Brain lesions impairing visual and spatial reversal learning in rats: Components of the "general learning system" of the rodent brain

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The "general learning system" (GLS) is conceived as an ensemble of brain structures essential for normal acquisition of a wide variety of laboratory tasks. Based upon earlier lesion studies, it was reasoned that the components of the rodent's GLS could be identified by determining which lesion placements within the rat brain would lead to defective acquisition of a spatial discrimination habit and its reversal as well as a brightness discrimination habit and its reversal. Of the 11 cortical and subcortical (frontal cortex, parietal cortex, occipital cortex, anterior cingulate cortex, posterior cingulate cortex, rostral caudoputamen, dorsal hippocampus, mammillary bodies, mediodorsal thalamic nucleus, parafascicular nucleus, and substantia nigra) sites investigated in this experiment, only two qualified as components of the GLS—the parafascicular nucleus and the substantia nigra. The possibility that the nigro-parafascicular-striatal complex constitutes a major part of the rodent's GLS is discussed.

In a series of studies designed to compare the effects of different cortical and subcortical lesions on retention of both a visual discrimination habit and a nonvisual incline plane (vestibulo-proprioceptive-kinesthetic) discrimination habit in albino rats (Thompson, 1976; Thompson, Arabie, & Sisk, 1976; Thompson & Thorne, 1973), it was possible to identify three functionally separate groups of brain structures. One group was concerned only with retention of the visual discrimination habit, the second was concerned only with retention of the incline plane discrimination habit, and the third was concerned with retention of both habits. Since the last group of brain structures—all being associated with the brainstem reticular formation, basal ganglia, or limbic midbrain area (Thompson, 1982a)—was subsequently found to play a significant role in retention of other types of learned activities, such as a complex maze (Thompson, 1974), latch-box problems (Spiliotis & Thompson, 1973; Thompson, Gates, & Gross, 1979), and avoidance responses (Thompson, 1978a), it was termed the "general memory system" of the rat brain (Thompson & Thorne, 1973).

The current study focused on what will be termed the "general learning system" of the rat brain. While the makeup of the general learning system (GLS) cannot be fully established until it has been demon-

strated that a lesion to each component part impairs acquisition of a broad spectrum of laboratory tasks, a reasonably good estimate of its composition can be obtained by ascertaining which structures of the rat brain, when damaged individually, will lead to significant impairments in acquisition of the following tasks: A position discrimination habit and its reversal and a white-black discrimination habit and its reversal. Selecting only these four problems—two dealing with visual discriminations and two dealing with spatial discriminations, the latter probably being mediated by the processing of vestibulo-proprioceptive-kinesthetic cues (Douglas, Clark, Erway, Hubbard, & Wright, 1979; Thompson, Hale, & Bernard, 1980)—to arrive at a provisional picture of the components and boundaries of the GLS was based on the findings that the composition of the general memory system (GMS) was largely exposed by investigating the effects of selective brain lesions on retention of only two problems: A visual discrimination and a vestibulo-proprioceptive-kinesthetic (incline plane) discrimination (Thompson & Thorne, 1973).

It is likely that several components of the GMS will be included within the GLS for the reason that damage to certain parts of the former leads to post-operative relearning scores on a variety of laboratory tasks which are greater than the corresponding pre-operative learning scores (see Thompson, 1978b). On the other hand, it can be assumed that the GLS will contain several components that are not included within the GMS. This assumption derives, at least

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in part, from the observations that amnesic patients with lesions of the medial temporal region or the diencephalon may show virtually normal memory of remote events preceding the onset of the amnesic syndrome, but are apt to display a profound disturbance in learning new material (Milner, 1970; Scoville & Milner, 1957; Squire & Moore, 1979; Talland, 1965; Victor, Adams, & Collins, 1971).

Several groups of brain-damaged rats have already been evaluated in connection with acquisition (and retention) of a series of individual spatial reversal problems in a single-unit T-maze adapted for escape-avoidance of footshock as a motive (Thompson, 1981, Note 1; Thompson, Kao, & Yang, 1981; Thompson & Yang, 1982). Those groups that were significantly retarded in learning the original problem as well as the first reversal suffered damage to the lateral frontal cortex, parietal cortex, occipital cortex, anterior cingulate (and medial frontal) cortex, posterior cingulate cortex, rostral caudoputamen, dorsal hippocampus, medial mammillary bodies, mediodorsal thalamus, parafascicular nucleus, or substantia nigra. It was the purpose of the present experiment to determine whether selective lesions to these structures would likewise produce significant deficits in both original learning and reversal learning of a white-black discrimination habit. Any one of the foregoing structures found to be critical for acquisition of these visual problems would, according to the argument introduced earlier, qualify as a component of the GLS.

METHOD

Subjects and Surgery

Under deep chloral hydrate anesthesia, male Sprague-Dawley albino rats (80-120 days old) were subjected to bilateral cortical ablations, bilateral subcortical lesions, or sham operations. Cortical injuries were accomplished by aspiration, while subcortical injuries were made stereotactically by passing a constant anodal current (1.8-3.0 mA) for 10 sec through an implanted stainless steel electrode with 1.0-2.0 mm of the tip exposed. The size and locus of the lesions of the various experimental groups were intended to be comparable to those suffered by the corresponding groups investigated earlier in connection with acquisition of the spatial discrimination tasks. The sham-operated group underwent anesthetization, shaving of hair over the cranium, exposure of the skull, and suturing without any additional treatment.

All animals were usually housed, two per cage, in medium-size wire cages containing a constant supply of food pellets and water. Throughout the recovery period, the animals were handled for approximately 5 min on every 3rd day. Preliminary training began 2-3 weeks after surgery. On the day prior to preliminary training, the vibrissae of each animal were shaved.¹

Apparatus

A two-choice Thompson-Bryant discrimination box, utilizing the motive of escape-avoidance of footshock, was employed. The apparatus has been described elsewhere (Thompson, 1978b). Two pairs of stimulus cards were used in the apparatus. One pair (two medium gray cards) was employed in training the animals to push aside a card in order to gain access to the goalbox. The second pair consisted of a white card and a black card.

Procedure

Preliminary Training

On the 1st day, each rat was permitted to explore the goalbox for 10 min. During this time, the windows were blocked to prevent the animal from entering the choice chamber. Each rat was then placed into a restraining cage for 10 min, after which the animal was returned to its home cage.

On the 2nd day, each rat was trained to run from the startbox, through the choice chamber, push aside a card blocking the window, and enter the goalbox in order to escape from or avoid footshock.

Original Learning

On the 3rd day, training on the brightness discrimination (original learning) was begun. An approach response to the unlocked white card (positive) admitted the animal to the goalbox. On the other hand, an approach response to the adjacent locked black card (negative) was punished by mild footshock, the animal subsequently being forced to respond to the white card in order to gain entrance to the goalbox. An error was defined as an approach response to the black card which brought the animal's forefeet in contact with the charged grid section, which extended 8.0 cm in front of the negative card. The position of the positive card was switched from the right to the left window in a sequence mixed with single- and double-alternation runs. Eight trials were usually given each day with an intertrial interval of 50-75 sec. The criterion of learning consisted of either a "perfect" (Grant, 1946) or a "near-perfect" (Runnells, Thompson, & Runnells, 1968) run of correct responses having a probability of occurrence of .05 followed by at least 75% correct responses in the subsequent block of 8 trials given on the next day. (This criterion is especially useful in the study of reversal learning when two or more groups of subjects differ significantly from each other in the rate of original learning—see Thompson et al., 1981.) In a few cases, the criterion was not reached within 100 trials. The animals involved received no further training on the original problem and were not transferred to the reversal problem.

The specific training procedure was as follows: The animal was placed in the startbox and the clear Plexiglas startbox door was opened.² Failure to leave the startbox within 5 sec was followed by footshocks until the animal entered the choice chamber. No further footshocks were administered unless the animal made an error or failed to respond to one of the cards within 5 sec. The animal was allowed to remain in the goalbox for 10 sec, after which it was transferred to the restraining cage to await the next trial. The animals were usually run in squads of two.

Reversal Learning

On the day following attainment of the criterion of learning on the original problem, the animals began training on the reversal problem—black card positive vs. white card negative. The training procedure was the same as that described in original learning. If the criterion of learning was not reached within 100 trials, training was terminated at that point.

Histology

At the conclusion of postoperative training, each brain-damaged animal was killed with an overdose of chloral hydrate, its vascular system perfused with normal saline followed by 10% Formalin, and the brain removed and stored in 10% Formalin for 2-4 days. Cortical injuries were reconstructed on Lashley-type brain diagrams. Each brain was then blocked, frozen, and sectioned frontally at 90 μ . Every fourth section through the lesioned area was retained and subsequently photographed at 12 $\times$ by using the section as a negative film in an enlarger.

Measurements of Performance

Both trials to criterion and initial errors to criterion were used as measures of performance. A third measure (total errors to criterion) was also included, since it was not uncommon for an animal to commit more than one error (contact with the charged grid

section located in front of the negative card) within a given trial. Multiple errors on a given trial occurred when the animal retreated from the charged grid section and then approached the negative card a second (third, fourth, etc.) time, each approach response resulting in footshock. Total errors to criterion therefore represented the sum of approaches to the negative card which resulted in footshock.

Since several brain-damaged groups were significantly inferior to the controls in original learning of the brightness problem, a reversal learning deficit cannot necessarily be inferred from the finding that a given brain-damaged group was significantly inferior to the controls in learning the reversal problem. On the other hand, a reversal learning deficit would be indicated by the finding that a given brain-damaged group committed proportionately more errors (or required proportionately more trials) in learning the reversal problem, as opposed to the original problem, than the controls. To accomplish this, individual "savings scores"—[(original learning score—reversal learning score)/original learning score] $\times$ 100—were computed for each measure of performance.

RESULTS

Original and Reversal Learning

Table 1 shows the three measures of performance on the original problem and the reversal problem for all groups. These results will be discussed in terms of the site of brain damage.

Control Group

All sham-operated control subjects learned the original problem within 51 trials. On the reversal problem, the poorest control animal learned in 52 trials. Based on the Wilcoxon test for paired replicates, the reversal problem was significantly more difficult to master than the original problem in terms of initial errors ($p < .01$) and total errors ($p < .01$) to criterion, but not in terms of trials to criterion ($p > .10$).

Cortical Groups

Frontal region. Five rats sustained varying amounts of damage to the frontal (motor) cortex with minimal

Figure 1. Diagrams showing the largest (areas surrounded by dotted lines) and smallest (areas surrounded by solid lines) lesions to the frontal (top), parietal (middle), and occipital (bottom) regions.

involvement of either the corpus striatum or anterior limbic area. Figure 1 (top diagram) shows the largest and smallest lesions received by this group. The extent of damage to the entire neocortical surface averaged 15.2%, with a range of 10%-21%. These animals learned the original problem approximately as fast as the controls, but were significantly inferior to the control group on the reversal problem with respect to both trials and initial errors to criterion.

Table 1
Mean Learning Scores on the Original and Reversal Problems for All Groups

Group	Original Problem				Reversal Problem			
	N	Trials	Initial Errors	Total Errors	N	Trials	Initial Errors	Total Errors
Control	11	29.6	14.2	21.5	11	35.3	21.8	34.5
Frontal Cortex	5	33.2	16.4	26.0	5	49.2*	28.6*	43.0
Parietal Cortex	7	46.1*	20.9*	32.3*	7	42.4	26.0	40.6
Occipital Cortex	6	81.8*	37.3*	49.7*	2	100.0+*	60.5+*	87.5+*
Anterior Cingulate	5	29.0	13.0	19.2	5	37.6	23.8	39.8
Posterior Cingulate	4	28.8	13.8	18.3	4	48.0*	29.3*	52.8*
Rostral Caudoputamen	4	30.0	15.5	19.5	4	27.5	18.5	32.8
Dorsal Hippocampus	6	31.3	14.5	22.3	6	43.5	23.3	40.0
Mammillary Bodies	8	19.1	9.9*	14.5*	8	27.1	18.3	38.1
Mediodorsal Thalamus	6	36.0	15.2	23.5	6	57.3	30.0	57.3*
Parafascicular Nucleus	7	43.7*	18.7	34.9*	7	62.7*	34.4*	62.1*
Substantia Nigra	5	72.4*	33.8*	47.8*	3	100.0+*	54.0+*	73.7+*

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

Parietal region. Seven rats with parietal ablations (Figure 1, middle diagram) suffered an average of 19.4% damage to the total neocortical surface, with a range of 15%-26%. This group was significantly retarded in mastering the original problem, but earned performance scores on the reversal problem that did not differ significantly from those earned by the control group.

Occipital region. The occipital cortex was damaged in six animals (Figure 1, bottom diagram), the extent of destruction to the neocortex ranging from 21% to 26% (mean = 24.2%). The underlying hippocampus and adjacent cingulate cortex were largely spared. Four of these subjects failed to reach the criterion of learning on the original problem within 100 trials. The two that did succeed in learning the original problem could not attain the criterion of learning on the reversal problem within 100 trials.

Anterior cingulate region. In five animals, the anterior division of the cingulate cortex (including major portions of the medial frontal cortex) was ablated. In two cases, the underlying septal area was also injured. Damage to the total neocortical surface averaged 8.0% (range = 6%-10%). Figure 2 (left column)

Figure 2. Photographs of unstained sections derived from two animals showing lesions to the anterior cingulate (left column) and posterior cingulate (right column) regions.

Figure 3. Photographs of unstained sections derived from three animals showing lesions to the rostral caudoputamen (A), dorsal hippocampus (B), and mammillary bodies (C).

shows a typical ablation in one of these animals. As a group, these subjects were virtually indistinguishable from the controls in acquisition of both the original and reversal problems.

Posterior cingulate region. The posterior division of the cingulate cortex (areas retrosplenialis granularis and agranularis) was destroyed in four animals. The average extent of neocortical damage was 7.8% (range = 7%-8%), and there was slight invasion of both the hippocampus and area striata in all subjects (Figure 2, right column). These animals readily learned the original problem, but showed a significant deficit in acquiring the reversal problem.

Subcortical Groups

Caudoputamen. Four rats received relatively discrete lesions of the rostral caudoputamen (Figure 3A). These animals performed as well as the controls on the two visual problems.

Dorsal hippocampus. The dorsal hippocampus was extensively damaged in six rats (Figure 3B). This group earned performance scores on the two visual problems which were not remarkably different from those earned by the control group.

Mammillary body region. The medial mammillary nuclei along with the supramammillary area were damaged to varying degrees in eight rats (Figure 3C). In terms of both initial errors and total errors to criterion, this group was significantly *superior* to the controls in mastering the original problem. Although this group tended to acquire the reversal problem faster than the controls, the differences in learning scores fell short of statistical significance.

Mediodorsal thalamic region. Six animals sustained lesions to the mediodorsal thalamic nucleus. Other structures that were invaded included the habenular nuclei, ventromedial thalamic complex, intralaminar nuclei, anterior thalamic nuclei, and parafascicular nucleus (Figures 4 and 5). As a group, these animals were not significantly different from the controls on the original problem, but they showed a significant impairment on the reversal problem in terms of total errors to criterion.

It is noteworthy to mention that the animal with the most caudally and ventrally placed lesion (Figure 5) required 89 trials to learn the original problem and subsequently failed to learn the reversal problem within 100 trials.

Parafascicular region. Seven subjects suffered ex-

Figure 4. Photographs of three unstained sections showing an "anterior" mediodorsal thalamic lesion in one animal.

Figure 5. Photographs of three unstained sections showing a "posterior" mediodorsal thalamic lesion in one animal.

tensive damage to the rostral and intermediate portions of the parafascicular nucleus (Figure 6). Other injured structures included the mediodorsal thalamic nucleus, habenular complex, pretectal area, and habenulopeduncular tract. This group was significantly retarded in learning both the original problem and the reversal problem. One animal failed to acquire the reversal problem within 100 trials.

Substantia nigra region. Two of the five animals in this group failed to learn the original problem within 100 trials. The lesions received by these two rats damaged the anterolateral portions of the nigra and the underlying cerebral peduncle. In addition, the lesions extended rostrally into the zona incerta (Figure 7). The remaining three animals were able to learn the original problem within 57 trials, but subsequently failed to learn the reversal problem within 100 trials. Their lesions were more discrete, damaging mainly the nigra and small portions of the underlying cerebral peduncle (Figure 8).

Reversal Deficit

As pointed out earlier, a given brain-damaged group showing a significant impairment in learning

Figure 6. Photographs of three unstained sections showing a parafascicular lesion in one animal.

the reversal problem may not necessarily have exhibited a "reversal deficit," since the original learning scores of this group may have been appreciably greater than those earned by the controls. To control for intergroup differences in the rate of original learning, individual savings scores were computed for each measure. Table 2 presents the means of these savings scores for all groups. It will be noted that only 3 of the 11 brain-damaged groups achieved significantly poorer savings scores than the controls on at least one measure of performance. These groups had lesions to either the posterior cingulate cortex, mammillary bodies, or substantia nigra. A marginally significant deficit in initial error savings scores ($p = .10$) occurred in the group with mediodorsal thalamic lesions.

Escape-Avoidance Behavior

All animals with cortical, caudoputamenal, hippocampal, or mammillary body lesions usually entered the choice chamber from the startbox and responded to one of the stimulus cards either without footshock or with the application of one or two footshocks dur-

ing learning. Two animals with mediodorsal thalamic lesions, three with parafascicular lesions, and one with nigral lesions, on the other hand, frequently required more than the usual number of footshocks to force escape responses in the apparatus. It should be noted, however, that these six animals, which required multiple footshocks to force escape responses, were not the slowest learners of their respective groups.

A more commonly observed "aberrant" escape-avoidance response consisted of extremely rapid running from the startbox toward the goalbox. This appeared in 5 control animals and 15 brain-damaged (1 with frontal, 4 with parietal, 3 with occipital, 1 with anterior cingulate, 1 with caudoputamenal, 4 with hippocampal, and 1 with mammillary body lesions) animals. This rapid running behavior usually diminished in frequency when the opening of the startbox door was delayed for 10-20 sec.

General Health

All brain-damaged (and control) animals appeared quite healthy and alert at the time preliminary training was instituted. The five animals (two with nigral,

Figure 7. Photographs of three unstained sections showing a lesion damaging the substantia nigra as well as the zona incerta and cerebral peduncle in one animal.

DISCUSSION

General Findings

Brightness Discrimination Learning

On the whole, the results of this experiment confirm the findings of earlier lesion studies in demonstrating the relatively high degree of cortical (Horel, Bettinger, Royce, & Meyer, 1966; Lashley, 1929; Thompson, 1960) and subcortical (Thompson, 1969, 1976) localization of the brightness discrimination habit in the rat. Of the 11 brain-damaged groups investigated in the current study, only 4 showed a significant impairment in acquisition of the original white-black discrimination problem. These 4 groups had lesions to either the parietal, occipital, parafascicular, or nigral areas. It should be apparent that the learning losses observed in the foregoing groups cannot readily be explained in terms of the nonspecific effects of brain damage inasmuch as no significant learning deficits arose from damage to the frontal cortex, anterior (or posterior) cingulate cortex, rostral caudoputamen, dorsal hippocampus, mammillary bodies, or mediodorsal thalamus.

That moderate damage to neocortical tissue rostral to the occipital area tends to retard acquisition of a white-black discrimination in rats is consistent with the results of Horel et al. (1966). On the other hand, the finding that bilateral destruction of the occipital cortex dramatically retards acquisition of a white-black discrimination in rats is inconsistent with the reports of Horel et al. (1966) and Thompson (1960). The most probable explanation of this discrepancy may lie in the fact that the current investigation, rather than using a learning criterion of 9 correct responses within 10 trials (the one used in the Horel et al. and Thompson studies), defined the learning criterion in terms of a perfect or near-perfect run of correct responses having a probability of occurrence

Figure 8. Photographs of three unstained sections showing a lesion largely confined to the substantia nigra in one animal.

two with parafascicular, and one with frontal lesions) that showed eating and drinking disturbances beyond the 3rd postsurgical day gradually recovered these habits when provided with wet mash in the home cage.

Table 2
Mean Savings Scores for All Groups

Group	N	Trials		Initial Errors		Total Errors	
		Mean	Range	Mean	Range	Mean	Range
Control	11	-29.0	-70 to 39	-59.1	-158 to 0	-72.8	-161 to 4
Frontal Cortex	5	-52.2	-90 to 2	-75.6	-107 to -53	-66.8	-81 to -55
Parietal Cortex	7	7.1	-19 to 40	-27.6	-110 to 24	-28.1	-46 to 0
Occipital Cortex	2	-191.0	-335 to -47	-232.5	-362 to -103	-239.5	-376 to -103
Anterior Cingulate	5	-48.0	-167 to 2	-102.8	-217 to -15	-109.4	-146 to -42
Posterior Cingulate	4	-70.5*	-90 to -41	-117.8	-177 to -68	-195.3*	-293 to -120
Rostral Caudoputamen	4	1.5	-21 to 31	-23.8	-53 to 4	-65.8	-116 to -26
Dorsal Hippocampus	6	-45.2	-135 to -18	-64.3	-120 to -29	-71.3	-192 to 0
Mammillary Bodies	8	-56.3	-150 to 11	-99.5	-260 to -36	-181.1*	-355 to -53
Mediodorsal Thalamus	6	-94.2	-243 to 0	-130.3	-220 to -46	-228.3	-614 to -27
Parafascicular Nucleus	7	-53.0	-163 to 37	-100.9	-207 to -30	-98.9	-326 to 13
Substantia Nigra	3	-85.3*	-92 to -75	-100.0	-117 to -79	-86.0	-115 to -48

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

of .05. Examination of the records of the six occipitlectomized animals involved in the present study revealed that had a 9/10 learning criterion been in force, these animals would have learned the original brightness discrimination problem in an average of 50.0 trials (a mean score comparable to that reported by Horel et al., although higher than that reported by Thompson). However, since these occipitlectomized rats performed the 9/10 run beyond the 19-trial limit to be significant at the .05 level (see Runnells et al., 1968), they were arbitrarily judged not to have learned the discrimination. This overall pattern of results suggests that the extent to which occipitlectomized rats will show a learning deficit on a white-black discrimination will be directly related to the stringency of the criterion of learning. The observation that rats with occipital lesions make significantly more errors on overtraining trials on a brightness discrimination relative to controls (see Thompson, 1969) can be interpreted as supporting this suggestion.

The finding that lesions to the nigral region of the midbrain profoundly impeded learning of the white-black discrimination is intriguing. Unfortunately, the lesions in four of the five subjects extended rostrally into the zona incerta and/or invaded the subjacent cerebral peduncle, and both of these structures bordering the substantia nigra appear to be implicated in the performance of visually guided behaviors in the rat (Howze, 1974; Legg, 1979; Thompson & Bachman, 1979; Thompson, Howze, & Pucheu, 1973). It is likely, however, that a group of rats with relatively discrete nigral lesions would still have exhibited an impairment in brightness discrimination learning. This possibility is based not only upon the finding that the one animal with an uncomplicated lesion of the nigra (see Figure 8) achieved trial and error scores which fell well beyond the range established by the control group, but also upon the report that normal rats trained on one or more visual discrimination problems are apt to earn negative savings scores following the induction of relatively discrete nigral lesions (Thompson, 1976).

The presence of a visual discrimination learning deficit in rats with parafascicular damage and the absence of such a deficit in rats with mediodorsal thalamic damage would appear to be in perfect agreement with an earlier set of findings on transoperative retention of brightness and pattern discrimination habits (Thompson, 1978b). In the latter investigation, over 50% of those rats subjected to parafascicular lesions earned negative savings scores, while all rats subjected to mediodorsal thalamic lesions earned positive savings scores. However, the findings of Means and his associates (Means, Huntley, Anderson, & Harrell, 1973; Waring & Means, 1976; Weiss & Means, 1980) and Tigner (1974) that damage to the mediodorsal thalamus hinders acquisition of visual-tactile and visual discrimination tasks would

seem to be at variance with those reported in the current study. Since the lesions examined by the foregoing investigators encroached upon the parafascicular nucleus, the possibility must be considered that the observed learning disturbances arose from either damage to the parafascicular region alone or combined damage to the mediodorsal and parafascicular regions. According to Jones and Leavitt's (1974) anatomical analysis of the intralaminar nuclei of the rat, the parafascicular nucleus extends much more rostrally within the thalamus (see Figure 3 of the Jones and Leavitt report) than previously supposed. Thus, the likelihood is great that an electrolytic lesion directed at the center of the mediodorsal thalamic nucleus would infringe significantly upon the parafascicular nucleus. (This complication was avoided in the current study—except in the case shown in Figure 5—by aiming the lesion electrode at the more anterior portions of the mediodorsal thalamic nucleus.) Obviously, a lesion experiment designed to determine the precise medial thalamic focus for visual discrimination learning deficits is needed to settle this important issue.

It is not uncommon that a given brain-damaged group will be observed to learn a visual discrimination habit significantly faster than controls. Such a facilitative effect, for example, has been reported by Sara and David-Remacle (1981) in hippocampal-lesioned rats and by Jeeves (1967) in frontal cortical-lesioned rats. While the results of the present study failed to confirm the foregoing observations, the mammillary body-lesioned group was found to learn the original white-black discrimination problem faster than the controls. Since these animals showed a mean response accuracy of only 42.3% on the 1st day of training, it is doubtful that the mammillary body lesions had somehow induced a preference for the white card over the black card. Conceivably, diminished responsiveness to spatial cues (which would reduce the error-producing tendency to avoid the side from which footshock was received on the immediately preceding trial) might be responsible for this rapid learning effect. This explanation is based on the fact that mammillary body-damaged rats have difficulty in acquiring (or reacquiring) maze habits that depend heavily upon appreciation of spatial cues (Rosenstock, Field, & Greene, 1977; Thompson, 1964, 1974). It remains to be seen, however, whether this facilitative effect induced by mammillary body lesions can be replicated with other sensory discrimination tasks.

Brightness Discrimination Reversal Learning

Six of the 11 brain-damaged groups observed in this experiment were found to be significantly retarded in mastering the reversal of the originally acquired white-black discrimination problem. Their lesions involved the posterior cingulate cortex, lateral

frontal cortex, occipital cortex, mediodorsal thalamic nucleus, parafascicular nucleus, or substantia nigra.

The finding that mediodorsal thalamic lesions are apt to impede visual reversal learning in rats agrees with that of Tigner (1974). On the other hand, the findings that damage to the lateral frontal cortex or occipital cortex produce visual reversal learning losses would appear to be inconsistent with those reported by Boyd and Thomas (1977) and Jeeves (1967). This discrepancy could largely be due to the size of the lesions investigated; the cortical ablations made in the current experiment were at least twice as large as those examined in the latter two experiments. As far as can be determined, visual reversal learning deficits arising from posterior cingulate, parafascicular, or nigral lesions have not been reported previously.

Impairments in visual discrimination reversal learning have previously been reported in rats prepared with hippocampal system damage (Becker & Olton, 1980; Samuels, 1972; Silveira & Kimble, 1968) or medial frontal cortical damage (Becker & Olton, 1980). In the present study, those groups with dorsal hippocampal or medial frontal (including anterior cingulate) damage showed no clear evidence of being deficient in learning the reversal of the original white-black discrimination problem despite the moderate size of the lesions sustained by these two groups. Although other explanations are certainly available, it is suggested that these conflicting findings may be due, at least in part, to the fact that the present study utilized the motive of escape-avoidance of footshock, while the earlier studies utilized an appetitive motive. That aversive motivation (including punishment for errors) can obliterate visual discrimination learning differences between certain brain-damaged animals and controls is not an infrequent observation (Krechevsky, 1936; Sechzer, 1964; Warren, Warren, & Akert, 1962).

Although a number of brain-damaged groups learned the reversal of the original white-black discrimination significantly more slowly than the controls, this result alone does not warrant the conclusion that a "reversal deficit" was present. To demonstrate the latter, a given brain-damaged group should earn poorer negative savings scores on the reversal problem than the controls. According to the results presented in Table 2, the only groups that exhibited a significant (or marginally significant) reversal deficit had lesions to the posterior cingulate cortex, mammillary bodies, mediodorsal thalamus, or substantia nigra. (Due to the termination of the occipitaleyctomized animals on the reversal problem prior to reaching the learning criterion, it was not possible to obtain an adequate assessment of their negative savings scores.) The finding that a reversal deficit was associated with mediodorsal thalamic and mam-

millary body lesions is especially interesting in view of the recent report by Oscar-Berman and Zola-Morgan (1980) that alcoholic Korsakoff patients (presumed to have damage to the mediodorsal thalamus and/or mammillary bodies) also exhibit deficits in reversal learning.

The General Learning System

Preliminary Findings

As discussed at the outset of this paper, there is a basis for the belief that those lesion placements impairing acquisition of both spatial and visual discrimination habits in the rat will define, at least provisionally, the components of the rodent's GLS of the brain. Of the 11 brain regions examined in this study, each of which was previously found to be implicated in acquisition of a position habit and its reversal (Thompson, 1981, Note 1; Thompson et al., 1981; Thompson & Yang, 1982), only 3 were found to be implicated in acquisition of a white-black discrimination habit and its reversal. These regions consisted of the substantia nigra, parafascicular nucleus, and occipital cortex.

In light of earlier findings, it appears quite reasonable to include the nigral and parafascicular regions within the GLS of the rat brain. Rats with bilateral damage to either of these regions have been reported to exhibit relearning disturbances (errors to relearn postoperatively being greater, in many cases, than errors to learn preoperatively) on a wide variety of laboratory tasks, including visual discriminations (Thompson, 1976), an incline-plane discrimination (Thompson et al., 1976), a 3-cul maze (Thompson, 1974), and latch-box problems (Spiliotis & Thompson, 1973; Thompson et al., 1979).

The occipital cortex, on the other hand, does not reasonably qualify as a component of the rodent's GLS, because damage to this cortical area has not been found to retard acquisition of certain nonvisual discrimination problems.³ For example, Finger and his associates (Finger, Cohen, & Alongi, 1972; Finger & Frommer, 1968; Gabrial, Freer, & Finger, 1979) have shown that occipital injuries in rats have little effect on either original learning or reversal learning of a tactile discrimination task. (Although nigral lesions have apparently not been examined in relation to acquisition of a tactile discrimination, parafascicular lesions have been reported to impede tactile discrimination learning—see Finger, 1972.) In addition, it has recently been observed in my laboratory that occipitaleyctomized rats learn an incline-plane discrimination about as efficiently as do operated controls.⁴

Thus, this limited lesion survey of the rat brain has yielded two brain sites, each residing in close proximity to the diencephalic juncture, that are pre-

sumed to be components of the GLS. (A second study of this type is currently under way in my laboratory to identify other components of the GLS.) Interestingly, the data of the present study do not warrant the inclusion of any neocortical or limbic forebrain region within the GLS of the rat brain. This, of course, does not imply that the neocortex and limbic forebrain are devoid of any role in learning. Rather, it means that neocortical and limbic forebrain areas may be critical for the acquisition of certain kinds of laboratory tasks, but not for the acquisition of other kinds of laboratory tasks. In other words, these telencephalic structures have specific functions in learning that are to be contrasted with the seemingly nonspecific functions in learning served by the parafascicular and nigral regions. This account is not inconsistent with that of Oakley (1981), who views the subcortex as participating in associative learning and the neocortical and limbic (principally the hippocampus) areas as being reserved for representational and abstract learning.

It is of further interest to note that the two structures found to qualify as components of the GLS are also components of the GMS (general memory system), while the nine structures found not to qualify as components of the GLS are likewise not components of the GMS (Thompson, Chetta, & LeDoux, 1974; Thompson & Thorne, 1973). This pattern of results suggests that there may be few, if any, sites within the rat brain which, when selectively damaged, will produce generalized anterograde amnesia *without also producing generalized retrograde amnesia*. The foregoing outcome would appear to be in conflict with the clinical literature to the extent that patients with damage to the hippocampus, mediodorsal thalamus, and/or mammillary bodies often exhibit anterograde amnesia with little or no retrograde amnesia (Milner, 1970; Scoville & Milner, 1957; Squire & Moore, 1979; Talland, 1965; Victor et al., 1971). However, it must be pointed out that amnesic patients can learn certain spatial discriminations (Oscar-Berman & Zola-Morgan, 1980), visual discriminations (Gaffan, 1972; Oscar-Berman & Zola-Morgan, 1980), and perceptual and perceptual-motor skills (Brooks & Baddeley, 1976; Cohen & Squire, 1978, 1980) about as rapidly as controls. On the strength of these findings, amnesic patients, such as H.M., N.A., and those with alcoholic Korsakoff's disease, cannot be viewed as having suffered damage to the GLS (of the human brain) and, as a consequence, cannot reasonably be compared with rats having parafascicular or nigral lesions.

Anatomical and Functional Considerations

Since the parafascicular nucleus is anatomically related to the ascending reticular system (Scheibel & Scheibel, 1958), the possibility must be considered

that the learning deficits resulting from lesions to the former are the products of interfering with the influence of reticular impulses upon certain forebrain mechanisms. While this possibility cannot be entirely dismissed, it is weakened by the finding that damage to the dorsomedial sector of the midbrain reticular formation immediately caudal to the parafascicular nucleus does not impair acquisition of a spatial discrimination habit or its reversal in rats (Thompson & Yang, 1982).

Another possibility is that the learning impairments accompanying parafascicular lesions are the consequences of disrupting the normal activities of the basal ganglia and, in particular, those of the corpus striatum. Evidence is increasing that the parafascicular nucleus is functionally (Ahlenius, 1980; Dalsass & Krauthamer, 1981), as well as anatomically (Clavier, Atmadja, & Fibiger, 1976; Gerfen, Staines, Arbuthnott, & Fibiger, 1981; Jones & Leavitt, 1974; Kuypers, Kient, & Groen-Klevant, 1974), related to the nigro-striatal complex. This possibility is of considerable interest to the extent that it would position both the parafascicular nucleus and the substantia nigra within a well-recognizable neurological system that may participate in some way in the formation of a wide spectrum of learned activities. It has already been established that interruption of the afferent and/or efferent connections of the corpus striatum leads to acquisition impairments on a variety of laboratory tasks in the rat (Fibiger, Phillips, & Zis, 1974; Kelly, Alheid, McDermott, Halaris, & Grossman, 1977). It remains to be seen, however, whether strategically placed striatal lesions (those damaging both neostriatal and pallidal elements) will produce retardation in learning the specific spatial and visual discrimination habits investigated in the current series of experiments. (The caudoputamenal lesions examined in the present study were too rostral, sparing all of the pallidum and most of the caudoputamen, to test the significance of the corpus striatum in visual discrimination learning.)

Given the likelihood that the corpus striatum and the parafascicular and nigral regions constitute a major portion of the GLS of the rat brain, the question arises concerning the role played by this neurological system in learning. An "arousal-activation" role and a "motor control" role are worthy of mention inasmuch as they have previously been advanced to describe the functions of both the parafascicular-center median complex (Delacour, 1971) and the nigro-striatal complex (Pycock, 1980; Ungerstedt, 1971) in behavior. What is intriguing about these proposals is that they do not bear directly on either the neural processes or the neural substrates involved in learning. If subsequent studies confirm either one (or both) of these roles for the entire nigro-parafascicular-striatal complex, then the GLS, as currently defined,

may simply provide those conditions (arousal and/or motor control) that are prerequisite for the engagement and/or expression of the learning process.

Final Remarks

Localizing the GLS by studying the effects of focal brain lesions on acquisition of two tasks has its weaknesses and limitations. One weakness relates to the claim that those lesion placements impairing acquisition of both spatial and visual discrimination habits will also induce impairments in acquisition of other classes of laboratory tasks. This claim will probably be subject to certain exceptions. (One exception already demonstrated in the current study involved the occipital cortex.) The possibility exists, for example, that lesions to the nigral or parafascicular regions may not retard acquisition of those tasks that fail to require the animal to advance from one locus within the environment to another to obtain a reward. Such tasks would include passive avoidance, taste-aversion, and classically conditioned responses. This possibility is mentioned because the nigro-parafascicular-striatal complex may emerge in importance in those situations involving "goal-reaching" activities in the unrestrained, free-moving animal (see Thompson, 1982a).

Another weakness has to do with the well-established observations that the appearance of a learning deficit in brain-damaged animals may be contingent not only upon lesion topography, but also upon lesion magnitude, whether or not the lesions are produced in one or two stages, the length of the recovery period, the age and previous experience of the subject, the difficulty of the task, and the like (Finger, 1978; Stein, Rosen, & Butters, 1974). Thus, the lesion-defined GLS exposed in the current study can hardly be viewed as having unlimited generality.

Perhaps the most serious weakness of the methodology adopted in this series of experiments concerns the uncertainty involved in interpreting the "global" learning deficit arising from damage to the nigral and parafascicular regions. At present, it is not known whether the cells intrinsic to these regions, the fiber pathways coursing through these regions, or both are significant for learning. Nor is it clear whether one sector of each region is implicated in spatial discriminations and another in visual discriminations or whether the entire region of each contributes equally to the acquisition of both spatial and visual discriminations. And it cannot be ascertained whether the learning losses following nigral or parafascicular lesions are due to secondary morphological, physiological, and/or biochemical effects.

Despite these shortcomings, the disclosure of a lesion-defined GLS is of value in demonstrating a correspondence between a global learning deficit and the destruction of discrete anatomical sites within the brain. Information of this sort, which has been conspicuously absent in the experimental literature, also

serves to call attention to a highly localized group of brain structures that could play a pivotal role in the learning process.

REFERENCE NOTE

1. Thompson, R. Unpublished observations, 1981.

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NOTES

1. Shaving the vibrissae has routinely been carried out in my laboratory on rats trained on visual discrimination problems in which an error is defined as an approach response to the negative stimulus card which brings the animal's forefeet in contact with

an electrified grid. This has been done in order to prevent the possibility that the vibrissae might serve to detect the presence of the charged grid section.

2. During original and/or reversal learning, some rats would dash out of the startbox toward one of the stimulus cards. In an effort to reduce running speed, a delay of 10-20 sec was occasionally imposed between inserting the animal into the startbox and raising the startbox door.

3. The occipital cortex has long been known to have a non-visual function (Lashley, 1943) which, according to a recent analysis (Thompson, 1982b), is probably related to some aspect of what O'Keefe and Nadel (1978) refer to as "place" learning. Since the spatial discrimination task most likely involves place learning and since the white-black discrimination task obviously involves visual learning, it would be expected that occipitlectomized rats would exhibit a learning impairment on both tasks. With the possible exception of the lateral geniculate nuclei, no other brain structures would conceivably play a role in both place learning and visual learning.

3. Four occipitlectomized rats and four sham-operated controls were trained on the incline plane discrimination problem described elsewhere (Thompson et al., 1976). The former learned the problem in an average of 29.8 trials and 13.3 errors, while the latter earned mean learning scores of 25.3 trials and 14.8 errors. None of the differences approached statistical significance.

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