualitative differences, if they exist. For tasks where difficulty is determined chiefly by reduplication of similar elements our data seem conclusive. The difficulty increases at an accelerating rate with extent lesion, but the relative difficulty of the several tasks remains the same."

Upon analysis of the results for the Lashley maze III the following conclusions were drawn:

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

2. It has been impossible to detect any influence of small injuries in the archipallium or thalamus

(when combined with extensive cortical destruction) upon the maze scores.

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

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

"For the motor, visual and somesthetic fields, the training records are essentially equal, the differences between the groups being less than their P.E., whereas all make approximately 5 times as many errors as do normal rats. The record for cases in field p is not consistent with the others, but significantly higher than the score of the normals, though significantly lower than the other areas. So it looks as if area p were relatively less effective than the other areas in effect upon capacity to learn the maze, though area p also produces significant deterioration of the function. But even with this large number of cases (13 in area p with an average lesion of 16%), statistical reliability is low, and data are inconclusive as to the exact equivalence of the various parts of the cortex for maze learning, though difference between area p and the other areas is probably significant."

The extent of the lesions in this study ranged from 1.3 to 60.2 per cent of the cortex. The subcortical lesions were for the most part slight and unilateral. In no case was there extensive bilateral injury to any thalamic center.

"One of the most difficult problems in the study of cerebral functions is that of accounting for the widely divergent symptoms following similar lesions in different individuals. In our present series, as in other similar studies, we find a number of animals with extensive lesions and yet with training records which are far better than the averages of other animals with equal amounts of destruction, and which may approach the records of normal animals..... Neither locus nor depth of lesion nor injuries to subcortical structures provides any apparent basis for the differences in scores..... The difficulty of the problem is increased by the clinical evidence, especially in the field of aphasia. The conflicting evidence on localization bespeaks a condition in man like that which we find in our series of animals. Except for the primary projection areas, negative cases have been reported for practically every cortical region..... A significant point for the problem, perhaps, comes from the repeated observation that the severity and duration of symptoms from brain lesions are less in young than in old and less in intelligent than in low-grade individuals. If true, this can only mean that the severity of symptoms is dependent not only upon the locus and extent of lesion, but also upon the general level of dynamic functioning of the organism.

"In spite of the marked individual variation, the consistency of the results on various functions studied by various investigators, and the uniform trend of this data suggest that there must be some constant causal factor in

consistency of maze performance dependent upon the mere quantity of cerebral tissue and not an artifact arising from the limitation of this or that special function."

In 1933, Lashley published a discussion of the integrative functions of the cerebral cortex. We shall review this discussion in some detail, for here Lashley raises several general questions which are made more specific in the discussion of our experiment.

He points out that the chief difficulties in the studies of behavior arise from uncertainty as to the validity of the tests, which validity must be established before the results justify any generalizations. The functions which they are intended to measure must be defined, and it must be demonstrated that the tests actually do measure the functions. "In experimental studies with animals, the validity of the test comes still more into question, so that it is scarcely safe to say that a test measures anything more than the performance in the test itself. There is a high correlation in the performance of certain tests, such as the learning of different mazes, which justifies some generalization concerning maze learning ability, but even here we do not know whether we are measuring fixation in memory or some process akin to puzzle solving(insight - Kohler)."

"The interpretation of results obtained by the extirpation method presents peculiar difficulties. Following operations, symptoms may arise not only as a result of the simple absence of functional tissue, but also from

derangement of the functions of the remaining tissue as in shock, general circulatory and nutritive changes, physical or chemical effects of necrotic or scar tissue, and the indirect depression of diaschisis.... Surgical shock, circulatory changes, and irritation may be controlled with considerable certainty by following changes in symptoms over a sufficiently long time, while precluding the possibility of relearning or of normal forgetting. The control of diaschisis is more difficult since the concept is not very clearly defined.

"Von Monakow's theory of diaschisis embodies the following points:

1. Destruction of a cortical area may result in symptoms which are due to the withdrawal of facilitation from some other area and the resultant inadequacy of functioning of that facilitated area.

2. Areas subject to such diaschisis effects recover their functions spontaneously, but no limit can be set to the time required for such recovery.

3. Only such functions as are permanently impaired can be ascribed to a destroyed area.

"This theory casts doubt upon the validity of any experiment involving re-education unless it can be shown that the functions studied do not recover spontaneously but do recover under the influence of training.... The evidence seems conclusive that some functions lost through cerebral injury may be recovered as a result of special training and do not recover spontaneously.... Loss of habits resulting from cerebral injury and their subsequent formation at a rate and with objective performance identical

with their pre-operative formation argue for a restitution rather than a substitution of functions. In every case where a localized area functioning vicariously has been sought, the experiments have had negative results. The number and variety of such experiments is not great enough to justify a denial of the principle of vicarious functioning, but does point to the necessity for further experimenting before the use of the principle as an explanation of recovery can be accepted. The alternative to vicarious functinning is a reorganization within a dynamic system of which only a part has been destroyed.... Direct evidence in support of such a theory is lacking.

"The limits to improvement set by lesions of various sorts have rarely been tested and few generalizations are possible. There are marked interspecies differences... but limits of improvement seem to be set by extent of injury and by the complexity or psychological difficulty of the problem.

"The difficulties of interpreting the results of extirpation experiments and the more essential controls of disturbing factors are thus illustrated. Few investigators have attempted any systematic study of such factors and the great body of work upon cerebral localization is consequently inconclusive. The conclusions drawn in the following sections must be regarded as tentative, pending further analysis of the role of diaschisis and of general circulatory and nutritive disturbances in the production of post-operative symptoms."

"As for cerebral function in instinctive behavior,

the evidence seems quite conclusive that such complex integrations as those involved in recognition of the female in heat and the selection of nest material develop independently of individual experience and there are indications that in mammals they cannot be performed in the absence of large portions of the cerebral hemispheres. This implies that some instincts, which seem to be those requiring accurate differentiation of stimuli, are dependent upon cortical mechanisms.Whether this dependence implies that the necessary integrations are carried out within the cortex, or only that severe diaschisis of subcortical centers after decerebration persists indefinitely, remains uncertain.

"The importance of the cerebral hemispheres for the learning process is obvious from a large number of experiments, but little has been revealed concerning the nature of its activities in learning. From the results with decerebrate animals we can infer little as to the modifiability of subcortical mechanisms in the intact animal. It may be that the removal of the cortex decreases the plasticity of the lower centers by withdrawing some nonspecific facilitation, which under normal conditions permits learning without actually contributing to the specific integrations.Crucial experiments are lacking."

Now Lashley offers a summary of the experimental data derived from studies of vision, which data he considers more complete than any other studies of the afferent paths in learning and maintainance of habits:

1. The habit of entering an illuminated and avoiding a darkened alley in the Yerkes discrimination box

is formed with equal rapidity by normal animals and by animals lacking the striate area, or any other part(up to at least 60 per cent) of the cerebral cortex.

2. Destruction of the striate areas in animals previously trained produces complete amnesia which does not recover spontaneously. Destruction of other parts of the cortex, even adjacent to the striate areas, does not disturb the function.

3.Animals with post-operative loss relearn the habit as rapidly as do normal animals.

4. The amount of training required is closely proportional to the amount of tissue destroyed, provided that there is some invasion of the lateral portions of both striate areas(correlation ratio equals .84).

5. Partial interruption of the optic radiations does not produce a proportionate loss of the habit.

6.When the habit is formed in the absence of the striate areas, subsequent destruction of any third of the remaining cortex does not seriously disturb the habit.

7. Animals trained in this habit with one eye blindfolded discriminate correctly when the blindfold is transferred to the other eye. This transfer takes place as readily in the absence of the striate areas.

8. Injuries to the superior colliculi retard the formation of the habit, somewhat in proportion to the extent of injury.

9. The formation of habits based upon pattern vision is not retarded by destruction of the colliculi.

10. Complication of the habit by requiring the discrimination of two lights approaching threshold dif-

ference results in a marked slowing of initial learning in animals lacking the area striata, though their threshold for brightness may be as low as that of normals.

11. Destruction of the striate areas in animals previously trained in this habit results in complete amnesia. Partial destruction of the areas sometimes seems to abolish the discrimination of two lights without markedly reducing accuracy in reacting to one light alone.

12. The degree of disturbance in learning involving two lights is somewhat proportional to the extent of destruction of the area striata.

13. Interruption of the optic radiations as they enter the external capsule from the lateral geniculate bodies permanently abolishes all reactions to discrete visual objects, but does not interfere with the formation of the reaction to light versus darkness.

14. Destruction of the lateral portion of both striate areas permanently abolishes all pattern discrimination; destruction of the median portions of the striate area or of any other cortical fields does not interfere with the formation of habits based on discrimination of patterns.

15. Lesions in the median portion of the striate areas produce an amnesia for habits based on pattern discrimination which may be reacquired by training. Lesions to other cortical fields do not produce such amnesia(unpub'd)

16. Animals trained on visual patterns react on the basis of the relative attributes of the stimuli, reacting at once to similarities when there is no identity of retinal elements stimulated.

From this data, Lashley finds it difficult to disentangle those results due to defects of vision and those due to disturbances of memory. "The habit based on the discrimination of light and darkness shows least dependence upon the cortex... The evidence at hand indicates that the associative mechanism for the habit lies in the subcortical structures. Lashley interprets his results as indicating one of two alternatives: either that in the learning of this habit in normal animals the cortex participates by the development of some nonspecific facilitative effects upon the lower centers, and that the specific integrations between receptors and the motor systems used in orientation are not contained in the cortex itself; or that the integrations of the habit involve a reorganization of the entire visual system without the local development of specific integrative connections.

"For pattern vision it seems clear that the discrimination of the gross visual characters of objects requires some cortical connections, but can be mediated by the frontal portions of the cortex. This part of the system can mediate reactions to the relative position of discrete objects within the visual field but fails to distinguish between surface area and luminosity. The clear differentiation of spatial attributes of the stimulus is dependent upon the striate areas. There is no indication that any other portions of the cortex than the striate areas are concerned in the performance of habits based on pattern discrimination....From the results so far obtained it seems clear that the habit is disturbed

only by lesions within the striate area and there are indications that the habit may survive the destruction of any part of the visual cortex up to about 50 per cent in both hemispheres." For Lashley, such data suggest that there may be both general facilitative effects and specific integrative relations involved, and that the concept of simple conduction pathways from receptor to the cortex, with the establishment there of an integrative pattern of neuron connections, is a quite inadequate picture of the afferent mechanism.

"The habit of traversing a complex maze is disturbed by injuries to any part of the cortex, and equal injuries in different areas have about equal effects. This indicates either a reduplication of specialized paths from lower centers to and from each part of the cortex, without significant transcortical connections, or for some reorganization of transcortical activity which involves all parts of the system."

"What evidence we have seems to rule out the formation of associations by the repeated passage of nerve impulses over specific paths.... The positive inferences concerning possible neurological bases of learning which can be drawn from recent studies are still too vague to permit of any experimental evaluation."

"The association of diverse cytoarchitectural areas with different functions is well established, but the fact has been emphasized until it obscures the really fundamental problems of cerebral physiology. The production of specific symptoms by specific cortical lesions furnishes a valuable clue to the nature of cerebral organization, but

considered alone, provides no adequate picture of the processes which determine integration.... The facts of cerebral localization leave unanswered the question of how the specialized areas carry out their functions and of how their finctions are interrelated. The character of organization within different cortical fields seems to differ fundamentally. The highest degree of subordinate specialization within single fields has been demonstrated for the stimulable cortex and for the visual area." Lashley and others have shown that for the so-called motor areas, the apparent specificity of excitable points within the secondary areas(head, arm, leg) may be a matter of temporary physiological organization of the area, rather than of stable anatomical connection. Minkowski interprets his results for the visual cortex as showing that each retinal point is projected, not to a single point, but to an extensive area of the cortex of the dog, so that only extensive lesions result in complete destruction of the representation of any point.

"Larionow(1898) and Eliason(Pavlov, 1927) have reported temporary loss of sensitivity to high or low tones following injuries to the auditory areas, but such results have been interpreted by Bornstein(1932) as the result of a general lowering of the level of functional activity and crucial experiments are lacking.

"It seems unquestionably established that within some cortical areas, as defined by anatomical methods, there is a subordinate differentiation of function constituting a sort of mosaic. It seems very probable that

this mosaic differentiation is not in any case as fine as the peripheral functional units. For other areas it seems equally certain that there is no subordinate spatial representation of functions and that any injury to the area reduces efficiency in a number of activities which are independent in behavior. It seems significant that the functions for which mosaic specialization within centers is established are just the ones which involve reaction of the organism to the spatial attributes of its environment."

"In the cortical fields where there is a projection of sensory surfaces, how do the points or foci corresponding to the sensory units function to produce differential reactions? Two opposing views have been advocated, the specialization of each point for a specific reaction, and the functional equivalence of the system.... The assumptions concerning a mosaic of inhibitory and excitatory centers is entirely inapplicable to the above data..... Opposed to the mosaic theory of functional activity of specialized fields is the concept that within the special area all parts are, in certain respects and for certain functions, equivalent.... Three principle lines of evidence have been presented in favor of the theory: the functional equivalence of receptor surfaces, the spontaneous reorganization of motor reactions, and the survival of functions after destruction of any part of nervous centers whose total destruction abolishes them.

"These three lines of evidence indicate that certain coordinated acts, known to be dependent upon definite cortical areas, can be carried out by any part(within undefined limits) of the whole area. Such a condition might

arise from the presence of many duplicate reflex pathways through the areas and such an explanation will perhaps account for all of the reported cases of survival of functions after partial destruction of their special areas, but it is inadequate for the facts of sensory and motor equivalence. These facts establish the principle that, once an associated reaction has been established, the same reaction will be elicited by the excitation of sensory cells which were never stimulated in that way during the course of training. Similarly motor acts, once acquired, may be executed immediately with motor organs which were not associated with the act during training."

"Goltz(1881) first suggested a relationship between the extent of cerebral destruction and the consequent degree of deterioration in more complex adaptive behavior. He interpreted the effect as due to a lowering of attention, implying that the latter is a function of the total energy of nervous activity available.Lashley and Franz raised the question as an experimental problem in 1917 and a number of statistical studies have since been reported. The use of the correlation coefficient is justified in these studies only as a crude indication of the existance of a relationship. Lashley and Wiley find that the retardation is best described as a logarithmic function of the extent of destruction. Beyond establishing that the retardation is disproportionately more severe after large than after small lesions and that the function is a continuous one, the mathematical expression of the relationship has little significance.

"The distribution in magnitudes of the coefficients of all these statistical studies confirms the genuineness of the relationship.... indicating that the functions fall definitely into two types, either completely independent of the extent of lesion(within limits of the tests) or closely dependent. It is not possible from the data to formulate any generalizations concerning the activities which fall into these two classes. Apparently the simplest sensory habits and the simplest maze habits show the least relationship to cortical lesion, but the absence of correlation for initial formation of the latch-box and delayed alternation habits does not fit this interpretation on the sole basis of simplicity."

"Lashley and Wiley found that the relative ease of learning simple and complex mazes is the same for normal and for operated animals. In these latter experiments only reduplication of elements in the tests was involved. Where qualitative differences in the tasks are involved there are indications that small lesions markedly retard some functions and leave others relatively undisturbed.

"The question of what determines relative difficulty of tasks for normal animals and for animals with brain lesions is complicated by many factors. Sensory and motor handicaps unquestionably play apart in retarding the operated animals, but in addition to this there are such conditions as the suitability of the task to the instinctive equipment of the organism, the number of elements which must be dealt with simultaneously in integration, and previous familiarity with these elements... and still another factor is that illustrated by the rela-

tive obscurity of different logical relations in the normal individual. Such variations in the difficulty of qualitatively different tasks and indications of specific organizing tendencies in nervous function suggest that the nervous mechanisms tend innately to certain types of integration and that the relative difficulty of tasks is in large measure dependent upon the extent to which they fit this pre-existing schema of organization.

"The fact of a relationship seems firmly established between amount of tissue destroyed and severity of deterioration for certain activities. Interpretation of the fact still remains in question." Lashley feels that analysis of all the pertinent facts imply that the activities of any part of the cortex in the acquisition, retention, and performance of more complex integrative functions are conditioned by the activities of all other parts. "How this influence is exerted and what its relation to the specific integrative functions of the different areas may be, remains obscure....It is uncertain what part non-specific facilitative effects may play in cerebral activity. Several lines of evidence suggest fluctuations in some general dynamic condition as important in the production of symptoms, suggesting that associations may exist at different energy levels, and this seems best expressed in terms of mutual facilitation of the associative mechanisms within the total system of integrations constituting the engram."

"Herrick(1930) has expressed the significance of such data as follows: there is "first, a known localization of stable structural elements whose functions are also known, and second, a localization of fields within which various

recurring patterns of performance or schemata are known to be fabricated and within which inhibition, modification, or conditioning of these patterns takes place." Lashley feels the evidence seems most consistent with the view that these schemata are dependent upon some dynamic patterns of organization."

In connection with an extensive study of the factors involved in learning various maze patterns, it became evident to Shepard that the rats were using some cue which had not been brought under control. This cue is brought out clearly by the"unit alike" maze consisting of a number of like or standard units followed by an exceptional unit in which the reaction necessary to avoid the blind is precisely opposite to that necessary in the standard units. Most of the rats learned the maze readily. Then, when the rats were placed in the maze at various of the standard units' entrance, they were able, after a brief period of exploration, to orient themselves accurately and to locate the exceptional unit with almost no errors. This demonstrates that the true paths of all the units, standard and exceptional, were not alike to them; that they obtained some differential cue either from within or without the maze. Numerous tests showed that the cue was not visual, olfactory, or kinesthetic-tactual in the usual sense at least. The maze platform was 2" thick and covered with a sort of asphaltic linoleum 3/16" thick and cut into 12" squares. Interchange of these flooring sections caused serious disorientation of the animal, but after a period of errors, he soon again made perfect reactions. Even

felt did not eliminate this cue for rats trained on ½" hair felt, or even felt covered with a soft thin rubberized sheeting.

Then it was found that the character of the subfloor was of more importance to the rat than the surface material, for the materials used, in determining the cue; for interchanging the surface materials of different units caused less disturbance than interchanging the subfloor, leaving the surface materials intact. In working with mazes of various patterns, the dead-end blind, the broken field maze and the circle blind, and various combinations of these types, Shepard found additional evidence of the importance of the factor of floor cue in the learning of these mazes and to adaptation to essential changes in their pattern after original learning. He considers the floor cue to be in some way one of resonance, probably of an auditory nature, but has no positive proof of this. He and his students are at present working on the unit alike maze in an attempt to find a floor upon which the rat cannot react differentially to the floor if inserted into the maze at a different unit each trial. Such a floor in such a maze situation would rule out the possibilities of visual, olfactory, kinesthetic, tactual, and floor cue. Shepard thinks then the trained rats would be unable to learn the maze, whereas on an ordinary floor, they are able to react correctly, no matter where they are inserted into the maze, in a unit alike maze with as many as 9 units. He found that if the rats could learn more than 3 units, they could learn any number of units up to 9, for they were then

using the floor cue. If they were reacting on the basis of kinesthesis alone, only 3 units at most could be learned.

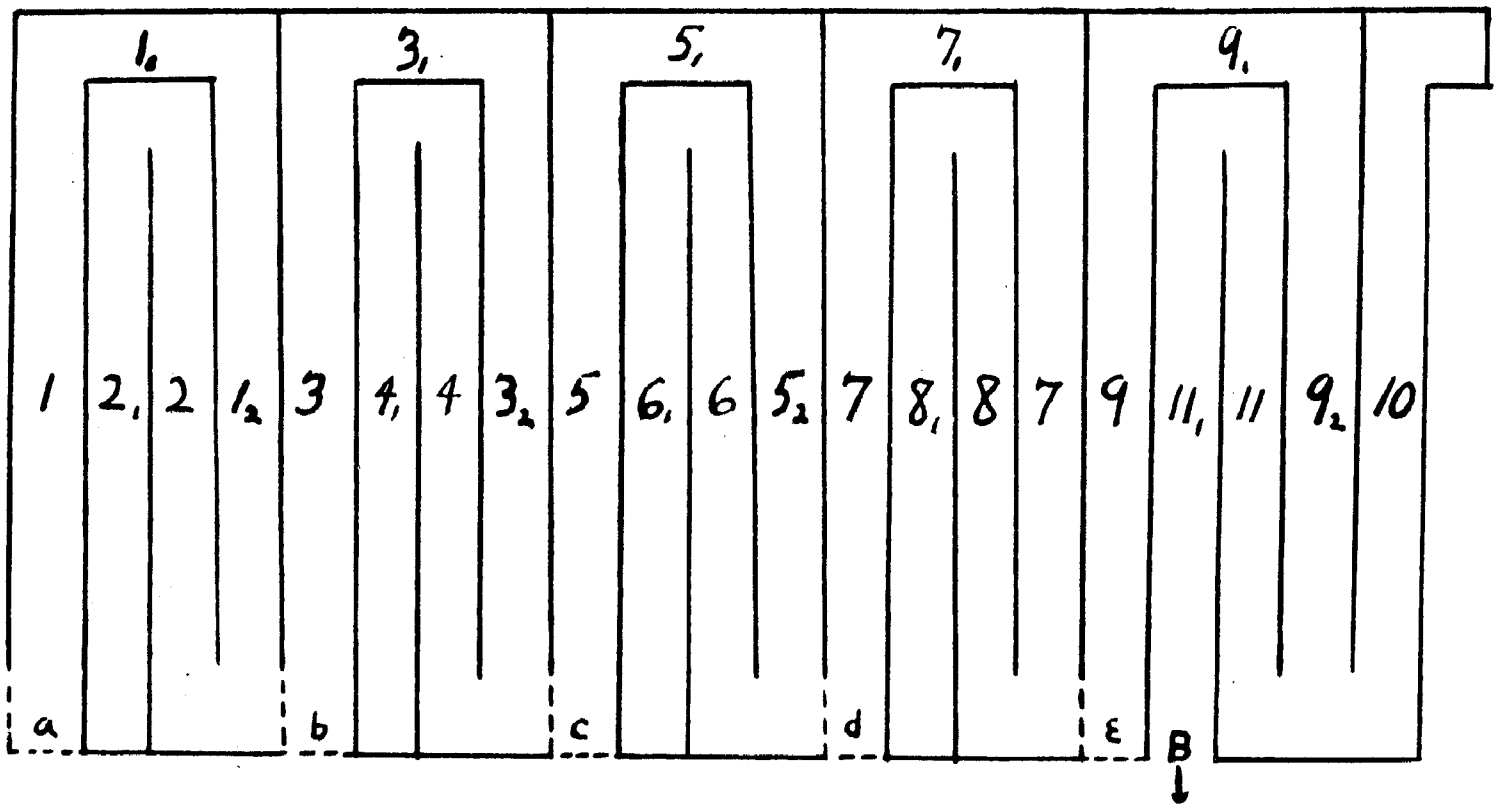
EXPERIMENTAL WORK

Experimental Work

During the course of his experiments Shepard found that, while some rats (to which we shall hereafter refer as Type A) were able to learn both the unique unit and the unit alike mazes, other rats (Type B) were unable to learn the unit alike maze, but were able to learn the various unique unit mazes in which he tried them with at least average facility. He found three kinds of Type B rats: some who, early in their training period, formed the habit of always turning to the right when they came to a junction, thus entering the blinds of every standard unit, and so to the food box; or always turning to the left at each junction, thus entering the blind of the exceptional unit, and so to the food box; some who had learned to run correctly for the first two or three units, but whose score fell down when they were inserted into the maze in a random order of units; and some who formed no persistent habits of any kind but were simply unable to learn the unit alike maze.

The present series of experiments was begun in an attempt to discover whether the removal of any specific region of the cerebral cortex, or any amount of cortex, would reduce Type A rats to Type B: that is, not interfere with the general learning ability, but with the specific factors that enable the rat to learn the unit alike maze.

With this in mind, two mazes were constructed; one a unique unit maze and the other a unit alike maze, each containing five units. In the unit alike maze the five units



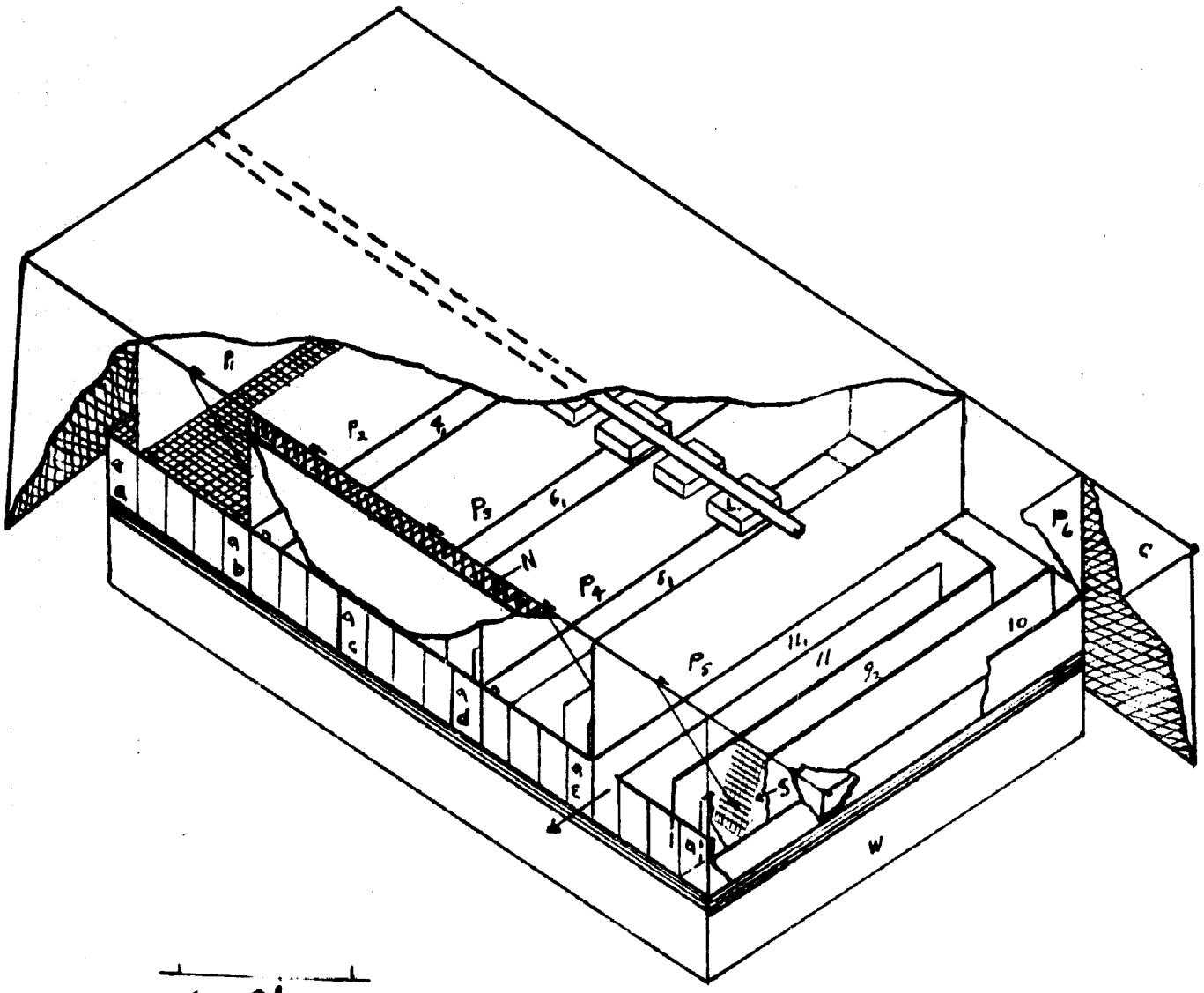
UNIT-ALIKE MAZE

FIGURE 1

were made as nearly as possible identical in every respect save one: since it was desired to emphasize the floor cue, a different sub-floor was used in each unit, this differential sub-floor being laid on the wooden platform (Fig. 2 W) on which the maze was built. In unit a (Fig. 1) the sub-floor was of wood; in unit b, celotex; in unit c, felt; in unit d, cotton; in unit e, paper, and in the extra alley, 10, sand. Over the sub-floors was placed a ½" layer of hair felt. This was covered with a cotton cloth, on which was laid the top floor material, masonite pressed wood, the latter being cut into pieces to fit the alleys of the maze.

In alley sub 2 of each unit, 6" behind every junction, a wire screen block was attached to the wire screen that covered the maze, and so arranged that it could be raised and lowered by the experimenter from the outside of the maze. These blocks were for the purpose of preventing the rat from backtracking, and were not lowered unless the rat had turned some little distance into the blind, or unless the rat had turned around and was retracing on the true path. In the latter case, the block was dropped in the unit just before that in which the rat was retracing, so that the dropping of the screen could by no means furnish a cue for the rat.

The maze was set up in a dark room and lighted by 14 overhead lamps hung in a straight line, so that two lights were directly above the center of each unit, while



← 2' →

UNIT-ALIKE MAZE

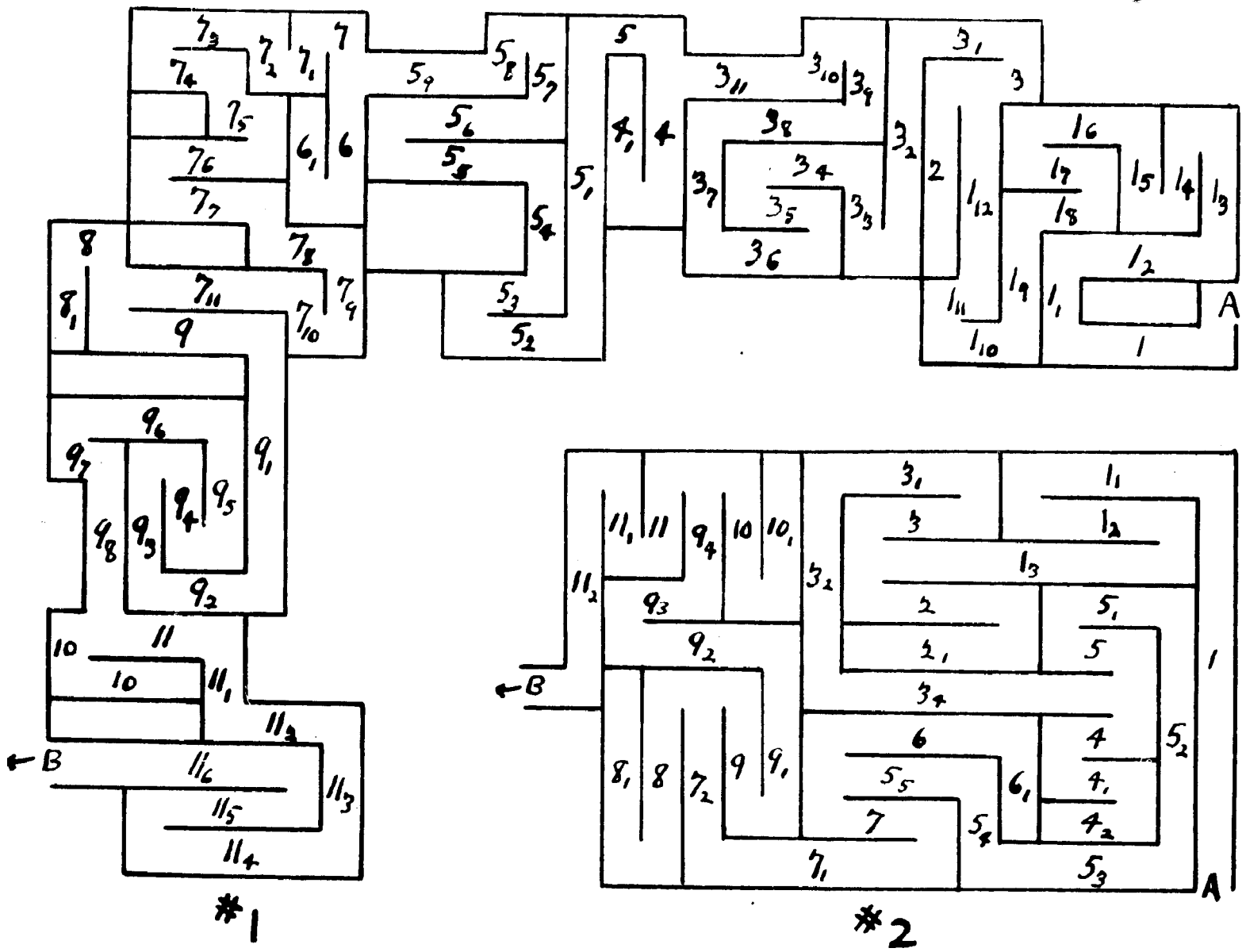
FIGURE 2

two more extended at each end of the maze (Fig. 2, L). The lights were so shaded that all the illumination was directed below the top of the walls of the maze. Between each pair of adjacent units, partitions (Fig. 2, p. 1, 2, 3, 4, 5 & 6) were erected for the purpose of isolating each unit, of eliminating possible differences in sound reflection, and of making sure that each unit is alike; for at the end units, it becomes necessary to have a partition of some sort; so by erecting these partitions, we prevent the end units from differing from the others in this respect. To further control any possible extraneous light or sound cues, the whole structure was covered with a double layer of fabric (cheese cloth, Fig. 2 N, and heavy canvas, Fig. 2, C) which dropped down below the top of the maze walls (Fig. 2). A small slit (Fig. 2, N) was made in the canvas for the purpose of observing the rats in the maze. The proportions of the maze are shown in Fig. 2 which is drawn to scale.

The dark room in which the maze was set up was not sound proof. Incidental noises could be heard by the experimenter from all sides as well as occasional brief noises from other workers; and, in the winter time there were sometimes noises from the radiator at one side of the room. The latter were not constant on any one day, and never appeared consistently enough to be used day after day by any rat as an orienting cue. If anything, they were a source of disturbance. An additional check on this is that a large part of the rats were run during the summers of 1932

and 1933 when the radiators were entirely silent, with no change in the kind of score. Another noise in the room was that from the ventilating fan in the nearby animal house. This was again only occasional, never appearing constantly for any one rat and when it appeared served more as a distraction.

The Unique Unit maze (#1, Fig. 3) was set up in an ordinary room, with a window at one side, there being no attempt at uniform lighting of the different units. The floor material consisted of two large sheets of masonite pressed wood laid end to end. These were laid on a $\frac{1}{2}$" layer of hair felt, which, in turn, rested on the concrete floor of the room. Shepard has found that such a floor offers more difficulty to the rat in obtaining a differential floor cue than a floor broken up into smaller pieces. In this maze, no attempt was made to rule out differential extraneous cues. Thus, in addition to the possibility for a floor cue, other cues available to the rat were: kinesthetic, from the differential kinesthetic patterns of the 5 units; and visual cues from the room, the lighting, the shadows in the maze, and from the difference in geometric pattern of the 5 units. To eliminate any possible cues from the observer in both mazes, the experimenter varied her point of observation at each trial. In the unique unit floor plan, it will be seen that most of the units are so constructed as to tend to throw the rat into the blind at each junction by virtue of the factor of kinesthetic swing. The potency of this factor has been emphasized and analyzed by Shepard. In this maze, the tendency is utilized to throw



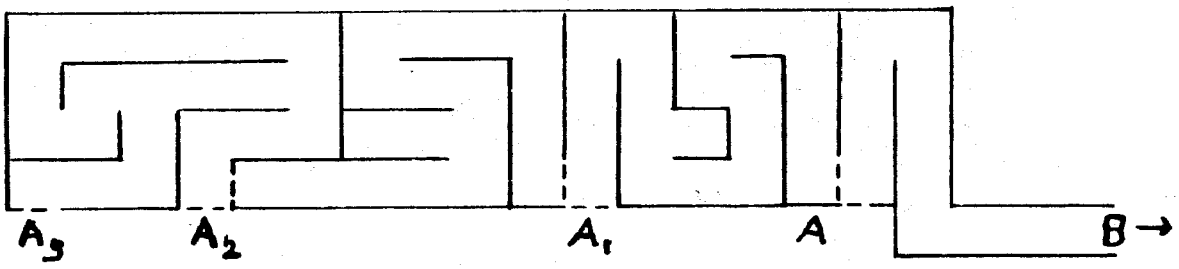
UNIQUE - UNIT MAZES

FIGURE 3

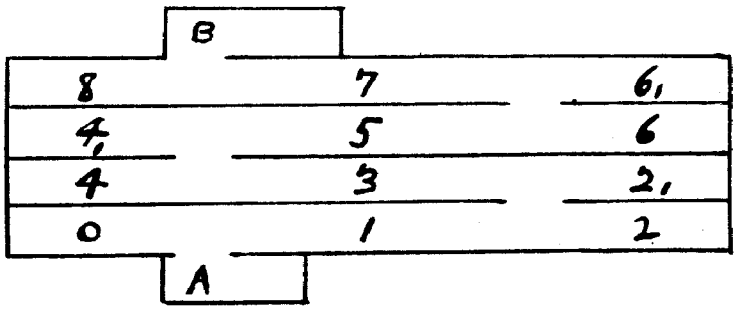
the rat into the blind, thus increasing the difficulty of the maze.

The work was begun on an all or none basis, the question at issue being simply whether the rat's ability to learn the unit alike maze would survive destruction of part of the cerebral cortex, and if not, whether the reduction of learning ability was a general one, which would also interfere with the unique unit maze, or whether it was a loss of some specific factor necessary for the learning of the unit alike and not the unique unit mazes. Since we were not then interested in how the unoperated rat learned the maze, for the first few rats, an incomplete or no preoperative record was kept. But it soon became evident from two rats who were the sole survivors of the practice operations, that rats with an appreciable amount of cerebral cortex removed, as well as some subcortical destruction, were quite able to learn the unit alike maze. Up to the time of this discovery, ten rats had been run in the maze. Thereafter a complete chronlogical record of errors in each trial was kept for every rat in every maze.

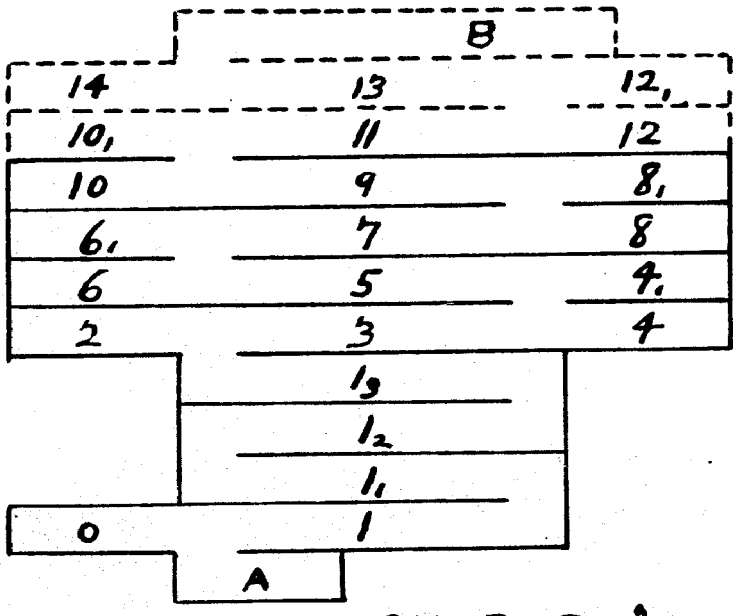
Shepard has pointed out that when a rat is placed in a maze for the first time it is impossible to determine what proportion of his initial efforts are given over to learning the maze, and what proportion to merely becoming oriented to the maze situation and to connecting the maze running with the food. To eliminate this uncertain factor all the rats were trained in a practice maze (Fig. 4) which was so constructed that its length could be progressively increased from one to five units. As rapidly as each rat



PRACTICE MAZE



LASHLEY MAZE III



SHEPARD'S MODIFICATION

FIGURE 4

learned to run one unit, another was opened, until he had learned to run from A_3 to B without wasting any time and without excess movements. In each case an average of about two weeks was spent in the practice maze. Rats that did not readily form the simple habit of running in the maze for food were dropped. Therefore the rats actually carried through the training series represented some amount of selection.

After completing the practice period, the rats were started in the unit alike maze, running at first only three units, c, d, and e (Fig. 1). When they were beginning to run the three units with an errorless score, the 4th and then the 5th unit was opened. When they had run 6 times from a to B without error, on the same day, the next day they were placed in the different units of the maze at random: now at a, now at b, now at d, etc. until they made no errors, no matter where in the maze they were started. Then, as an additional check against visual cues, they were required to run 6 times in succession without error in the dark, starting in the various units at random. In a few of the early cases, #1, 2, 5, 11, 12, and 20, no test in the dark was administered. But of some 50 rats who were so tested, only 1, #21, had any difficulty in running the maze perfectly in the dark after having attained a perfect test score in the light. For this rat, 56 additional trials and 25 errors were required to bring him up to the required performance in the dark. Usually, after one or two trips, the rats ran the maze equally as well in the dark as in the light. After he had passed this test, the rat was considered

to have learned the maze beyond a doubt by means of floor cue, and to be ready for operation. In computing the total scores for each rat, the last 6 perfect runs in the lighted maze are included in the trial scores in every case. The rats were run on an average of 6 trials a day, but this figure was not invariable; sometimes more, sometimes fewer trials were given.

Thirty-two rats were thus treated; 2 of these were operated upon before original learning of the unit alike maze; their scores, therefore, are not included in any of the averages (#1 and #6). Three of the group, #22, 59, and 60, failed to learn the unit alike maze, forming the persistent kinesthetic habit mentioned above. Of the 27 rats who learned the unit alike maze before operation the first ten run, as previously stated, had incomplete or no pre-operative records (marked ? in table 1). Of the 17 pre-operative rats in this group for whom we have a complete pre-operative score, the average number of trials required was 80.1 and the average errors, 61.5.

It has been proven that the unit alike maze was learned on a basis of floor cue, when the rats had passed the above defined criteria. No systematic attempt was made in this study to determine the specific nature of the floor cue, and no positive assumption is made concerning it; but it was thought that the cue was in some way one of resonance, probably of an auditory nature. If the maze were learned on the basis of a pattern of auditory discrimination, this pattern of discrimination would necessarily have to be very fine and highly specialized. If such were

TABLE I

		Unit Alike Maze				Unique Unit Maze #1			
#	%	Pre	Post	Pre	Post	Pre	Post	Pre	Post
		Errors		Trials		Errors		Trials	
2	16.0	?	108	140	165				
3	17.0	?	15	?	35				
30	18.2	?	32	?	69				
6	20.6	-	35	-	70				
4	20.9	?	42	?	81	-	?	-	68
9	21.4	?	27	?	49				
11	22.6	?	110	70	91	-	F	-	F
20	25.9	?	43	?	69				
5	26.2	?	16	?	35				
7	26.4	?	30	?	57				
1	27.0	-	60	-	140				
13	29.0	45	1	58	12				
14	30.8	47	4	49	25				
29	34.0	55	30	79	38				
15	34.4	114	2	150	11				
26	34.7	84	14	39	42				
25	35.8	48	26	54	54				
8	37.0	45	10	90	18				
23	39.0	45	35	47	53				
17	39.2	97	2	126	11				
24	39.3	60	11	47	48				
32	40.0	64	40	64	62				
18	42.0	38	21	54	21				
10	45.0	62	10	99	16				
33	45.2	71	40	74	29				
31	46.5	46	34	50	47				
28	49.0	67	37	65	41				
12	50.4	?	F	?	F	-	F	-	F
19	51.9	58	9	68	22				
22	-	F	-	F	-	?	-	45	-
59	-	F	-	F	-				
60	-	F	-	F	-				
Av.	34.7	61.5	28.8	80.1	43.3				

the case, it was thought probable that some such cerebral localization as Lashley found for pattern vision would be likely: that is, some part, not necessarily any specific part of the region of the cortex given over to the function of audition might be necessary for the learning of the habit. It was thought that so highly specialized a discrimination pattern as we consider this to be probably has some kind of localization if any learning of a discrimination habit is localized in the cortex. For this reason, it was considered best to attempt to focus most of the lesions around the area most probably concerned with the function of audition. The cortico-geniculate connections seem to be rich enough to take care of such a discrimination*

Gurdjian reports that in the rat "the median geniculate body is farther posterior than in higher forms and farther anterior than in lower forms. This migration forward in the evolutionary scale is probably indicative of the increasing importance of the cortex running parallel with the increasing subsidiary nature of the tectum. The cortico-geniculate connections are apparently much better developed in the rat than in lower forms."

The most logical focal point for the operations, then, seemed to be that area delimited by Fortuyn as area p. We have been unable to find any positive proof that the auditory tracts arrive in this area, nor whether total and exclusive extirpation of area p causes degeneration of the median geniculate body, or vice versa.*

Von Gros Clark offers some evidence that such might be the case, but in none of his reported cases did he have complete and exclusive extirpation or destruction of area p or the median geniculate body, so the study offers no answer to this particular question.

The burden of the evidence found in the literature on the subject indicate that the temporal region of the cortex is probably auditory in nature.*

Fortuyn offers the following information: "In the existing literature some suppositions have been uttered about the portion of the cortex which would be auditory cortex in rodents, but they are not confirmed by convincing proofs. Brodmann(09) gives to a large portion of my area p the figures 20,21 and 22, which are given in man to the areas of the temporal convolutions. As in man area 22 (the first temporal gyrus) certainly has an auditory function, I suppose that Brodmann would be inclined to attribute also auditory functions to a part of my area p. Winkler('07) says there are strong arguments in favor of the localization of the perception of hearing in the temporal part of the cortex of the brain. It is quite natural to consider my area p the temporal cortex. So also Winkler would judge the perception of hearing to be localized at least in part of my area p. Isenschmid quotes the supposition of Cajal that the auditory cortex of the mouse would be present laterally of the motor cortex. Isenschmid presumes that his area e could be meant by Cajal and I identified this with my area p. These suppositions are not supported by dicisive proof that area p is the auditory cortex... but probably area p has to be considered auditory cortex, though convincing proof of this could not be given by comparative anatomical researches."

Nor is it known definitely whether the temporal region of the cortex is auditory in function alone, granted that it is auditory.*

Fortuyn says on this score: "There is still another difficulty of a different nature. One knows that the systems of the cochlear and vestibular nerves are greatly mingled within the brain. Now the question occurs whether the temporal cortex is only auditory or also equilibrary cortex. Winkler('07) denies the existance of an area where the perception of equilibrium could be localized, as he denies the perception itself. Other authors, however, believe the existance of an equilibrary area to be indispensible, or they think both functions must be attributed to the temporal area. ....Assuming area p to be the auditory cortex as well as equilibrary cortex, I am quite unable to study the relation of its structure and function. But I will disregard these obstacles for a moment and assume area p to be auditory cortex, seeing that there are many arguments in favor of this.... No variation in the development of area p connected with its supposed function could be demonstrated. Partly this was due to the impossibility to decide which rodents

are quick of hearing and which are not. There was no apparent correlation between the development of some part of the auditory cortex and the number of cochlear nerve fibres. The relation of the breadth of the supragranular pyramids to the total breadth of the cortex seemed not to be connected with the development of associative hearing. The average of the breadth of layer III in area p and of the cellular density in this layer were somewhat less in the waltzing mouse, which is completely deaf, than in the mouse. No changes in area p which could be correlated with the age of the animals could be found."

Also, granting that the temporal region of the cortex is auditory, the various investigators do not agree as to whether the region is divided into sensory and psychic parts.*

Campbell says on this score: "It has been proved beyond a doubt that auditory impulses are first received as crude sensations and then converted into conscious perceptions in some part of the temporal lobe, and it also appears that the part principally concerned in this process is the superior temporal gyrus.... The balance of experimental evidence is in favor of the assertion that removal of the superior temporal convolutions abolishes the receptivity of auditory sensations....The area of the cortex laid down for the primary reception of simple auditory stimuli is that which I have mapped out and described as mainly covering the transverse temporal gyri of Heschl. In all liklihood, there is a second center specialized for the interpretation of and further elaboration of primary stimuli, probably distributed over the area forming a skirt for the audito-sensory area."

Fortuyn finds area p dimorphous in character for rodents, along with several other areas, but does not consider this alone as a suggestion of the way the region functions auditorily. Nor do the various investigators agree as to the exact delimitation of the auditory area. But in general for rodents there seems to be a reasonable amount of agreement.

In this experiment, an attempt was made to concentrate the lesions around area p for the most part, but to cover most of the cortex, less thoroughly by the

combined lesions(See composite diagram - Plate III). In a few cases, effort was made to destroy as much of area p as possible but very little cortex in any areas other than area p, necessitating in these cases relatively small lesions. For the rest, large lesions were attempted, and no effort was made to destroy the cortex primarily in any other area. For the most part, the large lesions did prove to have included destruction of a large portion of area p. Large lesions were desired for two reasons: first because we wished to discover if any given amount of destruction would reduce Type A rats to type B and second, because large lesions would be most likely to include the specialized area necessary for the learning or retention of the unit alike maze, if any such exists.

The cortical tissue was destroyed by means of an electrocautery, under deep ether anesthesia. In most cases it was found necessary to make a lateral skull trephine, thus necessitating the severance of muscle tissue from the side of the skull. The technique used for calculating the extent of injury was that devised by Lashley and described by him in his later 1929 publication. The brains were stained in Heidenhain's hemotoxylin. With the lesions projected on Lashley's diagram, the locus was readily determined and the extent of injury measured by means of a planimeter.

It seems obvious that any effort to determine the amount of cortical destruction as a whole must be but a rough approximation; this is even more the case if we try to measure the amount of cortical destruction in the different functional areas. Lashley reports that a survey of the cytoarchitecture of the brains of rodents reveals to

to him that the disagreements among investigators in this field are so great as to cast doubt on most of the areas differentiated, and that the confusion is particularly striking within the areas called auditory and somesthetic. A brief survey of the work of Campbell, Brodmann, Zunino, Fortuyn, Isenschmid, Winkler and Potter and Woollard reveals to us that the above statement of Lashley is certainly true if one looks for an exact correspondance. But it also reveals a reasonable amount of agreement in the types of cortical structure differentiated in the different areas for rodents, and a still more definite agreement in the higher forms, especially in man. The work of Fortuyn(1914) seemed to us to be especially well controlled. Lashley has used his maps and has modified Fortuyn's diagrams to fit the proportions of the brain of the albino rat, simplifying the whole procedure for us. We have followed Lashley's technique, superimposing a transparent diagram of Lashley's modification of Fortuyn's diagram of the cytoarchitectural fields on the diagram of the lesion in each case. The per cent of total cortex destroyed within each field was then measured with a planimeter(Table 3).

In every case there was more or less destruction of subcortical tissue. We have classified the kind of subcortical destruction involved as follows: 1) Those brains which, under higher magnification revealed some destruction of the white tissue immediately underlying the cortex (Table 3, Under W.); 2) those brains which revealed mild hippocampal destruction(Mild H.); 3) Those brains which revealed appreciable hippocampal destruction(App H.); 4) Destruction of brain tissue that just barely touched

upon the [redacted] caudate-putamen complex(J.T. S.); 5) Those which revealed a mild striatal destruction, in no case involving appreciable destruction of the caudate-putamen complex(nor cingulum nor amygdoloid nucleus), and in no case going as deep as the globus pallidus(Mild S.).

A period of ten days after operation was permitted to elapse before the rats were again started in the unit alike maze. In no case did this seem too short a time for recovery from the operation. The average extent of cerebral cortical destruction for this group of 27 rats was 34.7 per cent of the total cortex. The average extent of lesion for the 17 rats whose complete pre-operative record in the unit alike maze was kept was 40.3 per cent of the total cortex. All of this group of 27 rats relearned the maze, or retained the habit to some degree, with the exception of #12, who, after operation, developed blindness in one eye, held his head on one side, always turning to the right when possible, and who gave no observable response to a variety of sounds. In addition to these factors, #12 was also in very poor physical condition soon after operation, and developed peculiar eating habits. Another of this group, #11, never obtained a score better than five perfect runs out of six on any one day. When this rat was first started in the unit alike maze after operation, he would not run, so after 22 trials and 51 errors he was transferred to the unique unit maze #1, which he failed to learn. But when he was later returned to the unit alike maze, after 91 trials and 110 errors he attained five perfect runs out of six on the same day. For the above reasons, his score is not included in the average for the group. Nor is #30 included

in the average, for in this case the tympanic membranes were punctured and the ossicles dislodged before cerebral operation, in an effort to deafen the rat. This will be described later in detail. Rat #2 received treatment a little different from the rest of this group in this respect: it soon became evident that he was having unusual difficulty in learning the maze after operation, so that, during the latter part of his post-operative training period, wire blocks were inserted into the maze at the entrances to the blinds. With this help he soon learned the maze and when the blocks were removed, satisfied the test criteria. His score is included in the average for this group. #12 died after 30 trials in the unique maze #1, but during that time showed no signs of learning the maze.

Of the rats who relearned the maze, 24 were treated sufficiently alike to be included in the averages. The average number of trials required to attain the criteria after operation was 43.3; errors, 28.8. Of the 17 rats for whom we have complete pre-operative records, the average number of trials required was 32.3; errors, 25.7(Table 1)

Obviously, the rats retained something from the original training in the unit alike maze. The results raise, among others, the following questions: How much of the difference between the pre-operative and post-operative scores was due solely to the operation, either to the lack of cortical tissue, or subcortical tissue, or to some other factor resulting from the operation? Why should the unit alike maze show so little retardation after relatively large lesions, as compared with Lashley's maze III, which was smaller, of seemingly simpler pattern, which was learned

more readily or at least with no more difficulty by the normal animal than the unit alike maze (80.3 trials, 61.5 errors for unit alike as opposed to 24.8 trials and 54.5 errors found by Lashley for maze) and capable of being mastered by one generalization, i.e; right, left, right, left and food box right - a kinesthetic swing? It was thought that one factor possibly effecting this difference in results might be the suggestion of Lashley and Wiley that area p, around which most of these lesions were centered, might be of less valency in learning the Lashley maze than any other area. But the retardation in the unit alike maze as a result of the lesions was much less than was found by Lashley (with like lesions) in his maze. The best way to answer these questions seemed to be to give the animals pre-operative retention tests to compare the effect of a ten day interval with that of a ten day interval plus operation, and then to run the same rats in an approximate duplicate of Lashley's maze. Accordingly such a maze was set up in a dark room with one light overhead. No attempt was made to control extraneous cues; Lashley made none. The experimenter again varied her position in the room in observing the rat during each trial to rule out any possible constant cue from the operator.

Twenty-eight additional rats were trained in the practice maze for the purpose of obtaining an answer to the above questions. To obtain these 28 rats, about 40 were started in the practice maze, but those who proved to be poor runners were dropped; so the 28 rats chosen represent some degree of selection. Both in this and in the first

group male and female rats ranging from six months to one year in age were used as subjects. The females in both groups were #6, 23, 24, 25, 26, 28, 29, 31, 32, 33, 51, 52, 53, 54, 55, 56, 57, and 58. Of these 28 rats #55, 56, 57, and 58 had had ear operations before original training, and therefore their scores are not included in the averages of the group. These ear operations represented again, part of an attempt to deafen the rats, leaving them otherwise normal. We felt that such an attempt would help to decide whether or not the floor cue was of an auditory nature. This attempt will be described later in detail. Since #56, 57, and 58 showed no serious results from the operation, they were given the same treatment as the others of the group. It was gathered from Lashley's results that there was very little pre-operative amnesia after a ten day interval, as seemed natural for so simple a habit. No pre-operative retention tests for the Lashley maze were given to this group.

Leaving out of consideration the 4 rats with ear operations, 5 of the other 24 rats of this group were started in the unit alike maze before the Lashley maze was constructed, so we have no pre-operative scores for #16, 35, 36, 37 and 38 in the Lashley maze. Of the 19 rats of this group that were run in the Lashley maze before operation, the average number of trials required were 56.29 and the average number of errors, 62.6. Five to ten trials were given a day in this maze, and the criterion for learning was

Table 2

#	%	Unit Alike Maze Errors Pre	Ret	Post	Trials Pre	Ret	Post	Lashley Maze Errors Pre	Post	Trials Pre	Post	#1 Unique Unit Maze Errors Pre	Post	Trials Pre	Post	#2 Post E	T
57	21.8	74	82	33	92	121	58	-	48	-	24	-	-	-	-	-	-
54	25.1	58	8	3	62	30	12	69	49	76	13	-	23	-	16	-	-
58	25.7	70	92	35	73	145	43	-	301	-	109	-	-	-	-	-	-
49	26.8	75	1	5	74	7	24	48	29	66	24	-	-	-	-	-	-
34	34.4	33	1	60	44	7	76	59	-	30	-	-	-	-	-	-	-
42	34.5	56	4	14	73	8	30	72	94	63	41	-	-	-	-	-	-
52	35.0	79	9	29	100	19	33	70	90	65	41	-	-	-	-	90	41
40	35.0	85	14	51	84	47	39	66	322	48	131	-	F	-	F	F	F
35	36.1	*	-	69	*	-	77	-	17	-	22	38	F	40	F	-	-
46	38.8	60	3	18	61	12	30	65	332	52	114	-	F	-	F	71	37
39	38.8	68	21	51	85	42	64	61	182	44	79	-	-	-	-	-	-
44**	39.5	66	0	16	81	6	36	41	326	37	127	-	F	-	F	147	62
41	39.9	100	7	12	82	9	24	56	209	73	70	-	-	-	-	-	-
48	40.0	41	0	21	53	6	37	71	516	57	95	-	F	-	F	F	F
53	42.0	33	2	74	46	12	69	32	F	35	F	-	-	-	-	147	62
45	45.0	56	0	27	58	6	31	65	238	71	66	-	-	-	-	-	-
36	46.0	143	1	133	159	7	150	-	419	-	68	-	-	-	-	-	-
47	46.0	103	11	11	103	27	24	74	F	45	F	-	-	-	-	-	-
37	47.0	167	3	F	243	13	F	-	F	-	F	-	-	-	-	F	F
56	54.0	52	0	21	70	6	20	-	-	-	-	-	-	-	-	-	-
43	55.4	138	16	43	188	24	37	60	369	44	111	-	-	-	-	188	66
27	-	F	-	-	F	-	-	66	-	54	-	-	-	-	-	-	-
38	-	182	11	-	154	36	-	-	-	-	-	-	-	-	-	-	-
50	-	F	-	-	F	-	-	84	-	106	-	-	-	-	-	-	-
51	-	55	9	-	68	25	-	68	-	56	-	-	-	-	-	-	-
55	-	F	-	-	F	-	-	-	-	-	-	-	-	-	-	-	-
100	-	***	-	-	***	-	-	16	-	15	-	20	-	13	-	(10)	(16)
UAF	-	F	-	-	F	-	-	42	-	25	-	41	-	22	-	(33)	(16)
16	-	41	13	-	44	22	-	-	-	-	-	-	-	-	-	-	-
21	-	67	28	-	96	60	-	67	-	47	-	-	-	-	-	-	-
Av.	39.2	88.9	7.5	37.6	96.0	23.2	46.0	626	2274	563	716						

* Score described and explained in text

**Brain sectioned at uneven intervals, thus locus and extent of lesion inaccurate.

*** No score is presented for this rat as the unit alike maze in which he was trained, while of the same pattern and number of units, differed as to set-up, and the method of training was different; but the maze was learned readily.

10 runs without error in succession on the same day. The last ten successful runs are included in the trial score in every case.

Then these 24 rats were run in the unit alike maze. #27 who had readily learned the Lashley maze failed to learn the unit alike maze at all after 158 trials; he formed no persistent errors, and ran well, but never got more than 3 perfect runs in a row and this only once. Also, #50, who had learned the Lashley maze refused to run in the unit alike maze after the first few trials, seemed discouraged and just sat down on the job. No amount of deprivation of water and food could force him to run so after 24 trials he was dropped having shown no signs of learning during this time. Also #35, after 201 trials still failed to show definite signs of learning the maze, though no persistent habits were formed. He was then transferred to the unique unit maze, which he learned readily. Then he was brought back to the unit alike maze and after 24 trials and 12 errors, rached the criteria for learning the maze. Thus we cannot include his score in the average for this group in original learning of the unit alike maze. The average of all of this group who learned the unit alike maze (21 rats) were 96 trials and 88.9 errors. Then, after a ten day interval, this group of 21 rats was given a pre-operative retention test in the unit alike maze, the average number of trials required being 23.2, and errors, 7.5.

Then these 21 rats and #35 were subjected to the operation, as well as three of the rats with ear operation,

whose scores we do not consider here. Four of the 22 rats died during or soon after operation. Of the other 18, the average lesion was 39.2%. After a ten day interval, this group of 18 were again started in the unit alike maze. Only one rat, #37, failed to relearn the unit alike maze. He showed no signs of relearning after 123 trials. In his initial pre-operative learning of the unit alike maze, this rat required more practice to learn the maze than any other rat, 243 trials and 167 errors. #37 formed no persistent errors either before or after operation, but was still making no progress after 123 trials in post-operative running. He was a fair runner. It will be seen later than #37 also failed every other maze in which he was tried after operation, so that he cannot be classed in Type B. He was tried in no other maze than the unit alike before operation. Of the 17 who relearned or retained the unit alike maze after operation, the average number of trials required was 46.0, and average number of errors 37.6.

Then this group of 18 rats were run in the Lashley maze. Again #37 failed to show any signs of learning the maze after 151 trials and 1504 errors. Also, #53 showed no signs of learning the maze after 177 trials and 516 errors. #47 never got more than 3 perfect runs in a row, and that only once; for the most part he made an average of 1 perfect run out of 10 toward the end. So he is considered as having failed to learn the maze after operation. #34 died soon after he was begun in the Lashley maze. Of the 14 rats

who learned the Lashley maze after operation, the average number of trials required was 71.57 and the average number of errors 227.39.

The average for all the rats for whom we have complete scores (excluding of course #55, 56, 57 & 58) in learning the unit alike maze before operation was 95.4 trials and 70.5 errors (38 cases). The average of all rats for whom we have pre-operative retention scores was 23.2 trials and 7.5 errors (21 cases). The average of all rats who retained or relearned the unit alike maze after operation (except #1, 6, 56, 57 & 58) was 46.3 trials and 32.29 errors (43 cases). We see that the difference here between pre and post operative retention tests in the unit alike maze is very small considering the amount of brain tissue removed, or destroyed. We will later analyze the cerebral destruction in an effort to determine if there is any relation between extent or locus of cortical or subcortical tissue destroyed and performance in the unit alike maze.

When we examine the results from the Lashley maze, we find another story. The average number of trials required to learn the maze originally before operation, we found to be 56.29 and the average number of errors to be 62.6 (19 cases). The average number of trials required for post-operative retention was 71.57 and theaverage number of errors was 227.39 for the 14 rats who successfully attained the criteria for learning. For this same group, the average number of trials required to learn the unit alike maze prior to operation, we have seen to have been 96 and the average number of errors, 88.9. For pre-operative

retention in the unit alike maze this same group required an average of 23.3 trials and 7.5 errors. For post-operative retention they required an average of 46.0 trials and 37.6 errors. Thus, for the same rats we find a much greater disturbance in the Lashley maze than in the unit alike maze. This is perplexing in that the cerebral destruction, largely centered around area p, had little effect upon the performance of the maze, for the learning of which we considered an auditory cue essential, and seriously effected the learning of the maze for which we considered auditory cues unessential. The results may be interpreted as follows:

1. The basis for the learning of the unit alike maze is subcortical. The test for this possibility would be a systematic study of the effect on the performance in the unit alike maze of total and exclusive destruction of the various cerebral nuclei. It would seem desirable to begin with a study of the effect produced by total and exclusive destruction of the median geniculate body.

2. The basis for the learning of the unit alike maze is cortical but area p is not auditory in function. But Wiley's study and the other pertinent literature reviewed indicate an auditory function for area p. Also, our composite diagram reveals that the combined lesions cover most of the cortex. These lesions in no case reduced Type A rats to Type B.

3. Area p normally functions in the learning of the unit alike maze but the maze can be learned equally well in any other part of the cortex. Where something was retained from the learning of the maze prior to operation,

the retention is due to the portion of area p left intact, whether the retention is in terms of a specific memory trace of the differential floor cue, or merely a general attention to the floor cue factor in connection with the whole situation. The test for this would be a systematic study of the effect on the performance in the unit alike maze of total removal of the auditory cortex with as little involvement as possible of the other areas. Even if we had found all of area p to be destroyed in some of our cases, we could not be sure that no portion of the temporal area(if the temporal region is auditory) was left intact as our measurements of the per cent of total cortex destroyed in each of the functional areas are but a rough approximation of Fortuyn's areas, which, in turn, are not themselves established beyond a doubt for the animals from which he made the maps. But if the learning of the unit alike maze is at all comparable to the learning of a visual pattern discrimination or even discrimination of light versus darkness, the amount of retention should have some relation to the amount of area p left intact. This does not prove to be the case. In those cases we have where most of area p was destroyed, distinct retention is shown subsequent to operation. A rank order coefficient of correlation for the relation between the amount of area p destroyed and performance in the unit alike maze subsequent to operation would have little value both for the reasons above mentioned and because the amount of subcortical destruction and of destruction in areas other than area p varies to so great an extent for the different cases. For such value as it may have we have computed the correla-

Table 3

#	Sub-C	%p	%j	%w	%f	%k	% Lesion
2	Mild S	3.9	6.4	4.3	1.4	-	16.0
3	Mild H	9.8	5.1	2.1	-	-	17.0
30	Under W	3.4	3.8	9.5	1.5	-	18.2
6	Under W	17.6	1.9	1.0	-	-	20.6
4	J.T. S	13.9	5.5	-	-	1.1	20.9
9	Under W	17.7	2.5	.9	-	-	21.4
57	App. H	13.6	3.6	4.6	-	-	21.8
11	J.T. S	18.2	1.4	2.8	-	-	22.6
54	Under W	6.9	4.8	11.8	1.6	-	25.1
58	Mild H	16.4	5.3	3.9	-	-	25.7
20	Under W	20.0	5.2	.3	-	.3	25.9
5	Under W	8.7	13.8	2.1	1.6	-	26.2
7	Under W	16.3	7.8	1.1	-	.2	26.4
49	J.T. S	17.3	7.3	1.9	-	.2	26.8
1	J.T. S	19.0	3.9	4.1	-	-	27.0
13	J.T. S	18.6	7.3	3.1	-	-	29.0
14	J.T. S	15.6	10.4	4.8	-	-	30.8
29	J.T. S	18.0	6.9	9.0	-	-	34.0
15	Under W	14.4	13.9	4.7	1.4	-	34.4
34	App.H	14.6	4.3	14.5	.8	-	34.4
42	App.H	15.6	10.9	7.0	.8	-	34.5
26	Under W	10.9	14.0	8.1	1.6	-	34.7
52	J.T. S	3.1	9.9	11.1	10.9	-	35.0
40	Mild S	16.9	4.9	10.9	1.9	-	35.0
25	J.T. S	15.1	10.0	8.8	.7	1.1	35.8
35	J.T. S	22.5	10.3	2.7	.4	.3	36.1
8	J.T. S	10.3	10.8	13.7	1.8	-	37.0
46	J.T. S	17.4	9.8	10.0	1.6	-	38.8
39	J.T. S	21.3	9.9	5.7	1.0	.8	38.8
23	J.T. S	12.5	15.5	9.3	1.2	-	39.0
17	Under W	17.9	11.5	9.0	.6	-	39.2
24	Under W	16.9	16.9	4.6	.2	-	39.3
44	J.T. S	19.2	7.7	11.0	1.2	-	39.5
41	App. H	16.5	6.9	14.4	2.1	-	39.9
48	Mild S	16.4	11.3	10.7	1.3	-	40.0
32	Mild S	19.0	13.8	5.8	.3	1.0	40.0
53	Mild S	2.9	14.9	7.9	15.8	-	42.0
18	J.T. S	22.7	15.1	3.3	.5	.3	42.0
45	Mild S	9.1	15.6	12.5	7.7	-	45.0
10	Under W	16.4	17.0	9.5	1.8	.1	45.0
33	J.T. S	21.6	13.2	6.8	-	3.6	45.2
36	Mild S	19.3	20.9	.4	1.7	3.7	46.0
47	Mild S	6.8	16.6	9.8	12.2	-	46.0
37	Mild S	14.9	16.1	10.4	5.8	-	47.0
31	J.T. S	9.4	16.0	13.0	8.1	-	46.5
28	J.T. S	21.9	16.3	9.1	1.2	.2	49.0
12	Mild S	17.6	14.9	10.7	4.9	2.2	50.4
19	J.T. S	17.2	20.6	10.3	3.6	-	51.9
56	Mild S	20.7	15.4	12.9	3.6	1.3	54.0
43	Mild S	19.6	14.0	17.2	4.6	-	55.4
Av.		15.8	10.6	7.0	2.1	.33	35.4

tion and find p to be .29, with a $P.E._r$ of .09.

4. The unit alike maze is learned on a cortical basis or on a subcortical basis, but the resonance cue is not in terms of audition. The test for this would be to deafen the rats completely, leaving them normal in all other respects. We have made an incidental attempt to perform this test, which attempt will be described later in detail.

As we have said, Shepard has proved that the unit alike maze is learned on the basis of floor cue, in some terms, and that vision, olfaction, and tactual-kinesthesis are not utilized by the rat as differential cues in this situation. On the other hand, we believe that the Lashley maze is learned by the normal rat largely on the basis of kinesthetic swing, also possibly orienting in the general visual environment(direction of light, etc.). It seems possible that this maze could be learned on the basis of floor cue, but if such were the case, the normal rat would probably require much longer to learn the maze than he actually did require. This last is indicated by Shepard's records of rats that do use floor cue. The Lashley maze is certainly not a case of alternation in the sense that Hunter, Carr and Loucks use the term, the main difference being that the alternation box forces the rat to alternate over the same path, which is not the case in the Lashley maze. Also, as Lashley points out, his maze is learned much more readily by the normal rat than is the alternation habit.

Shepard ran 8 trained rats through a maze of the general plan of the Lashley maze III, so modified that the

rats were forced into the right-left-right swing before they arrived at the first choice in the Lashley maze(Fig.4) If the rats were learning the maze on the basis of such a kinesthetic swing, this should enable them to catch on to the swing more rapidly than they would without this modification. Two of the eight rats, after the first trial through the modification, made a series of errorless runs. The other six, after the first few trials, made a series of errorless runs. For the eight rats, the average number of trials required before the series of errorless runs was 3.5, and the average number of errors made during these trials was 13.5. The fact that these scores are decidedly less than the average scores of normal rats in the Lashley maze III indicates that the factor of kinesthetic swing plays an important role in the normal learning of the maze.

During these trials, the food was placed in alley 12, the rest of the maze being blocked. The rat was started at A and run to 12(Fig.4) After the rats had learned the maze in this way, Shepard removed the block, now placing the food at B, still starting the rats at A. The floor that had been under alley 10_1,11,12 was moved forward under B, and a new floor was inserted under alley 10_1,11,12 and 12_1,13,14. Now, when the rats were started at A, they invariably entered the blind 12, and usually also 12_1; then, in some cases they went on toward the food box, hesitatingly, and occasionally making errors from 12 to 14. In other cases, the rats retraced from 12, back to the beginning of the maze, again making errors and running hesitantly. This part of the experiment indicates that the rats had a distinct appreciation, in some terms, of the distance they

had traveled in the first situation. There was positive transfer from the one situation to the other in that the rats carried over to the new situation the general notion of kinesthetic swing, but there was also a negative transfer, making for errors where B had been before, for the rats also carried over into the new situation an appreciation of the distance traversed in the old situation.

It seems probable that a maze of this general plan with 16 culs de sac would offer little more difficulty to the normal rat than one of 8 culs de sac for original learning; but if the 8 culs de sac maze is learned first, and then the same rat is run in the 16 culs de sac maze, one cannot arbitrarily differentiate between the positive and negative transfer factors. Certainly this would not be simply a quantitative difference, as Lashley considers it.

Our results suggest that it may be something akin to this factor of kinesthetic swing with which the cerebral destruction, cortical, or subcortical, or both, interferes, thus increasing the difficulty of the performance in the Lashley maze out of all proportion to that in the unit alike maze. Here it might be well to present an analysis of the types of behavior found in the unit alike and Lashley mazes:

<u>Unit alike pre-operative:</u>

1. For those rats who successfully attained the criteria for learning prior to operation, the amount of practice required ranged from 39 trials and 33 errors to 243 trials and 182 errors. Where progress was shown, the training was carried on beyond 150 trials. The ease of original learning of the maze seemed to some extent a matter of chance, i.e, when the rat happened to attend to ...

to the floor cue in connection with the whole maze situation. As soon as the rat had begun to make a score above chance, the progress towards errorless runs, no matter where the rat was inserted into the maze, was rapid for the normal animal.

2. We found three kinds of failures in the group of normal rats: Some who , early in their training period formed the habit of always turning to the right when they came to a junction, thus entering the blinds of every standard unit, and so to the food box, or always turning to the left at each junction, thus entering the blind of the exceptional unit, and so to the food box; some who formed no persistant habits of any kind and who ran well, but were simply unable to learn the maze; and some who, after the first few trials, ran back and forth in the first blind they happened to enter, then sat down in the blind and remained there till they were removed from the maze. No amount of deprivation of food and water could modify this behavior so we were forced to drop these rats.#22,59 and 60 were examples of the first kind of failure, #27 and 55 of the second, and #50 of the third. Several more of the second and third kinds of failure were found than we have recorded here.

Lashley maze pre-operative:

1. All the rats proved able to learn the Lashley maze prior to operation, the number of trials required for errorless performance ranging from 30 to 106, the number of errors ranging from 32 to 84. Again it seemed partially a matter of chance as to how soon the rats would catch on to the swing, and again when the rats once caught on to the

swing they made rapid progress toward errorless runs, more rapid here and in the unit alike than the operated cases made. It seems likely that the good workers among the rats catch on to the swing factor more readily than the poor workers, and it must be remembered in this connection that all the cases reported here represent some amount of selection, in that the poorest workers were dropped while still in the practice maze. In no case did the rats trained in the Lashley maze prior to operation make persistant errors or perseverate long in the same pathway. We have recorded the number of entrances by this group in the various blinds, with the following results: 0 - 274, 2 - 202, 2_1- 180, 4 - 133, 4_1- 232, 6 - 61, 6_1-105, 8 - 15. We thus find that far more errors were made in the first than in the last half of the maze.

Unit alike post-operative:

1. Those rats who successfully reached the criteria for learning of the unit alike maze after operation required from 11 to 165 trials and from 1 to 133 errors. No consistent difference was noticed between the behavior of the rats in the unit alike maze prior and subsequent to operation.

2. There were but two post-operative failures in the unit alike maze, neither of which could be classed as Type B rats. One, #12, was in very bad physical condition after the operation, became blind in one eye, held his head on one side and gave no observable reactions to a variety of sounds. When it was seen that he was making no progress in the unit alike maze, and that he seemed to have an irresistable tendency to turn to the right whenever possible, wire blocks were inserted into the maze at the entrances

to all the blinds, as had been tried, and successfully, in the case of #2. But #12, even with this help, if such it was, proved unable to learn the maze. His record in the maze means very little because of his exceedingly poor physical condition. The other failure, #37, had more difficulty in learning the maze originally than had any other rat. Neither before nor after operation did he form any persistant errors, but after 123 trials was still making a chance score. It will be seen that this rat failed every other maze in which he was run. He was not a good worker.

Lashley maze post-operative:

1. There were but three failures in this maze subsequent to operation, two of which never did catch on to the swing, and rarely made a 180 degree turn. They would zig-zag through the maze, i.e, from 2 to 4 to 4_1 to 6 to 6_1 etc, toward the latter part of the training period.(#37 and 53). #47 was a partial failure, in poor physical condition during most of his time in the Lashley maze. All three formed persistant errors, running back and forth over the same straight path and seemingly disoriented with respect to the food. #47 made many more 180 degree turns than did the other two, and did not perseverate over the same path as long, but he never made more than one successful run out of ten.

2. Those rats who successfully reached the criterion for learning the maze after operation required from 13 to 131 trials and from 17 to 516 errors. We have computed the number of entrances into the various blinds made by the operated rats with the following results: 0 - 1035, 2 - 1002, 2_1 - 925, 4 - 839, 4_1 - 844, 6 - 490, 6_1 - 426,8 - 216.

Thus again there are many more entrances into the early than into the later blinds. The average number of errors, including those made by the failures, was 304. It is seen from these figures that the number of entrances into the blind, 8, which runs past the entrance to the food box, is out of all proportion to the number of entrances made prior to operation, the increase being greater relatively than the increase in entrances into the other blinds. All the rats after operation, whether successes or failures, had a tendency to some extent to pass by the doors without stopping to investigate. This tendency never appeared before operation. We may differentiate two types of successes:

a. Those rats whose behavior in the maze prior and subsequent to operation were very similar, except for a slight tendency during the first few trials after operation to pass by the doors without seeming to notice them and to perseverate in the same alleys(but never more than once or twice in the same alley). This group required from 2 to 20 trials to catch on to the swing. It seemed a bit more difficult for the operated rats to eliminate occasional errors after they appeared to have caught on to the swing, and this latter factor accounts for at least half of the errors. Rats in this group are #35,42,49,52,54, and 57.

b. This second type had a much more decided tendency to perseverate well into the training period, often retracing, especially at first, dashing back and forth in the same alley without seeming to notice the doors. This group did not seem to catch on to the swing suddenly, but went through a series of stages. First the perseverative tendency appeared; then they would go through the doorways

but make few 180 degree turns(i.e. go from 3 into 4_1 instead of into 5. Then they began to make the 180 degree turns and finally began to iron out the occasional errors. The dashing back and forth was characteristic of no particular group of rats, though it was more predominant in the latter type of success. Rats belonging to this group were #36,39, 40,41,43,44,45,46,48 and 58.

We cannot tell why the slow-moving and even the fast rats should pass by the doorways without seeming to notice them. In some cases, after the rat had passed the door, it was observed that he nosed toward the direction of the door, but had just passed it so felt only the wooden wall and went on. It might have been in these cases a belated visual or auditory reaction. Lashley reported no such behavior for the blind rats, so that the difference in resonance is more probable. It might have been more of an inability or unwillingness to turn rather than lack of stimulation by the doorways. We have no answer to this question. But we cannot agree with Lashley that the behavior of the operated animals in the maze situation shows a general disorganization, for no such disorganization appeared in the unit alike maze. This indicates that the disorganization found both by us and by Lashley in the Lashley maze results from a disturbance of some specific factor which is necessary for the learning of the Lashley maze and not for the unit alike maze. We considered that this specific factor might be in terms of kinesthesis, not simple bodily orientation and maintainance of balance, but a complex pattern, illustrated by kinesthetic swing.

A more general effect of the operations might have

been a decrease of attention to the maze situation, some such effect as Cameron found, greater difficulty in elimination of occasional errors, both in the Lashley and the unit alike maze.

As we have said, the unique unit maze #1 was constructed in such a way as to utilize the swing tendency for throwing the rat into the blind at the junction of the first, second and fourth unit. Accordingly, all the persistant errors proved to be in blinds 2,4, and 8. Only two rats of our group of 50 were tested in this maze before operation, both of whom learned the maze readily after failure in the unit alike maze(#22 and 35). In addition to these, two of Shepard's trained rats, which he had studied over a long period of time and in a wide variety of situations, were trained in this maze. One of these, #100, had readily learned every maze in which he was tested, including the unit alike maze. The other, UAF_3, learned all the mazes readily except those situations where floor cue was essential; he did not seem to be able to use the floor cue; at least he did not. Both of these rats also learned the unique unit maze#1 readily(Table 2) Nine rats were run in this maze subsequent to operation, and of these nine, seven failed to learn the maze.(Table 2). The compensatory swing seemed more effective for the operated than for the normal rats, throwing the rats into the blinds, and they did not seem to be able to break down this tendency. The two rats that had failed the unit alike maze after operation, also failed to learn this maze. #12 died after 30 trials in the maze, but during this time showed no signs of learning. Both

rats who learned the maze,#4 and 54, had relatively small lesions, largely centered in area p. We realize the records of 9 rats are not enough to give more than a suggestion,but it looked as if the swing factor was what made for difficulty in the case of the operated rats.

We have already mentioned that two factors made for unusual difficulty in this maze: the difficulty in obtaining differential floor cue and the swing factor. With this in mind, those rats that were still alive were tested in unique unit maze #2(Fig.3) The maze was set up on Shepard's big maze platform, 2" thick and covered with asphaltic linoleum 3/16" thick, cut into 12" squares. Here light factors were controlled; the maze was illuminated by 100 small shaded lights, 24" o.c. in each direction. So there was little chance for light cues in the learning of the maze, a factor making for greater difficulty in this maze. Also, the five units of this maze were longer and the pattern more complicated geometrically than in the unique unit maze #1. On the other hand, the swing factor was absent here and the floor more conducive to giving a differential floor cue.

Four rats who had failed to learn the unique unit #1 maze after operation, #40,44,46 and 48, were tested in this maze along with four other rats, #43,52,53 and 37; of the latter four, 53 had failed in the Lashley maze and 37 failed to learn any maze in which he was tested after operation. Of the first four, #40 formed one persistent error, blind #2; but did not enter the blind toward the end of his training period in about three out of every ten trials.

This rat is considered a partial failure. #48 failed this maze also, forming persistant errors. The other two who had failed the unique unit maze #1 were able to learn this maze, as also #52,53, and 43 were able to learn the maze. So we have one partial and two complete failures out of 8. Again we have too few cases to justify any conclusions, but apparently this maze was easier for the rats than was the other unique unit maze. Whether it was the swing factor or the floor cue that made for greater difficulty, or both, we cannot say. We had no pre-operative records for this maze, so #100 and UAF_3 were run here, both learning the maze much more readily than any of the operated rats. These two rats were also run in the Lashley maze for purposes of comparison(Table 2 shows their scores).

The results from all the mazes show rather conclusively that for the same rat with the same lesion, there is wide variation in the amount and kind of deterioration of performance in the different maze situations used here. No one maze tells the whole story of the operation. The behavior of the rat in any one maze can give a quantitative measure of the deterioration of performance in that particular maze due to the operation, but it does not show of what the retardation consists in terms of the processes involved. Certainly the operation does not produce a general disorganization that affects all mazes alike. Nor can we speak of "the maze situation" as if all mazes were qualitatively similar. In order to determine the specific processes involved, we must study the performance of a large number of normal rats in a wide variety of situations, each of which has been previously analyzed, so that we can

equalize every factor save one and study that one variable before and after operation. The operations too should be controlled in so far as possible, effort being made to destroy totally and exclusively each of the cortical functional areas and the various cerebral nuclei, in a large number of cases. With such a beginning we can hope to obtain positive and specific results from the extirpation method. Also the questions Lashley raises as to the possible effect of the factors of general nutritive changes, shock, diaschisis, etc., must be answered.

No conclusions can be drawn as to the function of any cortical area from the operations in this experiment. This was not the purpose of the experiment, and in those few cases we have that roughly approach total and exclusive destruction of area p, we find no clear-cut suggestions. We can say only that almost complete destruction of area p does not reduce Type A rats to Type B. This was also true for all the other areas included in our lesions, but the areas other than area p were covered much less thoroughly. We have 45 cases tested pre- and post-operation in the unit alike maze and treated sufficiently alike to justify statistical treatment. In all such treatment we have used the error scores, since these seem much more representative than the trial scores. We have computed the coefficient of correlation by the rank order method for the relation between extent of lesion and number of errors made in the unit alike maze subsequent to operation: we find p to be .13 with a $P.E._r$ of .10. For the relation between extent of cortical lesion and number of errors in the Lashley maze in

the case of 17 rats, we find p to be .81 with a P.E.$_r$ of .056. For such value as they may have, we have computed the rank order coefficient of correlation for the relation between the number of errors made in the unit alike maze pre- and post-operation; we find p to be -.29, with a PE_r of .11; and for the relation of pre-operative retention errors to post-operative retention errors, we find p to be .08 with a PE_r of .16. We have also computed the relation between original learning in the unit alike maze and pre-operative retention; we find p to be .58 with a PE_r of .11.

It looks as if our results may agree with Lashley's findings that the amount of cortical destruction anywhere makes for proportional difficulty in the performance of the Lashley maze, but we have not enough cases to be certain. The results show rather conclusively that no such relation obtains for the unit alike maze. In most cases where the rat had difficulty in learning the Lashley maze after operation, we find some striatal destruction, but again we find striatal involvment accompanying those cases that had little difficulty in learning this maze. Again we lack adequate evidence in this respect.

The data do reveal clearly that for the unit alike maze these operations did not destroy the habit, as the rats required more than twice as much practice to learn the maze originally as they required for post-operative retention. However we find a greater loss after operation plus a ten day interval than after a ten day interval alone. So there is a distinct lowering of efficiency in performance of the habit due somehow to the operation, but not proportional to the extent of cortical lesion. We cannot

tell what proportion of the reduction is due to cortical and what proportion to subcortical destruction. There is just a suggestion in the results that the higher error scores are accompanied by mild striatal involvement. More work must be done before this question may be settled definitely.

Lashley points out many other uncontrolled factors that might effect performance in the various mazes after operation. It is impossible for us to say anything in regard to the factor of diaschisis, and the factor of shock, general or specific. As to the presence of scar tissue in itself producing so potent a change in the total functioning of the animal as to seriously affect the results, if such should prove to be the case, then the whole experimental technique of extirpation is beside the point for no operated rat can be compared with an unoperated rat with but one variable in the balance; one could not separate the effects of absence of neural tissue from the presence of scar tissue. But if any of these factors are effective in these results, they must be much more potent in the Lashley maze than in the unit alike maze, for the operations in some way completely reverse the normal order of difficulty of learning the two mazes, and have an effect on performance in the Lashley maze out of all proportion to that in the unit alike maze. It is possible that the cortex dominates, or facilitates the performance in the unit alike maze much less than that in the Lashley maze, and thus Lashley's findings of mass action would not show here.

We have shown that the rat reaches the criterion

for the learning of the unit alike maze with much less practice after operation than before. Certainly it seems as if there must be some retention from original learning, for it is not likely that removal of brain tissue would enable to learn this maze more quickly. Herrick has suggested that such might be the case for very simple habits, but the unit alike maze would not seem to fall into this class. On how much higher a score does the rat start after operation, due to his original learning? We counted the number of errors made by those rats with complete pre- and post-operative records and found that the average number of errors made in the first six trials in original learning was 12.3 while the average number of errors made in the first six trials after operation was 9.5. But the difference is greater than these figures would indicate, for, in the original learning, the first six trials were always from c to a, whereas in the post-operative performance, the rat was inserted twice in c, twice in b and twice in a. Thus a chance score for the first six trials in original learning would involve less errors than a chance score in the first six trials of post-operative performance.(Figures 1 and 2) Therefore we conclude that the rats retained something from original learning of the unit alike maze.

The few results available suggest that the effects of the lesions may be even more severe in the case of the unique unit mazes than in the Lashley maze, especially unique unit maze #1.

Several attempts were made to deafen rats, leaving them otherwise intact, in an effort to determine whether

or not the unit alike maze is learned on an auditory basis. We found it impossible to sever the cochlear branch of the auditory nerve, or to sever the eighth nerve as a whole, and keep the rat alive.

Several attempts were made to appreciably reduce the hearing of the rat by Watson's method of destroying the tympanic membrane, dislodging the ossicles, then filling the middle ear with wax. But we were able to observe no reduction in auditory sensitivity; the rats responded reflexly to a variety of sounds, when precaution was taken to eliminate all other than auditory cues. #30 was one of the rats thus treated; one hour after operation it ran the unit alike maze with an errorless score.

Still another attempt was made to deafen rats, leaving them otherwise intact. Six rats were put under deep ether anesthesia. Then the tympanic membrane on both sides was destroyed, the ossicles dislodged, and the curved point of an electro-coagulator was inserted into the middle ear. This was pressed against the bone in the direction of the inner ear as previously determined by several dissections. A small amount of bony tissue was destroyed. Four of the six rats lived after the first few days, #55, 56, 57 and 58. #55 was in very poor physical condition after operation, and held her head on one side. The outer ear on both sides atrophied and never grew back. This rat never made more than three errorless runs in succession, and so is considered a partial failure. #56, 57 and 58 recovered well from this operation and learned the unit alike maze with scores below average. All three were then given pre-operative retention tests. #56 made no errors

in pre-operative retention, but #57 and 58 required many more trials than they had required for original learning, as well as many more errors, suggesting a progressively deteriorating ear disturbance. Then #56,57 and 58 were subjected to cerebral operation(Tab. 2 shows per cent of destruction in each case). All three showed distinct retention from original learning before operation, with scores below average, thus refuting the suggestion of progressively deteriorating ear disturbance, if the unit alike maze is learned on an auditory basis. We cannot explain the pre-operative retention scores of #57 and 58. We can only say, after Loucks, that it is better never to have learned than to have learned and lost. Tests seemed to show reflex auditory responses in the case of #57,58 and 56, but not in the case of #55. We are by no means convinced of the adequacy of these tests. The ears of #56, 57 and 58 have been preserved and will be studied in an effort to determine if any of the inner ear was actually destroyed.

These attempts raise questions rather than answer them, but this whole study is incidental, aside from the main point of the experiment, and no particular emphasis is laid upon it.

Conclusions

1. Within the limits of this experiment, no locus or extent of cerebral destruction was found which could reduce Type A rats to Type B.

2. Area p is not essential for the retention of the unit alike maze learned before operation.

3. Within the limits of this experiment, no correlation was found between the amount of area p left intact and amount of retention of the unit alike habit learned before operation.

4. No correlation was found between extent of cortical destruction and performance in the unit alike maze after operation.

5. No one maze situation offers an adequate measure, qualitative or quantitative, of the effect of the cerebral destruction.

It is suggested that in order to answer the many questions raised by the experimental work with cerebral extirpation, we must have at our command a variety of problem situations, each completely analyzed for the normal animal, with but one variable in each test situation. In such situations we can then test the operated rats, varying the lesions in amount and locus to answer the question of mass action and of equipotentiality raised by Lashley, destroying each of the primary areas as totally and exclusively as possible to determine the function of each area.

It is also suggested that an important factor affected in these operations may have been in kinesthetic

terms. This factor has not yet been analyzed completely for the normal animal, but we mean by this no mystical unknown. It is partly demonstrated by Shepard's studies of the swing factor, the cues used by the type B rats, etc.

BIBLIOGRAPHY

Bibliography

Brodmann, K. 1909 Vergleichende Lokalisationslehre der grosshirnrinde. Leipsig.

Cameron, N. 1928 Cerebral destruction in its relation to maze learning. Psychol. Monog.,vol.39,pp.1-68.

Campbell, [illegible] 1907 Histological studies on localization of cerebral function. Cambridge

Clark, W.E.Von Gros 1932 An Experimental study of the thalamic connections in the rat. Philos.Trans. of Roy. Soc. of London, series B, vol.222,pp.1-28.

Craigie, E.H. 1925 Finer anatomy of the central nervous system based upon that of the albino rat.Philadelphia.

Fortuyn, A.E.B.D. 1914 Cortical cell lamination of the brains of some rodents. Arch.Neurol.Psychiat., London, vol.6,pp.221-354

Franz, S.I. 1911 On the functions of the cerebrum: the occipital lobes. Psychol.Monog.,vol.13,pp.1-118.

1907 On the functions of the cerebrum: the frontal lobes. Arch. of Psychol.,no.2, pp.1-64.

1915 On the functions of the cerebrum: variations in distribution of the motor centers. Psychol. Monog.,vol.17,pp.80-162.

Franz,S.I., and Lashley,K.S. 1917 Retention of habits by the rat after destruction of the frontal portion of the cerebrum. Psychobiol.,vol.1,pp.3-18.

Gurdjian, E.S. 1927 The diencephalon of the albino rat:studies of the brain of the rat#2. Jour.Comp.Neurol. vol.43,#1.

Isenschmid, [illegible]. 1911 Zur kenntnis der groszhirnrinde der maus. Abhand. K. Preuss. Akad., Abhand.3,p. 1.

Hunter, W.S. 1930 A consideration of Lashley's theory of the equipotentiality of cerebral action. Jour. Gen. Psychol.,vol.3,pp. 455-468.

Jacobson, C.F. 1931 A study of cerebral function in learning: the frontal lobes. Jour.Comp.Neurol.,vol.52,p.271.

Liggett, J.R., and Liggett, M.W. 1927 On the modification of the rate of learning of the white rat following removal of the olfactory lobes. Ped. Sem. and Jour. Gen. Psychol.,vol.34, pp. 525-536.

Lashley, K.S. 1920 Studies of cerebral function in learning. Psychobiol.,vol.2, pp. 55-135.

Lashley, K.S. 1921a Studies,.....II. The effects of long continued practice upon cerebral localization. Jour. Comp. Psychol.,vol.1, pp.453-468.

1921b Studies,.....III. The motor areas. Brain, vol.XLIV, pp. 255-286.

1922 Studies,.....IV. Vicarious function after the destruction of the visual areas. Am. Jour. Physiol., vol.59, pp.44-71.

1923 Temporal variation in the function of the Gyrus precentralis in primates. ibid.,vol.65,pp.585-602.

1924a Studies,.....V. The retention of motor habits after the destruction of the so-called motor areas in primates. Arch. Neurol. and Psychiat., vol. 12, pp. 249-276.

1924b Studies,.....VI. The theory that synaptic resistance is reduced by the passage of the nervous impulse. Psychol. Rev. vol.31, pp. 369-375.

1926 Studies,.....VII. The relation between cerebral mass, learning and retention.Jour. Comp. Neurol., vol.41, pp. 1-58.

1929 Brain mechanisms and intelligence. Chicago.

1930 The mechanism of vision. II. The influence of cerebral lesions upon the threshold of discrimination for brightness in the rat. Jour. Genet. Psychol., vol.37, pp.461-480.

1931a Cerebral control versus reflexology. Jour. General Psychology, vol.5, pp. 3-20.

1931b The mechanism of vision IV. The cerebral areas necessary for pattern vision. Jour. Comp. Neurol., vol. 53, pp. 419-478.

1932 Studies,.....VIII. A reanalysis of data on mass action in the visual cortex. ibid.,vol.54,p.77.

1933 Integrative functions of the cerebral cortex. Psychol. Rev., vol.13, #1.

Lashley, K.S. and Ball, J. 1929 Spinal conduction and kinesthetic sensitivity in the maze habit. Jour. Comp. Psychol. vol.9 pp.70-106.

Lashley, K.S. and Frank, M 1932 Studies,.....IX. The lateral portions of the area striata in the rat. A correction. Jour. Comp. Neurol. vol.55, p.525.

Lashley, K.S. and Franz, S.I. 1917 The effects of cerebral destruction upon habit formation and retention in the albino rat. Psychobiol., vol.1, pp. 71-139.

Lashley, K.S. and McCarthy, D. 1926 The survival of the maze habit after cerebellar injuries. Jour. Comp. Psychol. Vol 6, pp. 423-433.

Lashley, K.S., McDonald, W.T., and Peters, H.N. 1933 Studies,.....X. The effects of dilatation of the ventricles on maze learning. Am. Jour. Physiol., vol. 104, #1.

Loucks, R.B. 1931 The efficacy of the rat's motor cortex in delayed alternation. Jour. Comp. Neurol., vol. 53, pp. 511-567.

Maier, N.R.F. 1932a The effect of cerebral destruction on reasoning and learning in rats. Jour. Comp. Neurol., vol. 54, pp. 45-75.

1932b Cortical destruction of the posterior part of the brain and its effect on reasoning in rats. Jour. Comp. Neurol., vol.56, pp. 179-214.

Lashley, K.S. and Wiley, L.E. 1933 Studies of Cerebral Function in Learning IX Mass action in relation to the number of elements in the problem to be learned. Jour. Comp. Neurol.,vol. 57, pp.3-55.

Wiley, L.E. 1932 The function of the brain in audition. Jour. Comp. Neurol., vol.54, pp. 1o9-141.

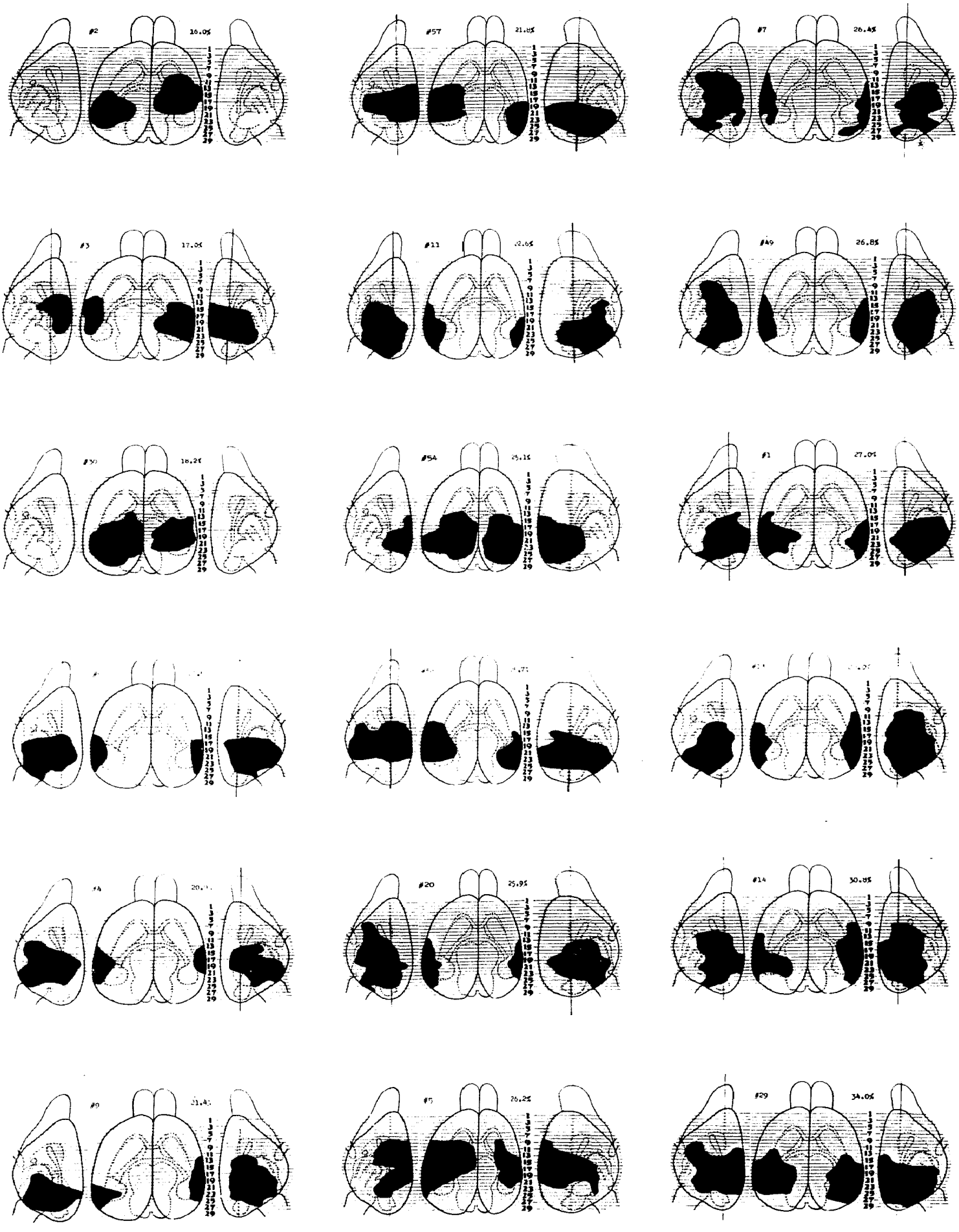
Woollard, . 1925 The cortical lamination of Tarsius. Eng. Jour. of Anatomy, vol. 60, p. 86.

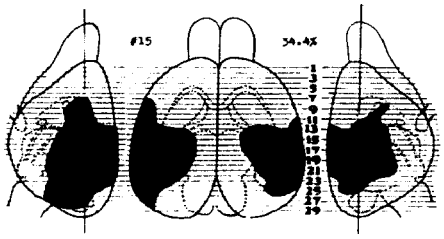
Zunino, G. 1909 A study of the myelino-architecture of the rabbit. Jour. fur Psychol. and Neurol. Bd.14, p.38.

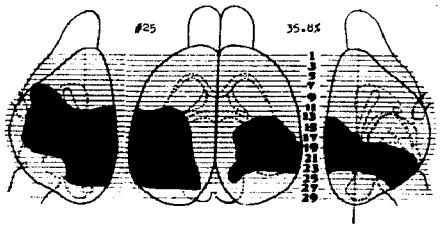
Shepard, J.F. 1926 An unexpected cue in maze learning. Psychol. Bull., vol.26, p.164.

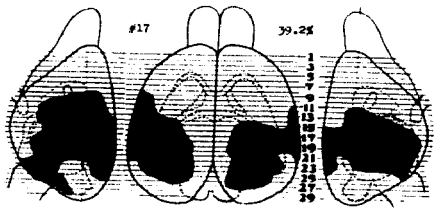
1931 More learning. Psych. Bull., vol.28, p.240.

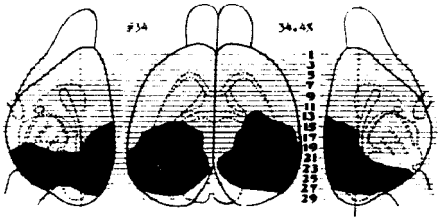
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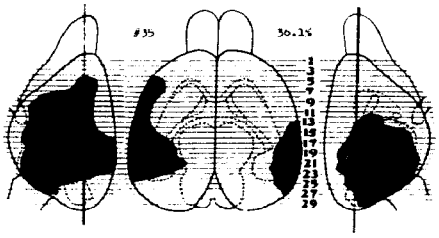

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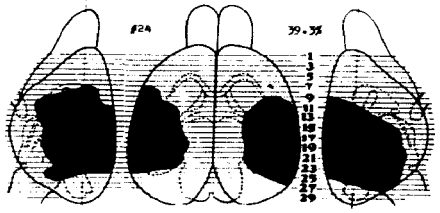

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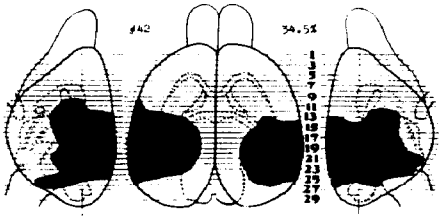

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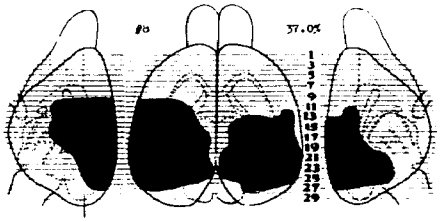

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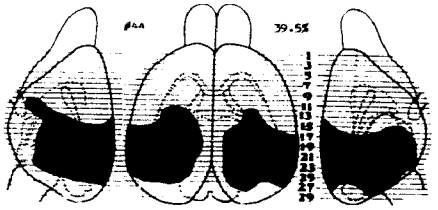

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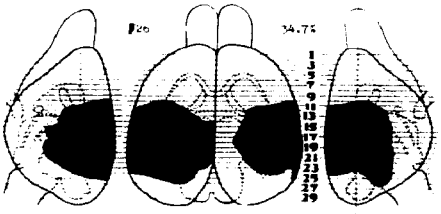

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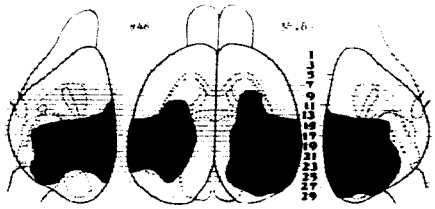

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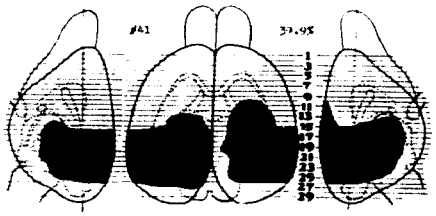

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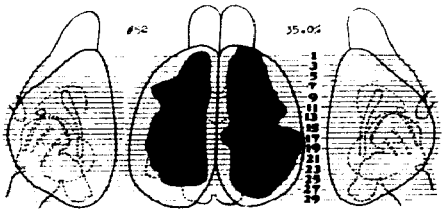

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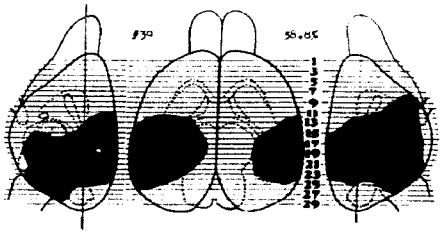

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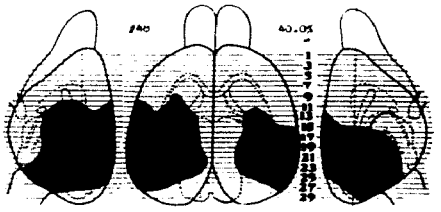

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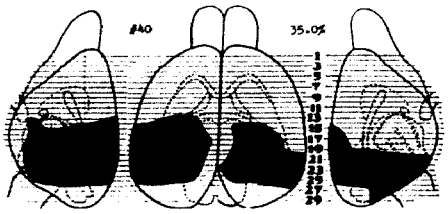

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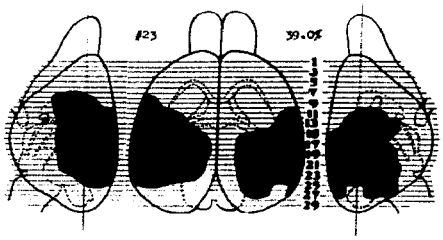

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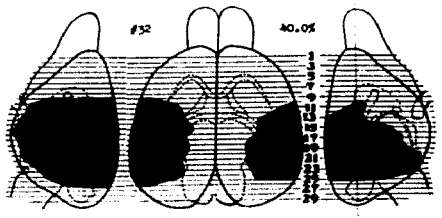

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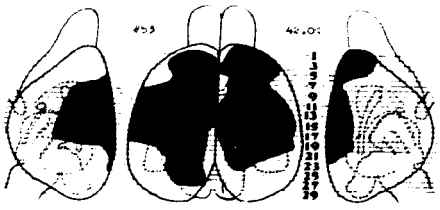

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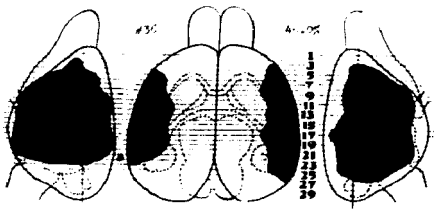

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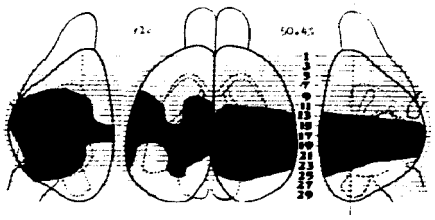

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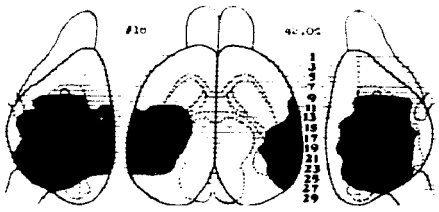

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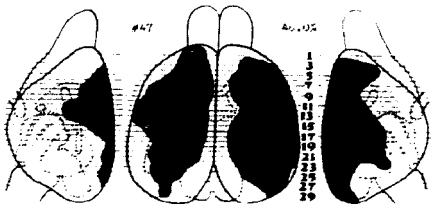

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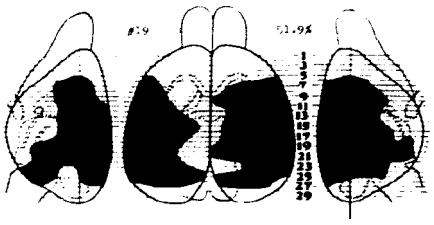


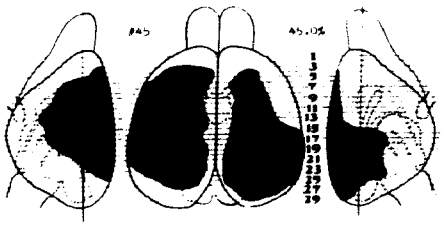


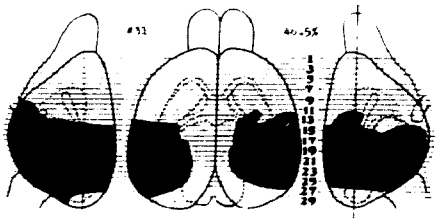


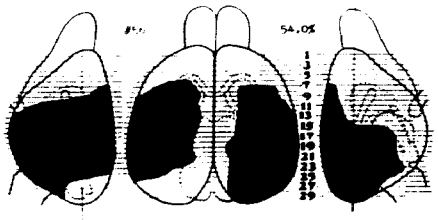


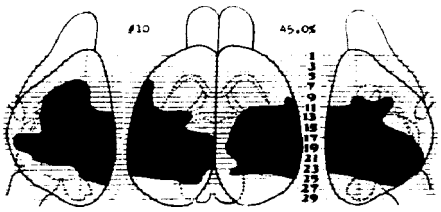


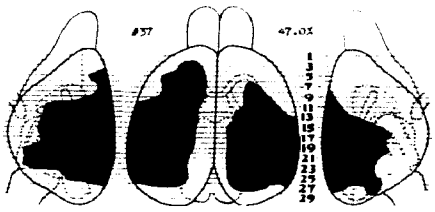


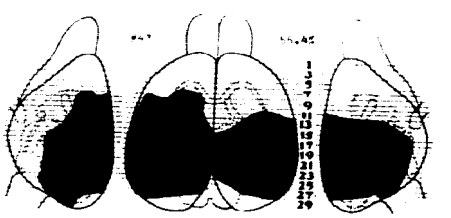


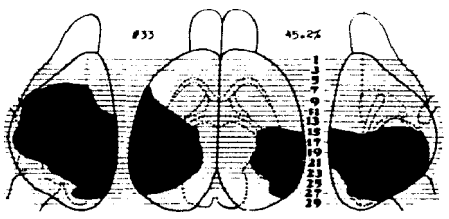


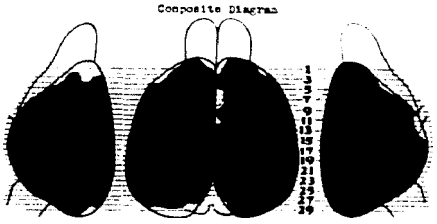












Composite Diagram