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Localization of the "maze memory system" in the white rat*

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Adult albino rats, previously trained on a three-cul maze, sustained bilateral cortical or subcortical lesions and were subsequently tested for retention. Those animals showing defective retention suffered damage to either the anterior neocortex, posterior neocortex, cingulate cortex, corpus striatum, hippocampus, septofornix area, thalamus (anterior, lateral, ventromedial, or posterior divisions), posterolateral hypothalamus, mamillary bodies, subthalamus, red nucleus, substantia nigra, central tegmentum, ventral portions of the brainstem reticular formation, or the cerebellum. Excellent retention was observed following damage to either the amygdaloid complex, rostral medial forebrain bundle, dorsomedial thalamus, or dorsal midbrain. These results, coupled with earlier findings, suggest that the maze habit is dependent upon the activities of three functional blocks of the brain: the first block (brainstem reticular formation) has integrative functions, the second block (sensorimotor cortex, cingulate cortex, cerebellum, and thalamus) has kinesthetic functions, and the third block (occipital cortex, hippocampus, septofornix area, and mamillary bodies) plays a role in the discrimination of spatial cues.

As discussed earlier (Thompson & Thorne, 1973), the lesion method provides one of the most straightforward and reliable ways to determine whether or not a particular part of the brain is essential for the "memory" (expression) of a previously learned response. Thus, by canvassing many different cortical and subcortical areas with small lesions (using, of course, different Ss for different areas), it becomes possible to map the "memory system" of the brain for any given learned response. Data already exist on the mapping of the "visual" memory system (Thompson, 1969; Thompson & Thorne, 1973), "kinesthetic" memory system (Thompson, Lukaszewska, Schweigerdt, & McNew, 1967; Thompson, Malin, & Hawkins, 1961; Thompson & Thorne, 1973), "manipulative response" memory system (Spiliotis & Thompson, 1973), and the "conditioned avoidance response" memory system (Rich & Thompson, 1965; Thompson, 1964; Thompson, Rich, & Langer, 1964) in the white rat. The purpose of the current study was to map the "maze" memory system in this animal.

Despite the fact that the maze was one of the first laboratory tasks used to study brain functions in learning and memory (Lashley & Franz, 1917), it has been employed only sparingly in the assessment of subcortical contributions. To some extent, this state of affairs stems from Lashley's (1929, 1943, 1950) neuropsychological analysis of the maze habit which, on the one hand, tended to discredit, or at least deemphasize, the role of the interior parts of the brain in learning and memory and, on the other, tended to localize the mechanisms of learning and memory within the confines of the cerebral cortex. Further discreditation of the role played by subcortical structures in maze learning came from an experiment by Brown and Ghiselli (1938). Using the lesion method, these investigators concluded that "no subcortical nucleus or tract was found which could be considered to be necessary or essential for the learning of the alley maze [p. 42]."

The results of more recent studies, however, provide the basis for reconsidering the importance of the interior parts of the brain in the performance of the maze habit. One of the first studies to implicate subcortical structures in retention of the maze was reported by Kaada, Rasmussen, and Kveim (1961). Contrary to what Lashley (1943) reported, these investigators found that lesions of the hippocampo-fornix system impaired maze performance. Thomas (1971) and many others have subsequently confirmed the finding that hippocampo-fornix lesions lead to deterioration of the maze habit. Other subcortical structures, such as the caudate nucleus (Potegal, 1972), cingulate cortex (Kaada et al, 1961), and mamillary bodies (Kaada et al, 1961), may also be concerned with the execution of a maze habit.

In the current study, virtually every part of the tel-, di-, mes-, and met-encephalon was examined as to its contribution to the performance of a maze habit. Because rats with certain subcortical lesions either have difficulty surviving the stress of prolonged water (or food) deprivation or, if they do survive, often show motivational-emotional involvements during testing (see Spiliotis & Thompson, 1973), it was decided to employ a three-cul maze utilizing the motive of escape from footshock.

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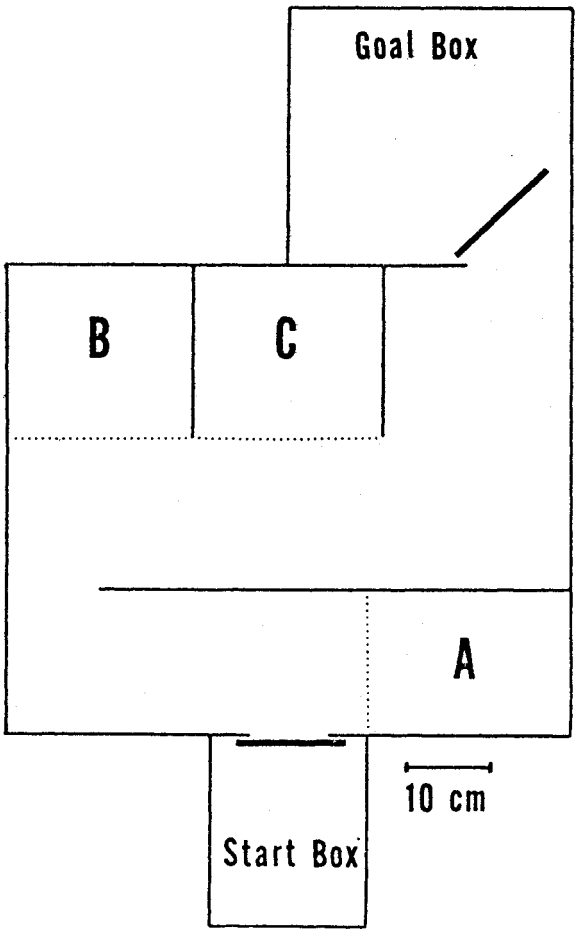

Fig. 1. Floor plan of the maze showing boundaries between the true path and each blind (fine dotted lines) along with the startbox and goalbox doors (heavy solid lines).

METHOD

Subjects

Ninety-three adult male albino rats of the Wistar strain were used. Prior to experimental training, each S was handled periodically and checked for eye disease and middle ear infection. Preoperatively, the Ss were usually housed (two per cage) in regular medium-size wire cages containing a constant supply of food and water. During the first few days following surgery, each S was allowed to recover in a separate cage, but was subsequently paired with another operated (or control) S at least 1 week prior to the retention test. Only five Ss died prior to the retention test. Two other Ss were discarded from the experiment because their lesions were grossly asymmetrical.

Apparatus

A three-cul maze utilizing the motive of escape (or avoidance) of footshock was employed (see Fig. 1). The startbox and the maze proper contained a grid floor, whereas the goalbox floor was made of wood. The walls were 25.4 cm high. All parts of the maze were painted flat black, except for the grid floor and the Plexiglas ceiling.

The maze was housed in a sound-attenuated room and illuminated by ceiling lights. Olfactory cues and extramaze visual cues were not controlled. However, sensory privation groups were tested in this study (see Results), and revealed that neither visual nor olfactory cues were essential for retention of this maze.

A Thompson-Bryant (1955) discrimination box was used in preliminary training to develop and shape efficient escape responses to footshock.

Procedure

On the day following preliminary training, S was placed in the goalbox of the maze for 5 min. A card blocked the goalbox entrance to prevent S from entering the maze proper. Subsequently, S was placed in the startbox and the guillotine door was raised. Failure to leave the startbox within 5 sec was followed by brief footshocks. No further footshocks were given unless S made an error (entered a blind alley by at least the length of its head and thorax) or stopped forward progression in the maze. Upon entering the goalbox, the card was positioned to prevent reentry into the maze.

Four trials were given daily with an intertrial interval of 75 sec. Training was terminated when S reached the criterion of 2 successive errorless days.

Both initial and repetitive (reentries into blind alleys) errors were recorded for each trial. However, since the majority of repetitive errors occurred during the first day of preoperative (and postoperative) training, only initial errors were used as an index of performance. Thus, a maximum of three errors per trial could be committed, one for entry into each of the three blind alleys.

From 4 to 24 h after learning, the majority of Ss sustained brain damage. Four Ss served as normal controls and an additional four as sham operate controls. The latter underwent anesthesia, placement within the stereotaxic headholder, exposure of the skull, and suturing without any further treatment.

Following a 2-3-week recovery period (or rest period in the case of the normal controls), each S was required to relearn the maze. The procedure was identical to that described in original learning. Retention was measured in percentage error savings, using the conventional formula.[1]

Surgery and Histology

Bilateral lesions were customarily carried out in one stage under deep chloral hydrate anesthesia. For cortical lesions, the suction method was used. Subcortical lesions were made stereotaxically with reference to the rat atlas of Massopust (1961). Depending upon the area to be destroyed, a constant (anodal) current, ranging from 2 to 5 mA for a duration of 5-15 sec, was passed through an implanted stainless steel electrode with 1.0-2.0 mm of the tip exposed. In some cases, lesions were performed in two stages, with an interoperative period of 7-10 days. Details of the surgical procedure have been reported elsewhere (Thompson, 1971).

Upon completion of the retention test, each operated S was sacrificed with an overdose of Nembutal, its vascular system perfused with 10% Formalin, and the brain removed and stored in 10% Formalin for 2 days. For cortical injuries, the lesion was reconstructed on Lashley-type brain diagrams prior to sectioning. Each brain was sectioned frontally at 90 microns with the use of a freezing microtome. Every third section showing the lesion was photographed by using the section as a negative film in an enlarger (see Thompson, 1971). These photographs of unstained sections yield differentiation of the brain field similar to that obtained with a fiber stain, and permit ready identification of the three major zones of the lesion—the vacuolated area, the rim of severely coagulated tissue, and the surrounding gliosis. Description of the lesions in the text, however, will be made only in terms of the central necrotic region (vacuolated and severely coagulated areas).

RESULTS

Original Learning

All Ss succeeded in reaching the criterion run within 8

days. Mean days and errors to criterion were 3.4 and 15.2, respectively. It is important to note that the three blind alleys were not of equal difficulty. Mean errors on Blind Alleys A, B, and C were 5.4, 4.2, and 5.6, respectively. The Wilcoxon test for paired replicates (Siegel, 1956) disclosed that significantly fewer errors were made at Blind Alley B than at either Blind Alleys A or C ($p < .01$). The difference in errors between Blind Alleys A and C fell considerably short of statistical significance.

Figure 2 depicts performance at each blind alley (and composite performance for the maze as a whole) as a function of practice. These curves were obtained by dividing the total number of precriterion trials for each S into quarters (Vincent fourths) and computing mean errors committed during the first, second, third, and last quarter. (The fifth point on the abscissa represents criterion performance.) While the learning curve for the maze as a whole reveals a not untypical sigmoid reduction in errors as a function of practice, the component learning curves provide a picture of the learning process which is appreciably more complex. During the initial quarter of practice, the Ss committed the greatest number of errors at Blind Alley B, the fewest at Blind Alley C, and an intermediate number at Blind Alley A—all differences were significant beyond the .01 level. During the last three quarters, however, the frequency of errors was greatest at Blind Alley C, least at Blind Alley B, and intermediate at Blind Alley A. When considering combined errors made during the last three quarters of training, all differences were significant at least at the .05 level.

It is important to note at this juncture that the maze used in the current study yielded a pattern of errors which is not unlike that obtained with other multiple-unit spatial mazes. According to the early studies on the distribution of errors made in the maze situation (see Munn, 1950), the "centrifugal swing" is the primary error-producing tendency during the initial phase of learning, while "goalbox orientation" and "choice-point expectancy" are the chief error-producing tendencies during the later stages of learning. Thus, the finding that more errors were made at Blind Alley B than at Blind Alleys A or C during the first quarter follows from the fact that the former is the only cul subject to errors based upon centrifugal swing. Similarly, the finding that more errors were made at Blind Alleys C and A than at Blind Alley B during the last three quarters is hardly surprising when it is considered that Blind Alley A "points" to the goalbox more conspicuously than does Blind Alley B and that Blind Alley C, being nearest to the goalbox, would lead to more frequent "anticipatory" entries than at Blind Alley B.

Retention

Control Groups

Retention of the maze habit was excellent in both the normal and the sham-operated control Ss—all achieved error savings scores of 90% or better. Three of the four normal rats and two of the four sham-operated rats earned perfect savings scores. Since these two groups were virtually indistinguishable from each other in retention of the maze, they were combined into one major control group for statistical treatment (see Table 1).

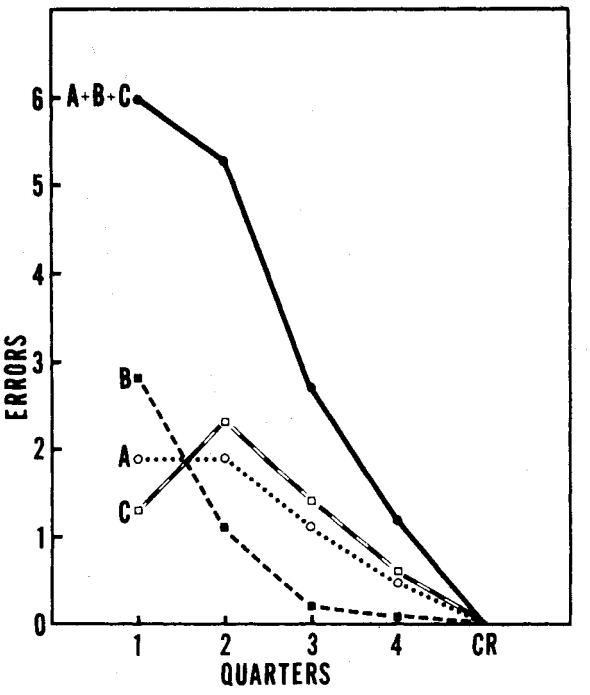

Fig. 2. Performance expressed in errors at each blind alley (A, B, and C) and for the maze as a whole (A + B + C) as a function of practice expressed in quarters of precriterion training.

Peripheral Blinding

Retention of the maze habit was only slightly (though significantly) impaired by enucleation of the eyes (see Table 1). The savings scores earned by the two enucleated Ss compare favorably with those reported in earlier studies investigating the effects of visual privation on maze retention (Lashley, 1929, 1943; Thompson, 1959).

Olfactory Bulbectomy

Three Ss sustained damage to the olfactory bulbs by aspiration. In all cases, the bulbs were completely ablated at the point immediately rostral to the frontal poles of the cerebrum. One S showed perfect retention, and the remaining two earned savings scores of 95% and 56%.

Neocortex

Anterior Region. Two Ss sustained bilateral ablations of the frontal cortex with minimal involvement of either the corpus striatum or anterior limbic area. In both cases, the damaged region included the frontal poles and

Table 1
Mean Learning (Errors) and Retention (Percentage Error Savings) Scores for All Groups

Group	N	Learning Mean	Retention Mean	Retention Range
Control	8	16.3	97.0	90 to 100
Peripheral Blinding	2	14.5	75.5*	64 to 87
Olfactory Bulb	3	21.3	83.7	56 to 100
Neocortex				
Anterior	2	19.0	−191.5*	−229 to −154
Posterior	2	23.5	−209.0*	−311 to −107
Other Telencephalic Areas				
Limbic Cortex	2	16.0	−471.0*	−667 to −275
Amygdala	2	13.5	83.5	73 to 94
Septofornix Area	2	10.0	−170.0*	−186 to −154
Hippocampus	2	14.5	−282.0*	−388 to −176
Corpus Striatum	2	8.0	−950.0*	−1825 to −75
Hypothalamus				
Anterior	4	14.5	58.3	−55 to 100
Midtuberal	4	15.7	−62.5*	−400 to 87
Mamillary Bodies	3	9.7	−339.3*	−518 to 0
Thalamus				
Anterior	2	16.5	−245.5*	−356 to −135
Dorsomedial	3	10.7	81.3	56 to 100
Ventromedial	2	17.5	−409.0*	−433 to −385
Lateral	5	14.0	−159.2*	−529 to 47
Posterior	2	15.5	−258.0*	−453 to −63
Subthalamus	3	12.8	−161.0*	−283 to 0
Brainstem Reticular Formation				
Anterior	3	17.3	−149.0*	−190 to −82
Dorsal	4	14.5	72.5*	35 to 92
Lateral	3	14.3	−2.3*	−130 to 75
Posterior	3	18.7	−100.0*	−225 to −25
Other Brainstem Areas				
Colliculi	4	19.8	81.8	40 to 100
Central Gray	2	17.0	88.0	76 to 100
Red Nucleus	3	15.7	−27.7*	−161 to 78
Substantia Nigra	4	22.2	42.2*	19 to 74
Central Tegmentum	2	11.0	−139.0*	−158 to −120
Cerebellum	2	12.0	−84.0*	−145 to −23

**Differed from the controls at least at the .05 level.*

the major portions of what Krieg (1946) has designated as Areas 10, 6, and 2. The extent of damage to the entire neocortical surface ranged 24%-33%. Both Ss failed to relearn the maze habit within the allotted 10 days, and both made at least one error on every trial of the retention test. These Ss required considerably more than the usual number of footshocks to force escape responses (particularly within the startbox) and displayed exaggerated emotional reactions to footshock, such as running "wildly," leaping upwards toward the lid of the maze, and biting the grid. It is important to note, however, that these Ss continued to make errors on those occasional trials in which no footshocks were applied.

Posterior Region. Two Ss received bilateral ablations of the occipital cortex with minimal involvement of the underlying hippocampus and mesial (cingulate) cortex. In both cases, the lesions completely destroyed Areas 17, 18, 18a, and 7 of Krieg (1946). The amount of damage to the total neocortical surface ranged 48%-58%. Although these Ss were excellent runners in the apparatus,[2] neither was able to relearn the maze within the allotted 10 days. In fact, neither made a single errorless trial during the entire retention test.

Other Telencephalic Areas

Limbic Cortex. Two Ss sustained bilateral medial cortical ablations. In both cases, the lesions extended from the level of the frontal poles to (or including) the retrosplenial cortex. The ablations extended approximately 2.0 mm laterally from the midline (damaging 8%-10% of the neocortical surface) and ventrally to the dorsal hippocampal surface. Figure 3 shows the lesion of one of these Ss. Neither S was able to relearn the maze habit and neither completed a single trial without committing at least one error.

Amygdaloid Complex. The amygdaloid area was damaged bilaterally in two Ss. Both suffered injuries to the corticomedial and basolateral nuclear groups, with the former receiving the greater destruction. The lesions extended the full length of the amygdaloid complex and included interruption of the stria terminalis (Fig. 4). One S exhibited excellent retention (94% savings) and the second showed only a slight loss of the maze habit (73% savings).

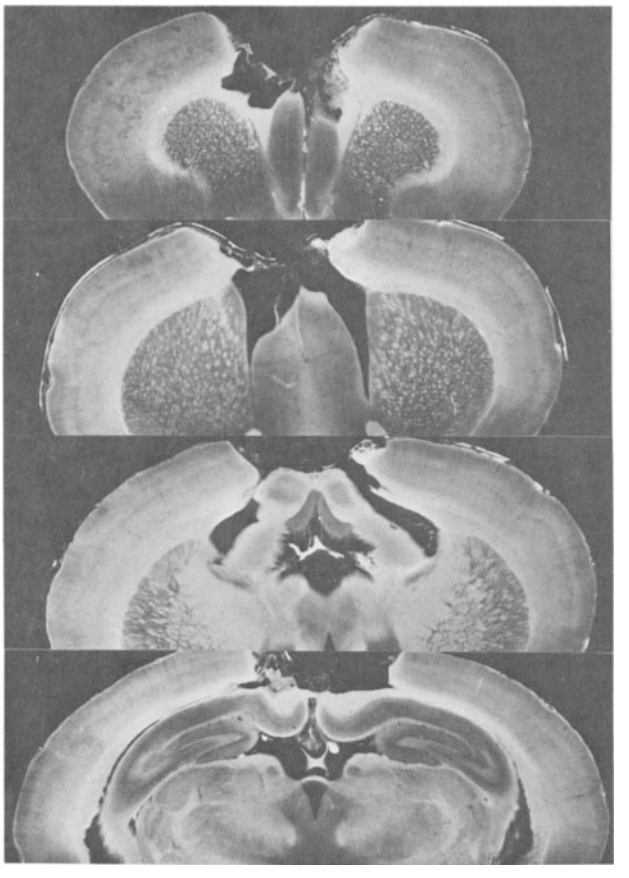

Fig. 3. Four unstained sections showing a cingulate ablation in a rat that failed to relearn the maze.

Septofornix Area. Two Ss sustained septal lesions which destroyed major portions of the medial nucleus and only partially damaged the lateral nucleus. In both cases, the lesions extended caudally to interrupt the descending fornix columns (Fig. 5A). Both Ss exhibited serious losses in retention, requiring more than twice the number of trials (and errors) to relearn the habit as was needed in original learning.

Hippocampus. Two Ss received multiple electrolytic lesions of the dorsal hippocampus. While there was only minimal invasion of the overlying medullary substance, the dorsal thalamus underwent superficial damage (Fig. 5B). Neither S was able to relearn the maze habit, despite the fact that each occasionally performed errorless runs during the last half of training.

Corpus Striatum. In two-stage operations, two Ss received multiple electrolytic lesions of the caudate-putamen-pallidum complex. In one, the lesion began at the level of the anterior commissure and damaged the ventral portions of the corpus striatum. This animal showed a serious retention deficit (–75% savings). The lesion of the second S, which began rostral to the level of the anterior commissure, damaged the more dorsal portions of the corpus striatum with slight involvement of the internal capsule (Fig. 5C). This animal failed to relearn the maze habit and was unable to perform a single errorless trial throughout the retention test. This S showed a defective escape response

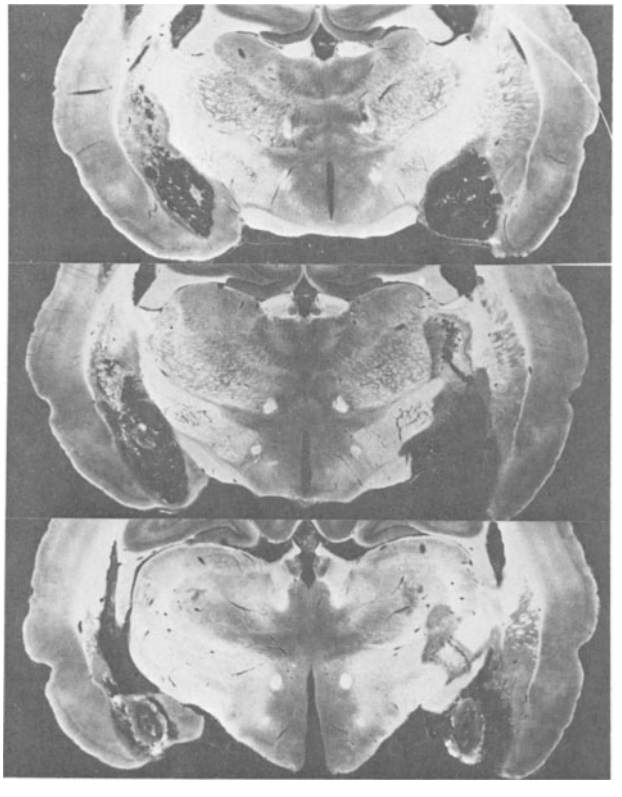

Fig. 4. Three unstained sections showing an amygdaloid lesion in a rat that earned a savings score of 94%.

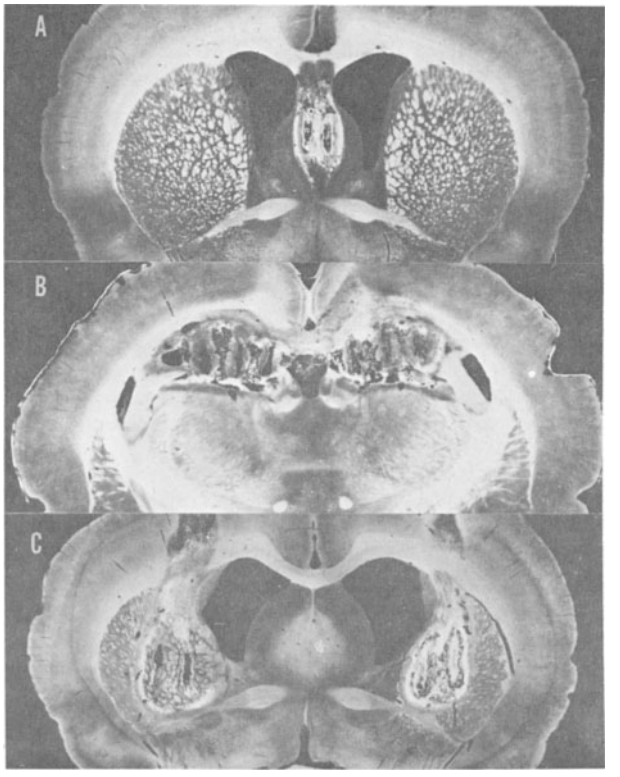

Fig. 5. Unstained sections derived from three rats showing lesions of the septofornix area (A), hippocampus (B), and corpus striatum (C). All three rats earned negative savings scores.

(requiring frequent footshocks to initiate forward progression) and exhibited difficulty in making a correction response once an error was committed, the latter being a reflection of "obstinate progression."

Hypothalamus

Anterior Levels. Two Ss sustained damage to the medial forebrain bundle at pre- and supra-optic levels (Fig. 6A). Both Ss earned savings scores falling well within the range earned by the controls.

Two additional Ss received lesions to the medial supraoptic hypothalamus. The S having the largest lesion (including bilateral interruption of the fornix columns) earned a savings score of –55% (Fig. 6B). The second suffered a discrete lesion between the fornix columns and the optic chiasma (Fig. 6C) and earned a savings score of 95%.

Midturberal Levels. The midline portions of the midtuberal hypothalamus were destroyed in two Ss. In both cases, the lesions extended caudally to invade slight portions of the medial mamillary nuclei. The S with the larger lesion (Fig. 7A) showed a moderate loss in retention (46% savings), while the one with the smaller lesion earned a savings score of 87%.

Two additional rats sustained more laterally placed lesions located primarily between the mamillothalamic tract and the fornix column. The S with the smaller

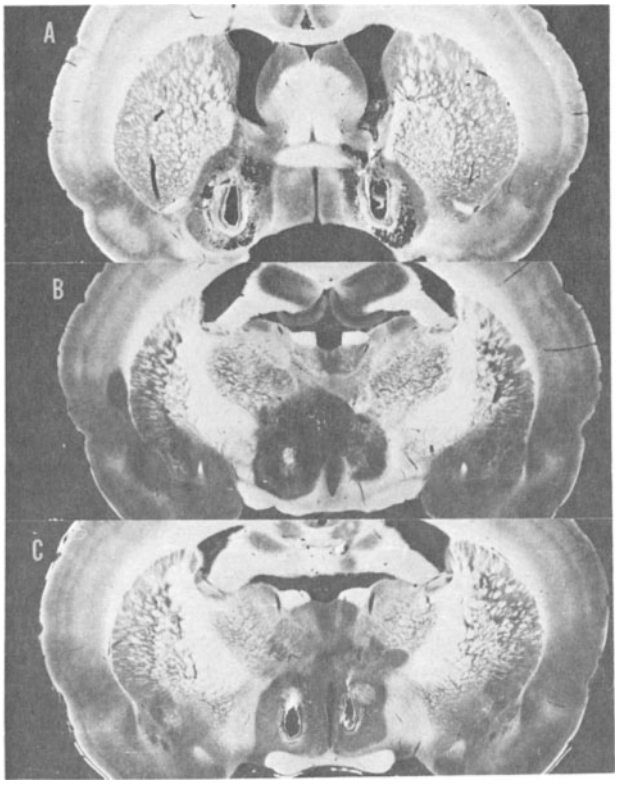

Fig. 6. Unstained sections derived from three rats showing lesions at anterior hypothalamic levels. The rat with damage to the fornix column (B) earned a negative savings score, while the remaining two (A and C) earned savings scores in excess of 94%.

lesion (Fig. 7B) earned a savings score of 17%, while the S with the larger lesion (Fig. 7C) not only failed to relearn the maze, but failed to make a single errorless trial. Throughout the retention test, this animal with the larger lesion required frequent footshocks to force escape responses, exhibited a defective correction response upon entering a cul, and reacted excessively to footshock by running and leaping wildly.

Mamillary Body Levels. Three Ss were subjected to bilateral lesions of the posterior hypothalamus which damaged significant portions of the mamillary bodies. The two Ss with the most laterally placed lesions neither relearned the maze nor completed a single errorless trial. One of these rats (Fig. 8A) was a poor runner throughout the retention test, behaving very much like the animals with frontal cortical lesions described earlier. The second rat (Fig. 8B) showed only a mild disturbance of the escape response. The third rat with a lesion confined largely to the medial mamillary nuclei (Fig. 8C) was an excellent runner, but earned a savings score of 0%.

Thalamus

Anterior Region. Two Ss received bilateral lesions which destroyed portions of the nucleus anterior ventralis, anterior medialis, anterior dorsalis, and reticularis with little or no involvement of the internal capsule (Fig. 9). Neither S was able to relearn the maze habit. However, both showed some improvement in response accuracy toward the end of the retention test, including several errorless trials.

Dorsomedial Region. Three Ss received medial thalamic lesions which destroyed most of the nucleus medialis dorsalis. In all cases, the lesions extended caudally to damage the lateral portions of the nucleus parafascicularis. Two Ss exhibited slight retention losses (Fig. 10A), while the third showed perfect memory of the maze (Fig. 10B).

Ventromedial Region. Two Ss received lesions localized at the base of the thalamus. Injured structures included the mamillothalamic tract and the nucleus ventralis medialis, reuniens, rhombiodeus, gelatinosus, and medialis dorsalis (Fig. 10C). Neither S was able to relearn the maze and neither made a single errorless trial during the retention test.

Lateral Region. The lateral sector of the thalamus at midthalamic levels was damaged in five Ss. The lesions in four were dorsally placed, damaging major portions of the nucleus lateralis and medialis dorsalis. The fifth S had a more ventrally placed lesion, which led to a greater amount of damage to the nucleus ventralis than to the nucleus lateralis. In all cases, the lesions extended caudally to damage the anterolateral portions of the

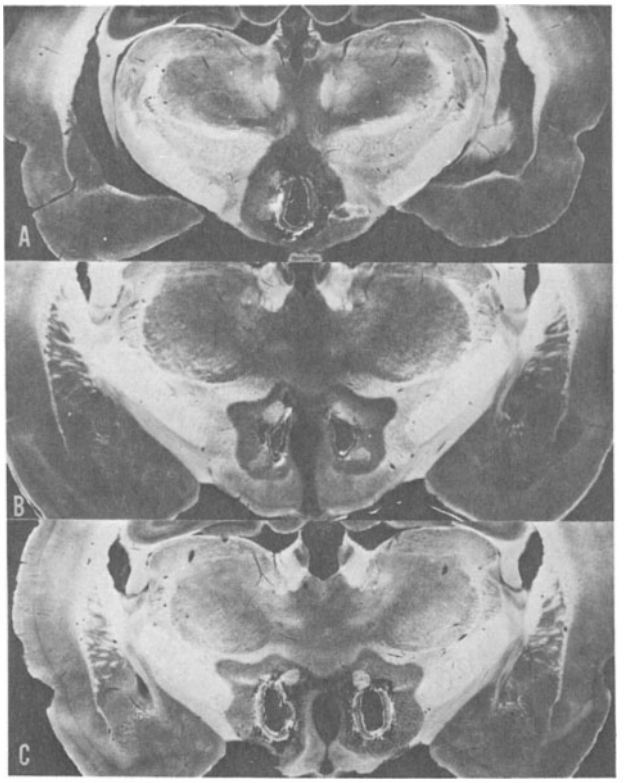

Fig. 7. Unstained sections derived from three rats showing lesions of the midtuberal hypothalamus. All three rats earned savings scores below 47%.

nucleus parafascicularis, but in no case did the lesions extend to the level of the posterior commissure. The S with the ventrally placed lesion earned a savings score of 0%. Of the remaining four, two failed to relearn and two earned savings scores of −14% and 47%. The latter S suffered the smallest lesion of the group. Figure 11 presents the lesions of those three Ss exhibiting the greatest losses in retention.

Posterior Region. Two Ss were subjected to caudal thalamic lesions which damaged the nucleus posterior, parafascicularis, and ventralis. The pretectal and lateral posterior thalamic nuclei suffered only minor damage. One S, with a slightly asymmetrical lesion (which led to unilateral damage to the habenulopeduncular tract), failed to relearn the maze and did not achieve a single errorless run throughout the retention test. The second S, with a symmetrical lesion (Fig. 12), succeeded in relearning the maze, but with a negative savings score.

Subthalamus

The three Ss with bilateral subthalamic lesions failed to relearn the maze habit even though they made frequent errorless runs during the last half of the retention test. In all cases, the subthalamic nucleus was injured along with the zona incerta, Forel's Fields H_1 and H_2, the lateral limb of the medial lemniscus, and the ventrolateral segment of the nucleus ventralis (Fig. 13). In no case did the lesions extend medially into the

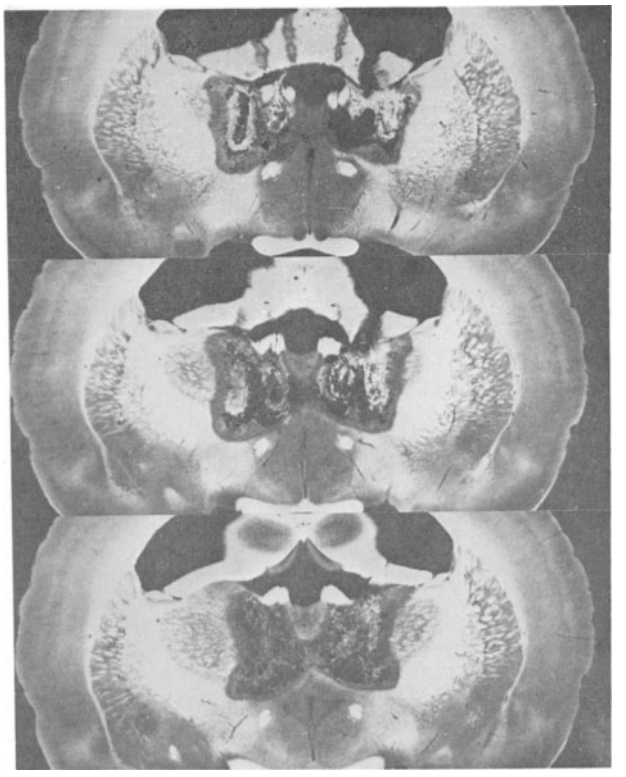

Fig. 9. Three unstained sections showing an anterior thalamic lesion in a rat that failed to relearn the maze.

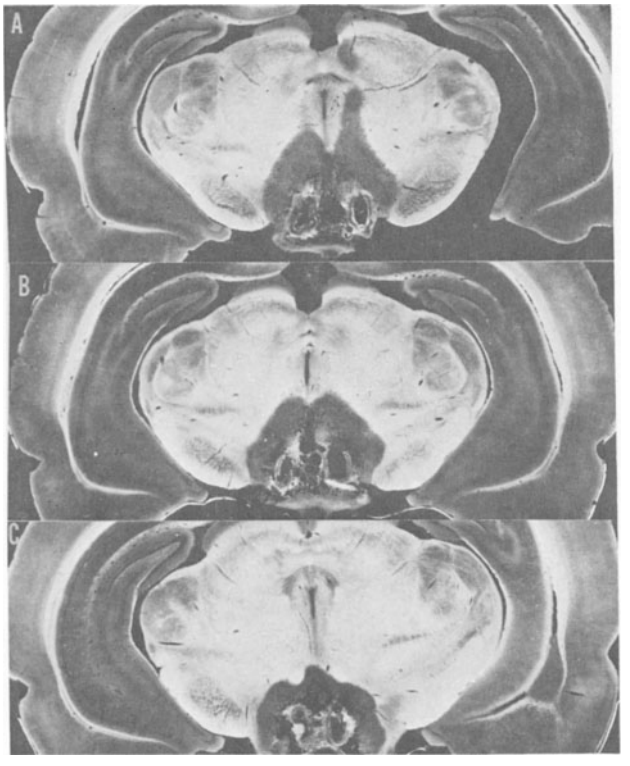

Fig. 8. Unstained sections derived from three rats showing lesions of the mamillary bodies. All three rats earned savings scores of 0% or less.

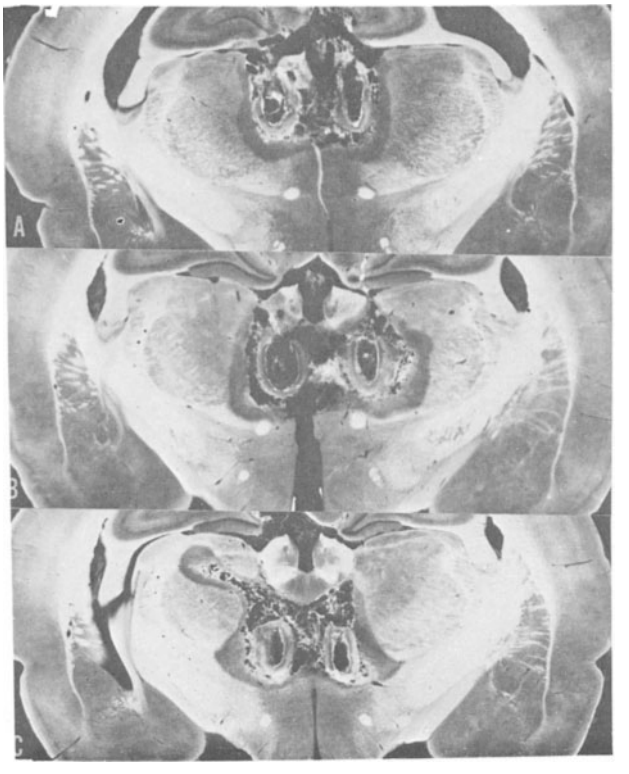

Fig. 10. Unstained sections derived from three rats showing lesions at midthalamic levels. The two rats with dorsal lesions (A and B) earned savings scores in excess of 55%, while the rat with the ventral lesion (C) failed to relearn the maze.

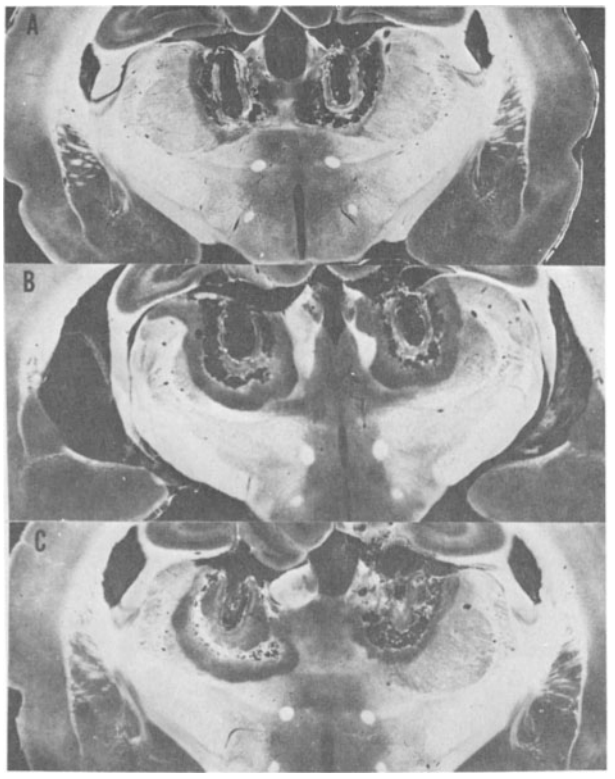

Fig. 11. Unstained sections derived from three rats showing lesions of the lateral thalamus. All three rats earned negative savings scores.

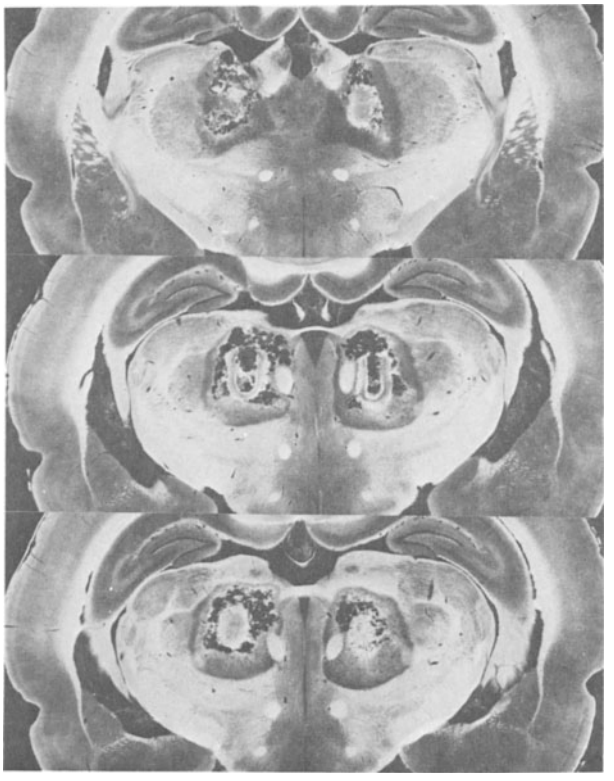

Fig. 12. Three unstained sections showing a posterior thalamic lesion in a rat that earned a negative savings score.

lateral hypothalamic area or caudally into the substantia nigra.

Brainstem Reticular Formation

Anterior Region. Three Ss sustained bilateral lesions to the rostromedial extension of the reticular formation at the dimesencephalic juncture (Fig. 14). In all cases, the lesions began at the level of the anterior limb of the posterior commissure and terminated oral to the red nucleus. Other injured structures included the posterior commissure and its associated nuclei, pretectal area, central gray, and the nucleus posterior and parafascicularis. Although two of these Ss failed to relearn the maze, they did perform several errorless trials toward the end of the retention test. The third S earned a negative savings score.

Dorsal Region. The reticular zone between the central gray and the red nucleus (which, in primates, is occupied by the central tegmental fasciculus) was bilaterally damaged in four Ss. The central gray substance received significant damage, but the red nucleus was largely spared (Fig. 15A). Three of the four Ss attained savings scores in excess of 76%.

Lateral Region. The reticular formation dorsal to the substantia nigra and lateral (or dorsolateral) to the red nucleus was damaged to varying degrees in three Ss. The S with the largest lesion earned a negative savings score

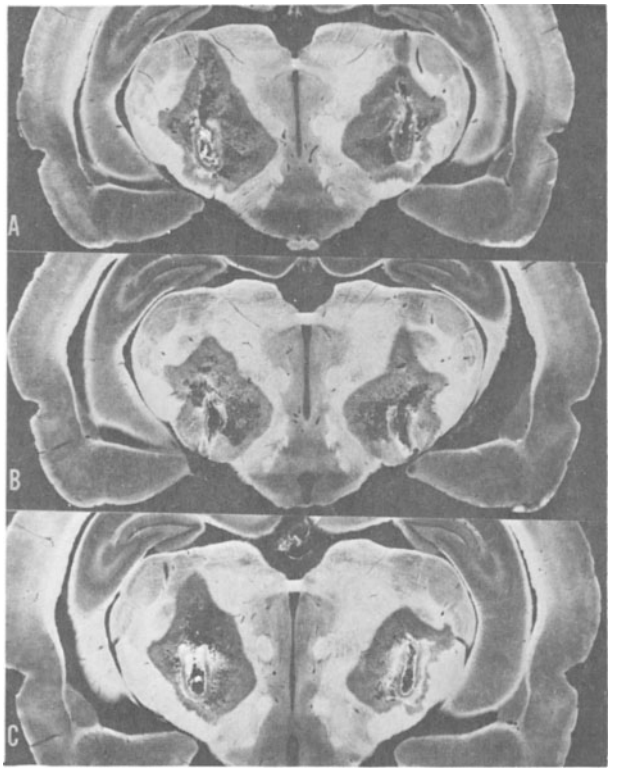

Fig. 13. Unstained sections derived from three rats showing lesions of the subthalamus. All three rats failed to relearn the maze.

(Fig. 15B), while the remaining two earned scores of 48% and 75%.

Posterior Region. Three Ss sustained bilateral lesions to the paramedial portions of the reticular formation at caudal mesencephalic levels. In all cases, the lesions extended caudally to destroy significant amounts of the nucleus reticularis pontis oralis. One S was unable to relearn the maze habit and failed to perform a single errorless trial during the entire retention test (Fig. 15C). The remaining two Ss earned negative savings scores.

One additional S, whose data are not figured in Table 1, suffered a more laterally placed lesion which did not invade the nucleus reticularis pontis oralis. This S showed perfect memory of the maze habit.

Other Brainstem Areas

Colliculi. Four Ss sustained bilateral lesions to the dorsal midbrain. Three suffered damage mainly to the superior colliculus and one received damage to the inferior colliculus. Two of these Ss, in addition to having damage to the anterior one-fourth of the superior colliculus, suffered severe destruction of the pretectal area—one of these earned a savings score of 40% and the other, 100%. The S with the largest lesion of the superior colliculus (Fig. 16A) showed perfect memory of the maze habit, and the S with damage to the inferior colliculus (Fig. 16B) earned a savings score of 87%.

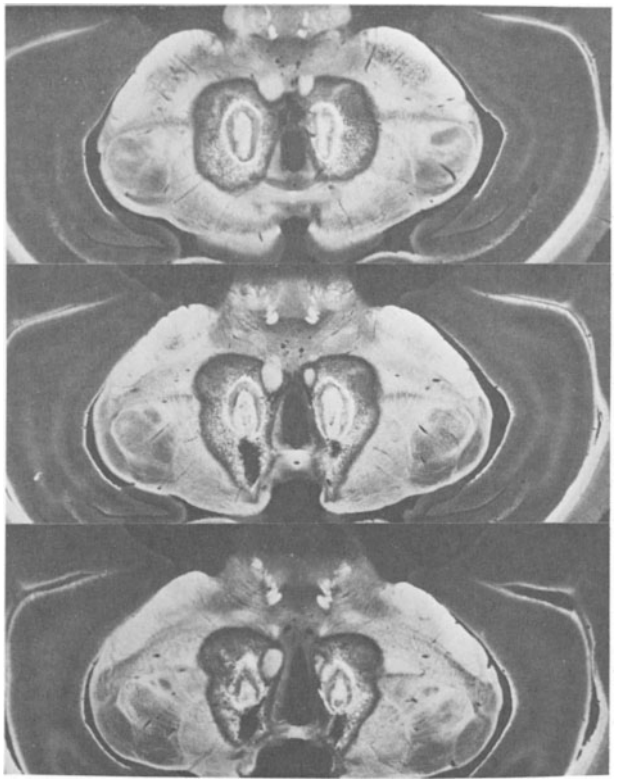

Fig. 14. Three unstained sections showing a lesion of the reticular formation at the dimesencephalic juncture in a rat that failed to relearn the maze.

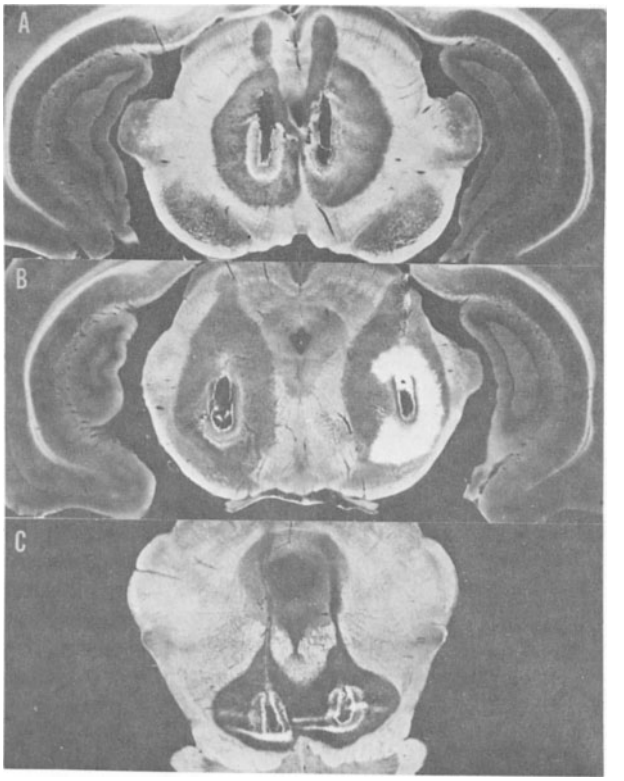

Fig. 15. Unstained sections derived from three rats showing lesions of the dorsal (A), lateral (B), and caudal (C) mesencephalic reticular formation. The rat with the dorsal lesion earned a savings score of 77%, while the remaining two earned negative savings scores.

Central Gray. Two Ss suffered lesions of the central gray substance and surrounding reticular formation. One showed a slight retention loss, while the other earned a perfect savings score (Fig. 16C).

Red Nucleus. Three Ss sustained lesions to the area of the red nucleus. In all cases, the lesions were somewhat asymmetrical (Fig. 17). One S failed to relearn the maze, while the second and third earned savings scores of 0% and 78%.

Substantia Nigra. The substantia nigra was partially destroyed in four Ss (Fig. 18A). In all cases, the lesions damaged both the lateral limb of the medial lemniscus and the immediately overlying reticular formation. Three showed moderate losses in retention (savings scores between 19% and 40%) and the fourth (having the smallest lesion) earned a savings score of 74%.

Central Tegmentum. Two Ss sustained midline mesencephalic lesions. In one, the lesion destroyed the tegmentum dorsal to the interpeduncular nucleus (Fig. 18B). The lesion in the second was located at levels caudal to the interpeduncular nucleus (Fig. 18C). Both Ss earned negative savings scores.

Cerebellum

In two Ss, the cerebellum was extensively damaged. A segment of the paraflocculus remained intact in both Ss

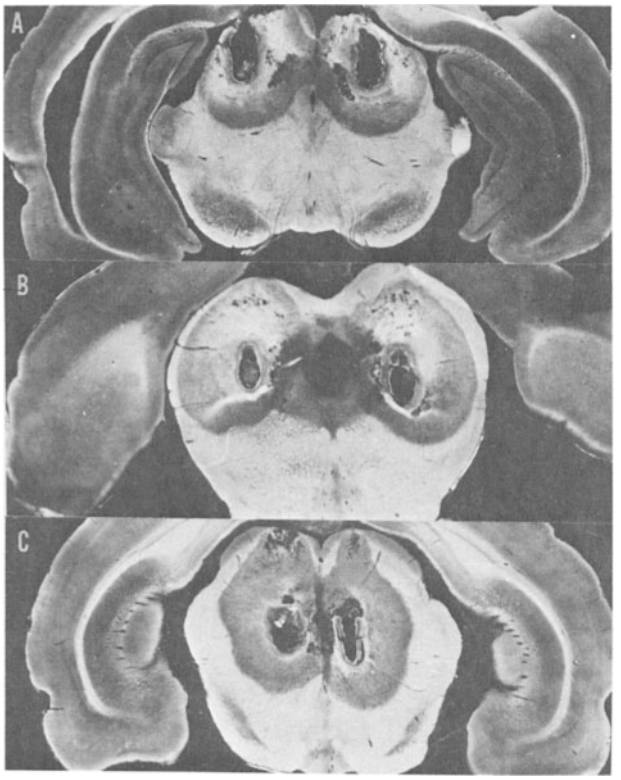

Fig. 16. Unstained sections derived from three rats showing lesions of the superior colliculus (A), inferior colliculus (B), and central gray (C). All three rats earned savings scores in excess of 86%.

and the anterior one-fifth of the cerebellum escaped injury in one S. Each was able to relearn the maze habit despite the presence of the classical cerebellar syndrome (locomotor ataxia). The S with the larger lesion earned a savings score of −145%, while the second S earned a savings score of −23%.

Summary

Of the 73 brain-damaged rats involved in this study, 43 showed complete loss of the maze habit (no positive savings). Their lesions were located within the anterior neocortex, posterior neocortex, cingulate cortex, septofornix area, hippocampus, corpus striatum, lateral hypothalamus, mamillary bodies, thalamus (anterior, ventromedial, lateral, or posterior regions), subthalamus, brainstem reticular formation (rostral, lateral, or posterior regions), red nucleus area, central tegmentum, or cerebellum. Nine rats showed a moderate loss of the maze habit (1%-50% savings). Their lesions damaged the medial hypothalamus, lateral hypothalamus, lateral thalamus, brainstem reticular formation (dorsal or lateral regions), superior colliculus, or substantia nigra. The remaining 21 rats showed either a slight retention loss or normal retention of the maze habit (51% savings or better). Injured structures included the amygdala, rostral medial forebrain bundle, medial hypothalamus, dorsomedial thalamic nucleus, dorsal (or lateral) reticular formation, superior colliculus, inferior colliculus, central gray substance, red nucleus area, and substantia nigra.

Error Distribution and Lesion Locus

Inspection of the individual records of those 27 Ss failing to relearn the maze habit revealed that 18 committed most of their errors postoperatively at Blind Alley B, 5 at Blind Alley A, and the remaining 4 at Blind Alley C. These data are plotted in Fig. 19 according to the locus of the lesion. Each capital letter denotes an individual rat, the approximate locus of the rat's lesion, and the blind (A, B, or C) that led to the highest frequency of errors. Since this plot suggested the existence of a differential effect between lesion locus and error frequency at a particular blind, it was decided to include in Fig. 19 the data of those brain-damaged Ss (lowercase letters) that relearned the maze, but with a savings score of 50% or less.

It will be noted, first of all, that Blind Alley B was the most difficult for those Ss having damage to the neocortex or cingulate cortex. In the region of the posterior hypothalamus, the majority of Ss (six out of seven cases) also made most of their errors at Blind Alley B. This was also the case for the two Ss with ventromedial thalamic lesions. On the other hand, all

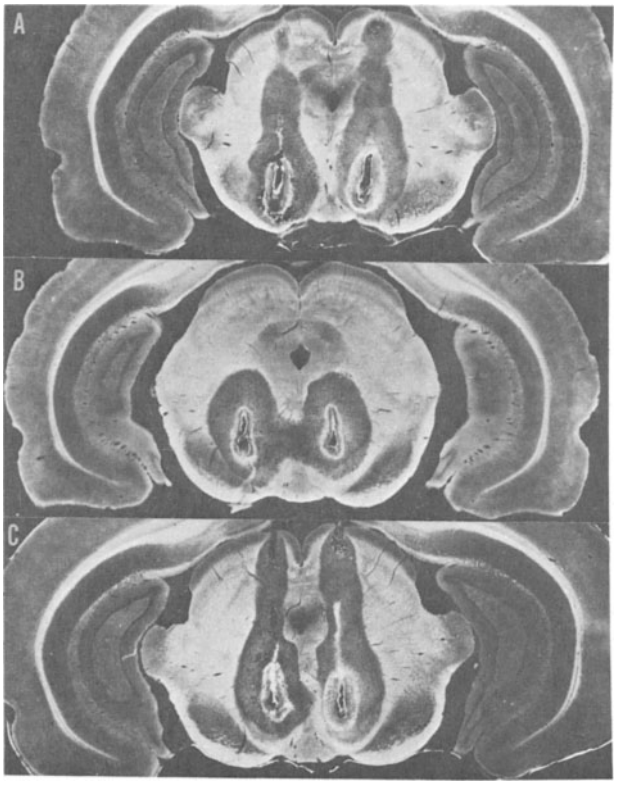

Fig. 17. Unstained sections derived from three rats showing lesions in the region of the red nucleus. One rat (A) failed to relearn the maze, while the remaining two earned savings scores of 0% (B) and 78% (C).

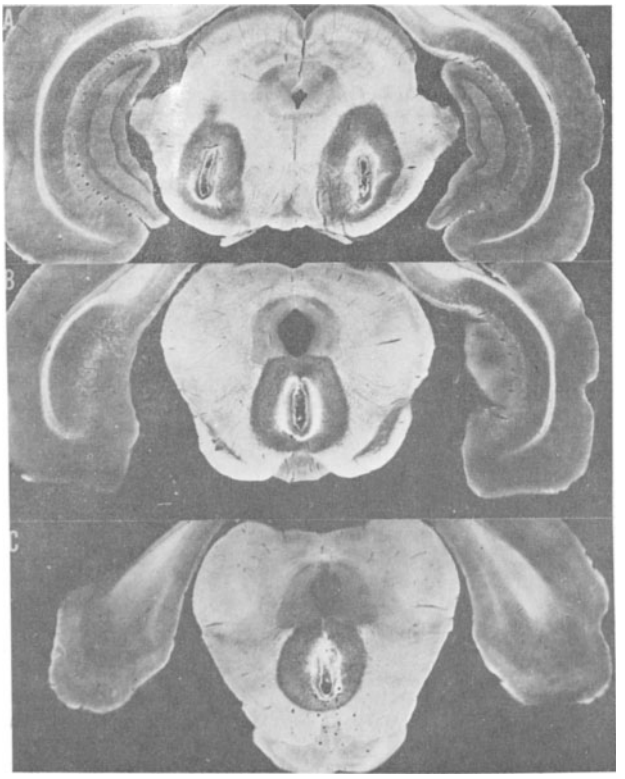

Fig. 18. Unstained sections derived from three rats showing lesions of the substantia nigra (A) and central tegmentum (B and C). The rat with the nigral lesion earned a savings score of 36%, while the remaining two earned negative savings scores.

seven Ss having lesions within (or encroaching upon) the juncture between the thalamus and the midbrain committed most of their errors at Blind Alley C. In the region occupied by the substantia nigra, red nucleus, and central tegmentum, five of the seven Ss committed most of their errors at Blind Alley A. The Ss with cerebellar or anterior thalamic lesions also made most of their errors at Blind Alley A. Mixed results were found in those Ss having lesions of the hippocampus, septal area, corpus striatum, lateral thalamus, subthalamus, or caudal mesencephalon.

DISCUSSION

General Findings

One of the most remarkable findings of this study concerns the unusually high degree of susceptibility of the maze habit to interference by lesions of various parts of the brain. A brightness discrimination habit (e.g., approach a white card and avoid an adjacent black card), for example, has been found to be subject to interference only by lesions of the posterior neocortex, corpus striatum, posterior diencephalon, ventral mesencephalon, or pontine reticular formation (Thompson, 1969; Thompson & Thorne, 1973). The maze habit used in the current study, in contrast, was abolished not only by lesions of the foregoing areas, but by injuries to the anterior neocortex, cingulate cortex, hippocampus, septofornix area, most regions of the thalamus and hypothalamus, and the cerebellum as well. Thus, the maze habit resembles the conditioned avoidance habit (see Rich & Thompson, 1965) to the extent that both show a low degree of localization of function at cortical as well as subcortical levels.

At the same time, however, it must be emphasized that the maze habit was not dependent upon the integrity of *all* regions of the brain. For example, those Ss suffering damage to the amygdala, rostral extension of the medial forebrain bundle, medial dorsal thalamic nucleus, dorsal midbrain, or lateral pontine area failed to exhibit serious deficits in retention. The fact that many of the lesions suffered by the foregoing Ss were as large as those suffered by several Ss failing to relearn the maze habit (e.g., rostral medial forebrain bundle vs mamillary body lesions, amygdaloid vs hippocampal lesions, tectal vs rostral reticular formation lesions) strongly militates against the notion that "lesion size" rather than "lesion locus" was the critical variable influencing the

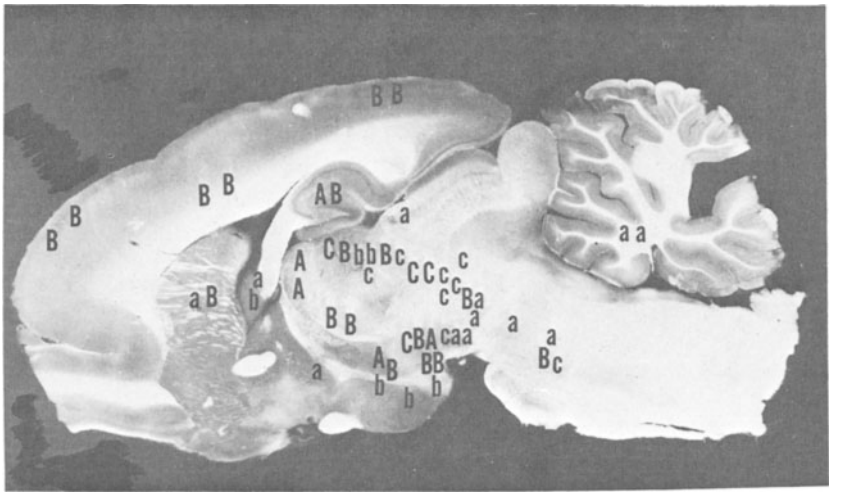

Fig. 19. Parasagittal section of the rat brain. Each letter denotes an individual rat, the approximate locus of the rat's lesion, and the blind that led to the highest frequency of errors. Capital letters refer to animals that failed to relearn the maze, while lowercase letters refer to animals that relearned, but with a savings score of 50% or less.

magnitude of the retention deficit.

It is also important to note that the Ss with neocortical lesions did not demonstrate the greatest deterioration in the performance of the maze habit. If total number of postoperative errors are considered, the four Ss with neocortical damage rank 8th, 16th, 17th, and 22nd. The brain-damaged Ss that did show the greatest deterioration (averaging at least 2.0 errors per trial throughout the retention test) had lesions within the cingulate cortex, lateral hypothalamus, ventromedial thalamus, or posterior thalamus. These results stand in marked contrast to those reported in earlier studies (Brown & Ghiselli, 1938; Ghiselli & Brown, 1938; Lashley, 1950), which indicated that neocortical lesioned Ss were inferior to subcortical lesioned Ss in the performance of complex mazes. This discrepancy is most likely due to differences in lesion locus and/or lesion size.

At this juncture, it might be argued that the results of the current study lack generality to other maze situations because of differences in the motive employed, punishment of errors, and design of the maze. Such an argument cannot readily be sustained in the light of several remarkable similarities between the results of previous studies (using more conventional methods) and those of the present experiment. These similarities include (a) the complete abolishment and retarded relearning of a maze habit following destruction of at least 20% of the total neocortical surface (Lashley, 1929), (b) the abolishment but subsequent relearning of a maze habit by *totally* cerebellectomized rats (Lashley & McCarthy, 1926), (c) the retention deficits of a maze habit following either septofornix or hippocampal lesions, with the latter showing greater retardation (Thomas, 1971), (d) the retention losses of a maze habit in the presence of either cingulate or mamillary body lesions (Kaada et al, 1961), (e) the absence of serious retention deficits following either peripheral blinding or olfactory bulbectomy (Casper, 1933), and (f) with respect to preoperative learning, the early elimination of culs subject to errors based upon centrifugal swing followed by the elimination of culs subject to errors based upon goalbox orientation and choicepoint expectancy (Munn, 1950).

Neocortical, Cingulate, and Cerebellar Lesions

The finding that bilateral ablations of either the anterior or posterior sectors of the cerebral cortex produced impairments in retention of the maze habit is in agreement with the results of previous studies utilizing either an enclosed maze (Lashley, 1929) or an open maze with peripherally blind rats (Thompson, 1959). Although the anterior and posterior neocortically damaged Ss showed equivalent losses in retention (none was able to relearn the habit within 10 days), they did differ conspicuously with respect to their conduct within the maze. The two Ss of the anterior group displayed exaggerated emotional excitement in response to footshock. At one time or another, they would freeze, jump upwards vigorously, bite the grid floor, or run wildly until they came to the end of a blind alley. In contrast, the two Ss of the posterior group showed relatively normal escape responses to footshock. This difference in responsivity to footshock following neocortical extirpations has been observed earlier (Thompson, 1969) and is consistent with the notion that the anterior regions of the rat's cerebrum are significant for pain-escape behavior (Runnels & Thompson, 1969). The extent to which the disturbance in maze performance exhibited by the anterior group is due to a defective escape response is not entirely clear. It is important to note, however, that similarly placed lesions do not interfere with retention of visual discrimination habits motivated by escape from footshock (Horel, Bettinger, Royce, & Meyer, 1966; Thompson, 1960a, 1969).

Because of the paucity of animals and the intragroup uniformity of the neocortical lesions, the data of the present experiment are inadequate to evaluate the mass action effect and cannot readily be applied to the question concerning the degree to which mass action represents either a reduction in "general facilitation" or an encroachment on two (or more) "critical" functional areas (see Gross, Chorover, & Cohen, 1965). Two observations, however, are worthy of mention. First of all, the finding that the Ss of the posterior group failed to relearn the maze while those of the enucleated group rapidly relearned the maze provides further support for Lashley's (1943) contention that the occipital cortex has a function in maze performance above and beyond that of reception and integration of optic impulses. The second observation to be made concerns Lashley's suggestion that occipital lesions produce retention losses of a maze habit due to a reduction in facilitation of remaining cortical areas. The main fault with this suggestion has to do with the fact that this proposed nonvisual function of the occipital cortex *has not been consistently demonstrated in the expression of other learned responses.* For example, extensive posterior cortical lesions in rats have little effect on retention of latch box problems (Spiliotis & Thompson, 1973), a kinesthetic discrimination problem (Lashley, 1929; Thompson et al, 1961), or a conditioned response signaled by onset of light (Breen & Thompson, 1966; Thompson, 1960b). Even Lashley struggled with this problem when he found that extensive cerebral lesions did not interfere with acquisition of either a light-dark discrimination (Lashley, 1929) or the double-latch box habit (Lashley, 1935). According to the recent work of Lubar, Schostal, and Perachio (1967) on cats, a two-way avoidance conditioning situation has the property of revealing a nonvisual function of the occipital cortex. Taken as a whole, however, these data show that the nonvisual function of the occipital cortex can only be disclosed in a limited number of learning situations and, therefore, fail to support Lashley's notion that the

occipital cortex exerts some nonspecific facilitative effect on other cortical (or certain subcortical) areas. The alternative notion (which will be discussed later) is that the nonvisual function of the occipital cortex is specific in nature, possibly being concerned with discriminative responses to spatial cues.

The two Ss having cingulate ablations showed dramatic losses in retention. In fact, both of these Ss made more errors on the retention test than did the poorest neocortically damaged rat. Kaada et al (1961) have previously reported losses in maze performance in rats suffering from cingulate lesions. Thomas and Slotnick (1962), however, did not observe deficits following section of the cingulum bundle. Undoubtedly, further work will be needed in order to establish whether the losses in maze performance resulting from cingulate damage are due exclusively to destruction of cells and/or fiber systems intrinsic to this region of the brain.

It was fascinating to observe the two cerebellectomized rats relearn the maze habit in the presence of locomotor ataxia. That the loss in retention was not entirely due to this locomotor handicap is suggested by three lines of evidence. First of all, less than 10% of the errors made by these cerebellectomized Ss appeared to result from "accidental" entries into culs by virtue of their staggering forward progression. (In most instances in which an error was committed, S directly and unhesitatingly entered a cul). In the second place, the presence of a locomotor deficit might be expected to lead to more errors at culs located in the vicinity of a 180-deg turn (Blind Alley B) than at culs located in the vicinity of a 90-deg turn (Blind Alleys A and C). Yet, the cerebellectomized Ss made most of their errors at Blind Alley A (mean errors: Blind Alley A = 12.0, Blind Alley B = 5.0, Blind Alley C = 4.5). Finally, destruction of the cerebellum has not been found to impair retention of previously learned visual discrimination habits requiring a relatively long approach to discriminanda positioned side by side (Thompson, 1969).

In the classic study by Lashley and McCarthy (1926), rats sustaining ablations of no more than approximately 50% of the total cerebellar mass showed excellent retention of an eight-cul maze. The one rat with complete destruction of the cerebellum (due, in part, to the presence of a cyst), however, exhibited a marked loss of the maze habit. While these investigators attributed the deficit in this one exceptional animal to compression of the medulla by the infection, the results of the current study suggest the possibility that total cerebellar removal is a sufficient condition to induce a loss in maze performance. These findings clearly constitute grounds for reexamining those notions concerning the kinesthetic basis of the maze habit (see Munn, 1950).

Subcortical Lesions

It was not surprising to discover that lesions of either the corpus striatum, subthalamus, posterolateral hypothalamus, posterior thalamus, central tegmentum, red nucleus, substantia nigra, or the ventral portions of the brainstem reticular formation impaired retention of a maze habit. Such lesions have previously been found to interfere with the execution of a wide variety of learned responses, including visual discrimination habits, a kinesthetic discrimination habit, latch box problems, and a conditioned avoidance response (see Thompson & Thorne, 1973). As pointed out earlier (Thompson & Thorne, 1973), these neural structures eminently qualify as the main components of the "general memory system" (GMS) of the rodent's brain to the extent that damage to any one of these structures interferes with the expression of virtually every learned task studied to date in the white rat.[3] It is interesting to note that pathology of these areas in humans is also associated with generalized intellectual deficits (see Luria, 1970).

The role of the GMS in the performance of maze habits (and other learned responses as well) must be viewed from the broader perspective that this system overlaps to varying degrees the reticular activating system (Moruzzi & Magoun, 1949; Shute & Lewis, 1967), the neural assemblies underlying hunger, thirst, and pain-avoidance behaviors (Runnels & Thompson, 1969; Ungerstedt, 1971), the intracranial self-stimulation pathways (Olds & Olds, 1963; Routtenberg & Malsbury, 1969), the extrapyramidal system (Jung & Hassler, 1960), and the recently identified system of "learning units" (Olds, Disterhoft, Segal, Kornblith, & Hirsh, 1972). On the basis of these findings, the disorganizing effects of GMS injuries may be interpreted as reflecting attentional, motivational, emotional, motorial, and/or associative dysfunctions. On the other hand, if the diverse systems mentioned above are found to share common neuronal elements rather than to interdigitate at common sites within the brain, then the functional significance of the GMS may be more appropriately expressed in terms of "integrating" constructs, such as "centrencephalic" (Penfield, 1954) or "command" (Kilmer, McCulloch, & Blum, 1968) functions, "central facilitory set" (Sperry, 1955), "suppression of error-producing tendencies" (Harlow, 1959), and the like.

The finding that destruction of either the hippocampus, septofornix area, mamillary bodies, anterior thalamus, or cingulate cortex abolished the maze habit is intriguing. These structures, of course, constitute the main components of a neural system proposed by Papez (1937) to be essential for emotional experience. It is difficult, however, to relate these findings on the maze habit to the theory of Papez. The pain-avoidance motive used in the current study might be considered as the basis for the emergence of the Papez circuit due to the presence of a strong affective element in any situation involving the application of noxious stimuli (see Melzack & Casey, 1968). This explanation is invalidated, however, by the finding that the retention of simultaneous visual discrimination

habits learned under the pain-avoidance motive is largely unaffected by lesions of the Papez circuit (Thompson, 1969). Furthermore, lesions of either the septofornix area, hippocampus, mamillary bodies, or cingulate cortex have been reported to impair maze performance based upon an appetitive motive (Kaada et al, 1961; Thomas, 1971).

Comparison of Five Specific Memory Systems

Before dealing with the problem concerning the functional significance of the various components of the maze memory system, it is necessary to review briefly the composition of other specific memory systems that have previously been mapped by the lesion method.

The visual memory system has been found to be composed of the GMS and the retino-geniculo-striate complex (Lashley, 1929; Thompson, 1969; Thompson & Pucheu, 1973; Thompson & Thorne, 1973; see Note 3).

The kinesthetic memory system includes the GMS and the anterior (sensorimotor) neocortex (Lashley, 1929; Thompson et al, 1961, 1967; Thompson & Thorne, 1973). In a study recently completed at the Louisiana State University laboratory, additional components of the kinesthetic memory system have been identified. They include the cingulate cortex, anterior thalamus, lateral thalamus, ventromedial thalamus, and the cerebellum.

The manipulative response memory system contains components roughly corresponding to those making up the kinesthetic memory system (Spiliotis & Thompson, 1973).

The conditioned avoidance response memory system also contains virtually all of the components of the kinesthetic memory system (Mitcham & Thomas, 1972; Thomas & Slotnick, 1962; Thompson, 1964; Thompson et al, 1964) plus the hippocampus, septofornix area, amygdala, and medial thalamus (Rich & Thompson, 1965; Thompson[4]; Vanderwolf, 1962).

As shown in the current study, the maze memory system includes all of the components of the kinesthetic memory system plus the posterior neocortex, hippocampus, septofornix area, and mamillary bodies.

In the light of these findings, several unique hypotheses can be formulated. First, the expression of any learned response depends upon the integrity of a "nonspecific" group of structures (the GMS) and a "specific" group of structures, the latter being a function of the nature of the learned response.[5]

Second, if the learned response is totally dependent upon and guided by visual cues, such as a simultaneous visual discrimination task, then the specific group of structures essential for the expression of that learned response primarily involves the retino-geniculo-striate complex.

Third, if visual cues are not necessary for the expression of a previously learned response, such as a kinesthetic discrimination task, latch box problems, conditioned avoidance response to nonvisual cues, or a complex maze, then the specific group of structures necessary for the performance of that learned response involves the sensorimotor cortex, cingulate cortex, cerebellum, anterior thalamus, ventromedial thalamus, and possibly the lateral thalamus.

Fourth, the amygdaloid complex and the medial thalamus emerge in importance in the expression of a previously learned response only when that response is initiated and maintained by the fear drive, such as a conditioned avoidance response.

Additional hypotheses are developed in the succeeding section.

The Maze Memory System: An Interpretation

It has repeatedly been shown that the performance of a maze habit by rats does not seriously deteriorate following peripheral interruption of either visual (Casper, 1933; Lashley, 1929, 1943; Thompson, 1959) or kinesthetic (Asdourian & Preston, 1972; Ingebritsen, 1932; Lashley & Ball, 1929) pathways. As a consequence, some authors have viewed the negotiation of a maze to be dependent upon the development of a "cognitive map" (Tolman, 1948), a "directional set" (Tryon, 1939), or some other central (symbolic) process (Lashley & Ball, 1929). Whatever mechanism is involved in constructing and monitoring sequential responses in the absence of peripheral feedback (see Taub & Berman, 1968), it is very likely to be dependent upon the activities of those *central* structures having kinesthetic functions. This notion is clearly supported by the findings that those brain regions which are necessary for the expression of a kinesthetic discrimination habit are also necessary for the expression of skilled movements (Spiliotis & Thompson, 1973). On the strength of this argument, it would be expected that those central nervous areas underlying the performance of a kinesthetic discrimination habit would also participate in the performance of a maze habit. The results of the current study bear out this expectation. However, the performance of a maze habit appears to require the integrity of an additional assembly of structures (the posterior neocortex, hippocampus, septofornix area, and mamillary bodies). Thus, the expression of a maze habit is dependent upon the intactness of three "functional blocks" of the brain (see Fig. 20): the first block constitutes the GMS (areas occupied by black dots), the second block corresponds to the specific areas composing the kinesthetic memory system (areas occupied by vertical lines), and the third block consists of the posterior neocortex, hippocampus, septofornix area, and mamillary bodies (blackened areas).

The foregoing analysis of the maze memory system is complete, except for the functional significance of the third block. While it is quite possible that each of the structures composing the third block may play a separate role in the performance of a maze habit, the position taken here is that these structures form a neural assembly which carries out a specific function. This

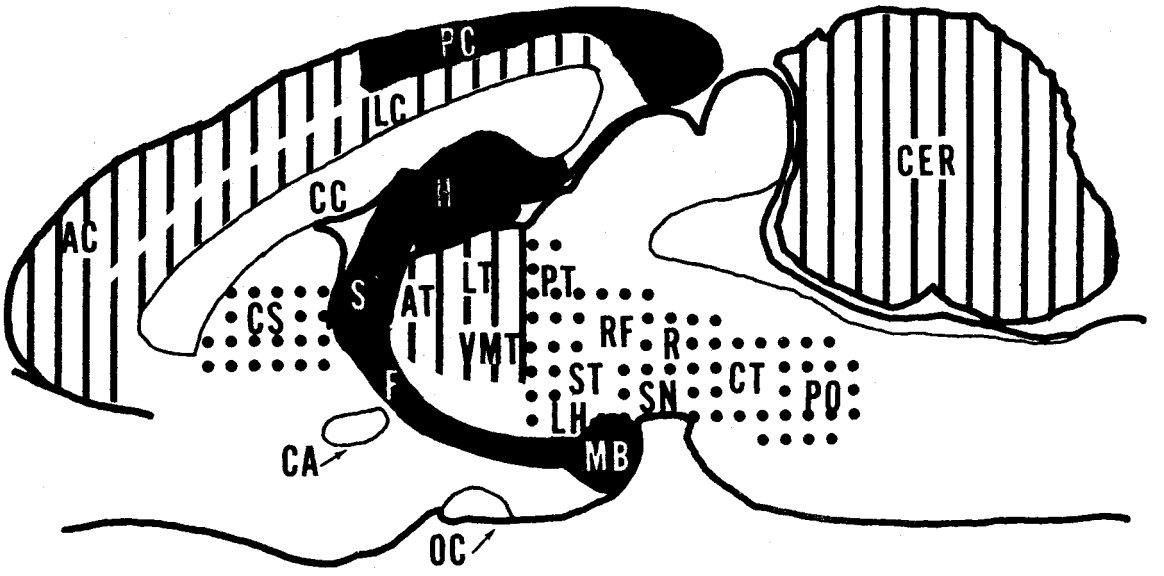

Fig. 20. Schematic drawing of a parasagittal section of the rat brain showing the three blocks of the maze memory system: the first block (areas occupied by black dots) consists of those structures having integrative functions, the second block (areas occupied by vertical lines) consists of those structures having kinesthetic functions, and the third block (blackened areas) consists of those structures having functions related to the discrimination of spatial cues. Abbreviations: AC = anterior neocortex; AT = anterior thalamus; CA = anterior commissure; CC = corpus callosum; CER = cerebellum; CS = corpus striatum; CT = central tegmentum; F = fornix column; H = hippocampus; LC = limbic cortex; LH = lateral hypothalamus; LT = lateral thalamus; MB = mamillary bodies; OC = optic chiasma; PC = posterior neocortex; PO = pontine reticular formation; PT = posterior thalamus; R = red nucleus; RF = midbrain reticular formation; S = septal area; SN = substantia nigra; ST = subthalamic area; VMT = ventromedial thalamus.

function is hypothesized to be "sensory" in nature and related to the discrimination of spatial cues (e.g., left vs right). More specifically, this assembly is conceived to deal with the process by which the output of central kinesthetic structures is integrated to form the basis of left-right discriminations. Perhaps the most persuasive evidence favoring this notion comes from lesion studies on the "successive" visual discrimination habit (e.g., go left in the presence of two white cards, go right in the presence of two black cards). It will be noted that this task involves not only a visual discrimination, but a left-right discrimination as well. It would be expected, therefore, that lesions of the posterior neocortex, hippocampus, septofornix area, or mamillary bodies would have greater disturbing effects on the performance of a successive visual discrimination than on the performance of a simultaneous visual discrimination (approach the white card and avoid the adjacent black card). This prediction has already been confirmed in studies dealing with the posterior neocortex (Thompson & Malin, 1961) and the hippocampus (Kimble, 1963). Furthermore, it would be expected that lesions of the specific areas composing the kinesthetic memory system would also have a greater deleterious effect on a successive discrimination than on a simultaneous discrimination. This prediction has been confirmed in studies dealing with the anterior neocortex (Thompson & Malin, 1961) and the anterior thalamus (Thompson, Baumeister, & Rich, 1962).

Other data supporting the notion that the third block of the maze memory system functions in discrimination of spatial cues come from studies on the repeated reversal of a position habit. In one series of experiments (Thompson, 1964; Thompson & Langer, 1963), performance on this task was significantly impaired following damage to any one of the components of the third block, except the posterior neocortex. It should be pointed out, however, that a single-unit T-maze was used, thus maximizing the difference between the spatial cues. When the right-left difference was reduced through the use of a second apparatus, posterior neocortical lesions were found to produce dramatic disturbances in the performance of the repeated reversal of a position habit (Thorne & Thompson, 1970).

Studies on the relative difficulty of an object (visual) reversal task vs a position reversal task in brain-damaged animals are also relevant here. In a series of elegant experiments on rats, Samuels (1972) has convincingly shown that hippocampal lesions selectively impair performance on the position reversal task. Similar findings have been obtained in monkeys subjected to either hippocampal (Jones & Mishkin, 1972; Mahut, 1971) or fornix column (Mahut, 1972) damage.

It should be apparent that the preceding discussion involving the limbic system in maze performance is "unconventional" in two respects. First of all, sensory (kinesthetic) functions have been ascribed to most elements of the limbic system (cingulate cortex,

hippocampus, septofornix area, mamillary bodies, and anterior thalamus). In the second place, the concept of "inhibition," which is currently in widespread use in relation to the limbic system (see Douglas, 1967; Kimble, 1968), has been eschewed. This raises the question concerning the extent to which other behaviors linked to the activities of the limbic system might also be adequately interpreted in terms of sensory contributions without resort to response inhibition, drive inhibition, and the like. To illustrate the potential usefulness of this "sensory" approach, consider the fact that lesions of certain components of the limbic system are associated with impaired passive avoidance learning and greater resistance to extinction (see reviews by Douglas, 1967, and Kimble, 1968). According to Mowrer's theory (1960) of learning, passive avoidance and extinction behaviors *have a kinesthetic basis*. This poses the possibility, therefore, that the deficits in these behaviors associated with limbic injuries may reflect dysfunctions of the kinesthetic sensory systems of the brain.

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NOTES

1. If S failed to reach the criterion within 10 days, the retention test was terminated and the savings score computed as though S had reached the criterion on Days 11 and 12.

2. An "excellent" runner refers to the following characteristics: (a) no more than 2-3 footshocks are necessary to force escape responses either from the startbox or from a blind alley, (b) except for the first day of the retention test, repetitive errors are rarely made, and (c) footshocks do not provoke either wild running, leaping upwards toward the lid of the maze, or biting of the grid. In subsequent sections of this paper, an S may be considered to be an excellent runner, unless otherwise specified.

3. In the report by Thompson and Thorne (1973), the corpus striatum was not included within the general memory system because of insufficient data on visual discrimination habits. Recently acquired data from the Louisiana State University laboratory, however, clearly implicate the corpus striatum in the normal performance of a brightness discrimination habit.

4. Thompson, R. Unpublished manuscript on the amygdala, 1965.

5. The fact that lesions of the GMS lead to disturbances in the expression of certain genetically endowed behavior patterns, such as eating, drinking, pain escape, and the like, suggests the possibility that the functional significance of the GMS is not restricted to the performance of "learned" responses. In other words, the neural networks mediating "instinctive" behaviors may occupy the same regions of the brainstem as those mediating learned behaviors.

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