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Radial-maze performance in the rat following lesions of posterior neocortex

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Summary

The present experiment was designed to investigate the role of posterior neocortex (areas 17, 18 and 18a) in the maintenance of performance on the radial maze. Following training to criterion on the 8-arm radial maze, rats received either sham operations, bilateral eye enucleations, lesions of posterior neocortex, or combined enucleations and lesions of posterior neocortex. While the enucleated animals with intact brains showed a slight, but significant performance decrement relative to the sham-operated group, the other two groups, with lesions of areas 17, 18 and 18a, each showed a massive deficit. This large deficit was observed even in the group in which both the eyes and neocortex had been removed. These results suggest that the visual projection areas of cortex not only play an important role in the maintenance of accurate radial-maze performance in sighted animals, but that the integrity of these areas is necessary for the maintenance of criterion performance in blind animals.

Introduction

Rats, like most animals, are able to distinguish places they have visited from places they have not. In the laboratory, this ability has been effectively demonstrated with the use of the radial maze, an apparatus consisting of 8 equally-spaced arms, projecting radially from a central platform [10]. The radial-maze task differs from more traditional maze tasks in that the animal is not required to follow the 'correct' route from start box to goal in order to collect food; instead, it is released on the central platform and is allowed to obtain a small amount of food from the end of each of the 8 arms. A rat's performance on this task is measured in terms of the number of arm-entries it makes before obtaining all of the food. Thus, a rat must make at least 8 choices, but may require many more before choosing each of the 8 arms at least once.

Rats are remarkably efficient on this task. Olton and Samuelson [10] found that after only a minimal amount of training their rats could accurately discriminate between those arms which they had previously entered and those which they had not. This discrimination was shown to be largely dependent on cues available outside the maze itself [12], and several recent studies have indicated that visual cues are of primary importance for accurate performance on this task [1,9].

The present study was designed to examine the effects of peripheral blinding (enucleation) and lesions of posterior neocortex (areas 17, 18 and 18a) on rats' behavior in a radial maze. It was

based on two sets of previous findings: those derived from studies employing the more traditional mazes, in which the rats were required to follow a particular route from start to finish, and those employing the radial maze, in which they were required to remember which arms they had already visited.

A number of investigatory [5,6,16,17], using the familiar Lashley III maze, found that lesions of primary visual neocortex (principally area 17) produced larger deficits in acquisition and retention than did either peripheral blinding by enucleation or sectioning the optic radiations. Moreover, Lashley [6] showed that combining enucleation and lesions of posterior neocortex resulted in even greater deficits, and according to Tsang [17] the cortical lesions produced large deficits in previously enucleated rats, even if they had been blinded as neonates. Both Lashley and Tsang suggested that the visual areas in cortex not only play an important role in the learning of complex mazes when vision is intact, but also influence spatial orientation when visual cues are not, and never have been available.

The more recent radial-maze studies have also found acquisition and retention deficits following enucleation [1,18], although the size of the retention deficit following enucleation was shown to decrease as the amount of preoperative training was increased [1]. Large lesions of neocortex centered on area 17 have also been shown to interfere with the acquisition and retention of accurate radial-maze performance in sighted animals [3]. In none of these studies, however, was the effect of cortical lesions directly compared with that of enucleation. In other words, it is unclear whether lesions of the visual areas of neocortex produced larger deficits in radial-maze performance than did enucleation (as they had in the earlier studies employing Lashley III mazes).

Therefore, in the present study, the effects of enucleation, of lesions of posterior neocortex, and of combined enucleation and cortical lesions were directly compared in different groups of rats trained in the radial maze. By making such comparisons, it was possible to evaluate the earlier suggestions of Lashley [6] and Tsang [17] that area 17 and the peristriate areas of cortex play a major role in the performance of spatial tasks even when visual input has been completely disrupted.

Method

Subjects

Twenty-one male Long-Evans hooded rats about 4 months of age were used in this experiment. Throughout the testing periods they were maintained at 80% of their free-feeding weights by food deprivation. All the animals were fed within 2 h after each test session and had continuous access to water in the home cage. All the animals were individually housed at 20-22°C under a 12 h : 12 h light/dark cycle. The housing room was dimly illuminated by a 40 W desk lamp directed towards one corner of the room during the 'dark' phase of the cycle, and brightly by fluorescent ceiling lamps and the desk lamp during the 'light' phase of the cycle.

Apparatus

An elevated straight runway was used in pretraining and an elevated eight-arm radial maze was used in testing. Both were constructed of 1.2 cm and 2.4 cm thick plywood. The elevated straight runway was 112 cm long and 8.5 cm wide. It was unpainted and its surface was 62 cm above the floor. A small plastic cup was placed 4 cm from each end of the runway. The plastic cups were made from the tops of Kodak 35-mm film containers with internal diameters of 3 cm and sides of 0.5 cm high.

The elevated radial maze consisted of 8 equally-spaced arms projecting radially from an octagonal platform, with adjacent arms being separated by 45°. The central platform was 30.8 cm in diameter and each of the arms was 76 cm long and 7.5 cm wide. The maze was painted grey and its surface was 62 cm above the floor. There was a small rectangular metal container measuring 7.5 × 5 × 2.5 cm located at the end of each arm of the maze. Food pellets placed in these containers were not visible from the central platform. All pretraining and testing was conducted in a large room (5 × 5 m) containing a variety of objects (e.g. blackboard, bookcase, desk, curtains) constituting a visually heterogeneous environment. The rats were always tested during the dark phase of the light/dark cycle and the level of illumination in the testing room was between those in the housing room during the light and dark phases of the light/dark cycle.

Procedure

All animals received preoperative training, were then subjected to one of several surgical procedures (see below), and were then tested postoperatively. Some animals received a second surgical operation followed by a second block of postoperative testing.

Pretraining

Each animal was given two daily 5-min sessions on the straight runway. Before each session, two 45 mg Noyes pellets were placed in the cups at each end of the runway. All the animals started each session at the center of the runway, facing towards one end on the first session and towards the other end on the second session. Each rat was permitted to move freely about the straight runway for the 5-min duration of each trial. The pre training provided the animals with experience moving above elevated platforms and narrow pathways and facilitated the later radial-maze testing.

Preoperative training (Phase 1)

Each animal was administered one trial a day on the radial maze for 5 or 6 days every week. At the beginning of a trial, each of the 8 containers on the maze was first baited with one 45 mg Noyes food pellet, and the rat was then placed on the central platform in front of, and oriented towards, one of the 8 arms. The starting position was varied from trial to trial according to a pseudo-random schedule. After being placed on the central platform, the rat was allowed a maximum of 7 min or 16 choices (arm entries) to choose each of the 8 arms of the maze. An arm choice was recorded whenever the rat entered an arm as far as the root of its tail; that is, whenever all of the animal's body, except its tail, passed the vertical plane separating the arm

from the central platform. Once the rat entered an arm, it invariably continued towards the food container.

The sequence of arm entries (choices) and the time required to reach the final food container were recorded manually. The arms were assigned the numbers 1 through 8 (in a clockwise direction) for recording the choice sequences. The main response measure was the 'choice score,' the number of different arms chosen in a rat's first 8 choices on a trial. The rats were trained preoperatively to a minimum of 15 trials, and then received additional training until they reached a performance criterion of 5 consecutive trials with a mean choice score of 7.2. After reaching criterion, each rat was assigned to one of four different experimental groups (see below). An attempt was made to match animals in the different groups according to the amount of preoperative training they received and the final mean choice score they achieved.

Surgery

All surgical manipulations were carried out under deep anesthesia (Equithesin: 3 ml/kg i.p.). Six animals were peripherally blinded by enucleation (Group E). Five animals received aspiration lesions of posterior neocortex (Group VC). An attempt was made to remove all tissue in area 17 with excursions into areas 18 and 18a. Five animals were both peripherally blinded and had lesions of visual cortex (Group E + VC). Finally, 5 additional animals were used as a control group (Group C) and received sham operations in which the scalp was incised, holes were drilled into the skull, but no tissue was removed. All animals were allowed 2 weeks to recover before postoperative training began.

Postoperative testing (Phase 2)

All the animals were tested on the radial maze under the same procedure used preoperatively. Each rat was tested until it re-attained the performance criterion of 5 consecutive trials with a mean choice score of 7.2, up to a maximum of 50 trials.

Postoperative testing (Phase 3)

Some of the animals reaching criterion within 50 trials were given a second surgical manipulation. All 5 animals in Group C were given a second sham operation, and all 6 animals in Group E were given lesions of posterior neocortex (becoming Group E → VC). None of the other animals underwent surgery. All of the animals receiving this second round of surgery were given at least 2 weeks to recover before retested on the radial maze. The same testing procedure outlined above was used during this last phase of the experiment.

Histology

At the end of the experiment, the 16 rats with brain lesions were killed with sodium pentobarbital and were perfused with 10% formalin. Their brains were removed and fixed in sucrose formalin for several days. The dorsal surface of the entire brain was photographed and the gross extent of the cortical lesions was plotted on a standard diagram of the cortical surface. The brains were then frozen and coronal sections were cut at 40µm through the posterior thalamus. Every third

section was mounted and stained with cresyl fast violet. Pars dorsalis of the lateral geniculate nucleus and the lateral posterior nucleus were examined for retrograde degeneration.

Results

Histology

As Fig. 1 illustrates, the cortical ablations were large and appeared to include most of area 17 and large portions of area 18 and 18a in the majority of animals. Area 7 and to a much lesser extent area 29 were also invaded in some animals.

Fig. 1 also illustrates the amount of retrograde degeneration observed in the dorsal part of the lateral geniculate nucleus and in the lateral posterior nucleus of the thalamus of each animal. In most of the animals, degeneration was complete or nearly complete throughout the entire lateral geniculate. However, in two of the animals, Subject 7 from Group VC and Subject 26 from E + VC, there was a moderate sparing of normal cells on one side, indicating that the ablation of area 17 in those animals was far from complete. Retrograde degeneration in the lateral posterior nucleus varied considerably from animal to animal but seemed to be correlated with the amount of degeneration observed in the lateral geniculate nucleus. This was probably due to the fact that the larger lesions, which included all of area 17, invariably encroached upon areas 18 and 18a and sometimes area 7 [4]. The hippocampus was damaged in only one animal, Subject 7 from Group VC, and even here the lesion extended into hippocampal structures on only one side. In none of the animals was there significant degeneration in the ventral nucleus of the thalamus and gross inspection of the cortical lesions indicated that neither motor cortex nor entorhinal cortex had been invaded in any animal.

Behavior

Three aspects of postoperative behavior were compared in the 4 groups of animals: their initial choice accuracy immediately postoperatively, the rate at which they reattained criterion, and the serial position of their errors. Choice accuracy was measured in two different ways: the number of errors per trial and the number of different arms chosen in the first 8 choices (the 'choice score' described earlier).

Initial choice accuracy

The mean numbers of errors for each rat over the last 5 preoperative trials in Phase 1 and the first 5 postoperative trials in Phase 2 are shown in Table 1. The mean number of errors made during the last 5 trials of Phase 2, before reaching criterion or completing 50 trials, are also summarized in this table along with the mean number of errors in the first 5 postoperative trials of Phase 3. Only Groups C and E were exposed to Phase 3.

Since the groups were matched on the basis of their preoperative performance, it is not surprising that their preoperative error scores were highly similar. However, as Table 1 summarizes, at the

Fig. 1. Schematic drawing of locus and extent of the cortical lesions. The solid black area on the dorsal view of the whole brain indicates the size and location of cortical damage. The solid black areas in the left and right coronal diagrams of the thalamus represent the amount of retrograde degeneration in the lateral geniculate nucleus (LGN_d) and later posterior nucleus (LP) on each side. Group VC received only cortical lesions; Group E + VC, combined enucleations and cortical lesions; and Group E → VC, enucleations followed by cortical lesions.

beginning of Phase 2, the sham-operated group (Group C) continued to make few errors, while the enucleated group (Group E) showed a moderate decrease in performance. In contrast, the group with cortical lesions (Group VC) and the group with enucleations as well as cortical lesions (Group E + VC) both exhibited large increases in their error scores. Not surprisingly, a two-way analysis of variance, in which the effects of lesion and of enucleation were treated as separate factors, revealed a significant main effect of lesion, [$F(1, 17) = 95.3, P < 0.01$] although the main effect of enucleation was not statistically significant [$F(1, 17) = 2.7, P > 0.1$].

There was, however, a significant enucleation $\times$ lesion interaction, [$F(1, 17) = 11.1, P < 0.01$]. Fig. 2, in which this interaction is plotted, suggests that enucleation had more of an effect on animals without brain lesions than it did on those with lesions of posterior neocortex. This interpretation is supported by the results of a posthoc analysis (Tukey's test: $\alpha = 0.05$) which indicated that the enucleated animals without brain lesions (Group E) made significantly more errors than the sham operates (Group C), and that both groups with lesions (Group VC and E + VC) made more errors than the enucleates although they did not differ from one another.

By the last 5 trials of Phase 2, the sham operates (Group C) and the enucleates (Group E) were making relatively few errors per trial and there was no significant difference in their

Table 1. Mean number of errors per trial and mean choice scores for the five trials preceding and following each surgical manipulation

Group	Subject	Phase 1		Phase 2 (postop.)*		Phase 2 (preop.)		Phase 3**	
		Error score	Choice score	Error score	Choice score	Error score	Choice score	Error score	Choice score
C	3	1.8	7.2	2.6	7.0	1.6	7.6	0.8	7.2
	9	1.2	7.6	0.6	7.6	0.6	7.6	1.4	7.4
	13	1.4	7.2	0.6	7.4	0.6	7.4	1.2	7.0
	16	1.8	7.2	2.0	6.6	1.2	7.4	0.6	7.4
	25	1.6	7.4	1.2	7.2	0.4	7.6	0.8	7.4
	x	1.56	7.32	1.40	7.16	0.88	7.52	0.96	7.28
E	1	1.8	7.2	2.8	6.8	0.8	7.2	5.2	6.4
	14	1.6	7.2	4.6	6.8	2.8	7.2	6.6	6.2
	17	1.8	7.4	2.2	6.8	3.0	7.2	9.0	5.2
	22	1.4	7.4	3.2	6.8	1.0	7.4	4.2	6.2
	23	1.2	7.4	3.6	6.4	3.8	7.2	9.2	5.4
	30	2.4	7.2	5.4	5.6	3.8	7.2	7.4	5.8
	x	1.70	7.30	3.63	6.53	2.07	7.23	6.93	5.87
VC	2	2.4	7.2	7.2	5.4				
	5	2.6	7.2	7.4	5.6				
	7	1.2	7.4	7.6	6.2				
	18	1.8	7.4	7.2	5.2				
	27	1.2	7.2	7.0	5.4				
	x	1.84	7.28	7.28	5.56				
E + VC	11	1.2	7.4	6.0	5.6				
	12	2.4	7.2	8.6	5.2				
	19	0.6	7.6	5.4	6.4				
	24	2.4	7.2	7.2	6.0				
	26	1.0	7.2	5.4	6.2				
	x	1.52	7.32	6.52	5.88				

* Before Phase 2, Group C received sham operations, Group E enucleation, Group VC cortical lesions and Groups E + VC combined enucleations and cortical lesions

** Before Phase 3, Group C received a second sham operation and Group E received cortical lesions.

performance. However, while the sham-operated group made no more errors per trial at the start of Phase 3 than at the end of Phase 2 [$t(4) = 0.25$, $P > 0.1$], the enucleated animals showed a dramatic increase in their error scores [$t(5) = 6.26$, $P < 0.01$]. The large number of errors per trial made by this group after the cortical lesions was very similar to the numbers of errors per trial in the groups given concurrent enucleation and cortical lesions or cortical lesions alone.

As Table 1 illustrates, a similar pattern emerges when the choice scores for each group are examined. Preoperatively in Phase 1, there were no differences between the 4 groups. In Phase 2, however, a two-way analysis of variance revealed a significant main effect of the lesion on

Fig. 2. Mean number of errors per trial in the first 5 trials of Phase 2 in the sighted and blind animals. Group C received only sham operations; Group E, enucleations; Group VC, cortical lesions; and Group E + VC, combined enucleations and cortical lesions.

postoperative choice-scores [$F(1, 17) = 34.27, P < 0.001$], although the main effect of enucleation was not significant [$F(1, 17) = 0.63, P > 0.4$]. The significant interaction between enucleation and lesion [$F(1, 17) = 6.05, P < 0.05$] again suggests that enucleation had more of an effect on animals without brain lesions.

The sham-operated group obtained slightly lower choice scores after the second sham operation (7.28) than before it (7.52), with the difference being significant at the 5% level [$t(4) = 3.21$]. In contrast, the cortical lesions produced large choice decrements in those animals which had already been enucleated. This drop in performance was highly significant statistically [$t(5) = 7.22, P < 0.01$] and similar to the decrements resulting from simultaneous enucleations and cortical lesions or from cortical lesions alone.

Re-attaining criterion performance

Since the groups were matched in terms of preoperative experience, there were no significant differences in the number of errors made by each group in attaining criterion performance in Phase 1 (Fig. 3). In Phase 2, however, the error scores for each animal were clearly influenced by the surgical manipulations. Whereas the sham-operated group re-attained criterion after making an average of only 8.0 errors, the enucleation group made 78.3 errors. As Fig. 3 shows, the brain-damaged animals were even more impaired, the group with cortical lesions alone making 274 errors and the group with combined enucleations and lesions making 250.4 errors. The group means for the latter two groups underestimate the size of their impairments for two reasons. First, most of the subjects in each group failed to re-attain criterion performance within the

Fig. 3. Total number of errors required to reach criterion in each of the three phases of the experiment. Group C received only sham operations; Group E, enucleations and then as Group E → VC, cortical lesions; Group VC, cortical lesions; and Group E + VC, combined enucleations and cortical lesions.

arbitrary limit of 50 trials. Secondly, two animals, one in each group, achieved criterion in 8 trials, postoperative performances which were very different from the other animals in those groups. Indeed, the error scores of these two animals (Subjects 7 and 26) were in the low end of the range for the enucleated animals (40 and 34 errors, respectively). It is interesting to note (see Fig. 1) that subjects 7 and 26 were the only operated animals to show normal cells throughout large parts of the lateral geniculate nucleus and the lateral posterior nucleus of the thalamus. Even with their scores included, a one-way analysis of variance carried out on logarithmic transformations of the raw scores revealed a highly significant difference between the groups [$F(1, 17) = 18.42, P < 0.001$], and a post-hoc analysis indicated that the two brain damaged groups made significantly more errors than either the enucleated or the sham-operated group ($P < 0.01$). The enucleates also made significantly more errors than the sham-operates ($P < 0.01$).

In Phase 3, the sham-operated group re-attained criterion as quickly as it had in Phase 2. As Fig. 3 illustrates, however, none of the animals in the enucleation group re-attained criterion within the 50-trial limit after receiving lesions of posterior neocortex at the end of Phase 2. Their performance in Phase 3 was significantly worse than in Phase 2 [$t(5) = 7.76, P < 0.01$], and their error scores were at least as large as those produced by combined enucleation and cortex lesions or cortex lesions alone.

Serial position of errors

One final aspect of the results which was analyzed was the nature of the errors (arm repetitions) made by each group in Phase 2. In this analysis, only those trials in which an animal repeated an

Fig. 4. The percentage of choices (arm entries) after 7 consecutive different choices that were the same as the first three on that trial. Group C received only sham operations; Group E, enucleations; Group VC, cortical lesions; and Group E + VC, combined enucleations and cortical lesions. The performance of all the animals in Phase 1 is indicated by the dot on the right hand side of the graph.

arm after seven consecutive different choices were considered. Olton and Samuelson [10] have already shown that intact animals tend to avoid repeating their most recent choices, with the consequence that the initial few choices on a trial are repeated far more often than later choices (even after correcting for the differential opportunity to repeat the early choices). This observation was confirmed in the present experiment when the frequencies with which the intact animals in Phase 1 repeated serial positions 1-3 and 4-6 were compared. Serial position 7 was not included in the statistical test because it was virtually never repeated; that is, only once in 181 opportunities. As Fig. 4 shows, the intact animals exhibited significant tendencies to repeat their initial choices [$\chi^2(1) = 26$, $P < 0.01$]. The same was true for the sham operated [$\chi^2(1) = 11$, $P < 0.01$] and the enucleation group [$\chi^2(1) = 9$, $P < 0.01$] in Phase 2. However, as Fig. 4 illustrates, the animals with cortical lesions [$\chi^2(1) < 1$] and those with combined enucleations and cortical lesions [$\chi^2(1) < 1$] repeated initial choices no more often than they did later ones. They apparently failed to distinguish between their initial choices and more recent ones.

Discussion

It is clear from the results of the present experiment that lesions of striate and peristriate neocortex in the rat produced greater postoperative defects in radial-maze performance than did peripheral blinding by enucleation. Moreover, such lesions produced deficits in enucleated rats which were similar in every respect to those produced in sighted animals. These results are consistent with those of Foreman and Stevens [3] who found that lesions of posterior neocortex produced large acquisition and retention deficits in rats tested in a similar radial maze. Foreman and Stevens suggested that rats with ablations of area 17 lacked the ability to discriminate between those arms which they had entered and those which they had not. Since all the rats in their study were sighted, however, it might be argued that the poor performance of their visual decorticates was simply due to the reduction in visual acuity that follows striate removal [2]. But the fact that the enucleated animals with intact brains in the present study performed so much better than either sighted or enucleated animals with lesions of visual projection areas of cortex strongly suggests that such lesions disrupt more than the processing of incoming visual information. The difference between the serial position of errors made by the animals with cortical damage and those with intact brains lends support to this argument.

While the current findings also agree in large part with those obtained by Lashley [5,6] and Tsang [16,17] on the Lashley III maze, Tsang found, in contrast to the present study, that combining lesions of posterior neocortex with enucleation produced greater deficits than cortical lesions alone. Although this difference in results may have been due to differences in the size of the lesions, one other plausible explanation is related to differences in the task requirements of the two situations. An animal learning a Lashley III maze must remember the same 'correct' route from trial to trial, whereas an animal tested on the radial maze must learn to discriminate between arms it has visited on a given trial and those it has not: it need not follow the same route from trial to trial. Although it would be possible for normal rats to 'solve' a radial maze in much the same way as they solve more conventional Lashley III mazes (that is, by following a stereotyped response sequence on every trial), they do not appear to do so. Instead, their response sequences can vary enormously over trials [1,9]. This suggests that the spatial relations of the extra-maze cues provide a frame of reference within which an animal can organize its behavior quite flexibly. It is possible that visual cues can be used effectively by a posterior decorticate only when the correct route is invariant across trials.

The lesions in the present experiment were large, including area 17 and extending into areas 18, 18a and the posterior portion of area 7 in most animals. Thus, the cortical targets of both the lateral geniculate nucleus and the lateral posterior nucleus of the thalamus were affected. At present it is unclear whether one or both of these pathways (or their cortical elaboration within area 7 or other areas of neocortex) are critical for performance on the radial maze. In this regard, it is interesting to note that in Foreman and Stevens' [3] study, rats with lesions of the superior colliculus were not nearly so impaired as posterior decorticates. This suggests that the cortical projection from the lateral posterior nucleus, which receives a major inflow from the superior colliculus, may not be as vital to the performance of this task as the geniculostriate pathway.

Thomas [13] and Thomas and Weir [14] have argued, on the other hand, that lesions of ‘parietal’ cortex (apparently including area 7) on the rat interfere with retention on the Lashley III maze. Whatever the outcome, the fact that Subject 7 and 26 in the present study, which had only moderate retrograde degeneration in the lateral geniculate nucleus and almost none in the lateral posterior nucleus, did so well on the radial maze suggests that some part of the visual projection areas is inessential to the performance of this task. Since the amount of cortical tissue removed in these two animals was comparable to that removed in the other operated rats in the study, it cannot be easily argued that any neocortical lesion of the same size will interfere with radial maze performance. While other parts of neocortex have not been systematically explored in this context, as far as posterior neocortex is concerned, the critical area or areas seem to coincide with the visual projection areas.

Extensive lesions of the hippocampus or interruptions of its major afferent pathways (entorhinal cortex, septum, or postcommissural fornix) have also been reported to produce deficits in radial-maze performance [8,11]. The deficits observed in the brain damaged animals of the present study, however, could not have been due to invasion of this hippocampal network since only one animal, Subject 7, sustained any damage to the hippocampus and, even in that animal, it was confined to one hemisphere. The postoperative performance of Subject 7 was also superior to that of any other animal in its group. In none of the animals was entorhinal cortex invaded. Moreover, the animals with interrupted hippocampal connections in the study of Olton et al. [11] typically showed perseverative behavior and often repeated sequences of choices within a trial, a type of behavior that was not observed in the lesion groups of the present study.

In the hippocampal studies described above, control lesions of the dorsolateral neocortex (just above the hippocampus) produced only negligible deficits in radial-maze performance. These lesions were often located within parts of areas 17, 18 or 18a, the same areas which when aspirated in the present study resulted in large and persistent deficits. This apparent contradiction in results can be easily accounted for by differences in the size of the lesions. Although Olton et al. [11] do not report the amount of retrograde degeneration in thalamic nuclei, their cortical lesions were clearly much smaller than even the lesions of Subjects 7 and 26, neither of which exhibited large performance deficits. Had their lesions eliminated the entire geniculostriate projection, they too might have observed deficits comparable to those described in both the present experiment and the study by Foreman and Stevens [3].

The fact that hippocampal lesions disrupt performance on the radial maze has added to the speculation that this ancient telencephalic structure may be an essential neural component in the formation of ‘spatial memories’ [10,15] or ‘cognitive maps,’ which provide an internal representation of space. The poor performance of posterior decorticates, as indicated by both the number of postoperative errors as well as the nature of such errors, suggest that this cortical area may play an important role in the organization of sensory input before its transmission to ‘higher-order’ systems, involving structures such as the hippocampus. Certainly, in intact animals, extra-maze visual cues dominate in the development of efficient performance on the radial maze [1,18]. Under normal circumstances, visual cues provide the most detailed and

precise information about the layout of the environment, and therefore the rat's representation of space might be expected to depend primarily upon visual, rather than auditory, kinesthetic, or olfactory cues. Yet despite the apparent reliance on visual cues, when rats were enucleated after reaching criterion performance on the radial maze, they showed only transient postoperative deficits in performance. On the other hand, lesions of posterior neocortex severely disturbed behavior postoperative in both sighted and peripherally blinded animals. These results suggest: (a) that non-visual cues might gradually be incorporated into a multi-sensory representation of space whose basic structure is determined by the dominant visual cues, and (b) that this representation of space depends in part upon neural circuits within 'visual' cortex. Such an hypothesis can account for the large effects of lesions of the visual projection areas on enucleated animals in both the radial and the Lashley III maze. It can also account for the decline in the effects of enucleation upon radial-maze performance as the amount of preoperative training is increased [1].

Of course, animals need never use visual cues in learning to perform efficiently on the radial maze. Enucleated rats can learn this task without any prior experience, although they typically take much longer than sighted animals to reach criterion performance [1]. In a recent experiment (manuscript submitted for publication) naive enucleated rats which eventually reached criterion were then subjects to lesions of posterior neocortex. In sharp contrast to the findings of the present experiment, they showed only minor and transient deficits in postoperative performance despite the fact that the lesions are at least as large as those described in the present study. While these results suggest that the visual projection areas are implicated in the learning of the radial maze only when animals are initially trained with visual cues available, there were some other differences between the two experiments. Unlike the animals in the present study, the naive enucleates in this experiment were trained under water deprivation and also received a large amount of overtraining. Consequently, they showed a great deal of response patterning and tended to choose the 8 arms in a particular order from trial to trial. Thus, it is possible that it was this response strategy that enabled the animals to perform so well after posterior decortication. But whatever the reason, the results clearly indicate that there are some conditions under which areas 17, 18 and 18a do not disrupt radial maze performance. In the light of these observations, it remains puzzling why, in an earlier study, Tsang [17] found that enucleated rats, trained as naive subjects on the Lashley III maze, where a particular route is followed by trial to trial, were severely disturbed by lesions of these areas of neocortex. This may have been a direct result of the early age (13 days) at which enucleation of the eyes was performed in his study. Removal of the eyes in such young animals may have led to a reorganization of neocortex; areas that would normally have been committed to visual processing may have developed under the influence of other inputs. If this were the case, the effect of lesions in such reorganized neocortex would be difficult to interpret.

At present the hypothesized role of the visual projection areas of neocortex in the performance of spatial tasks such as the radial maze can be described in only very general terms (as can the role of the hippocampus, or any other neural structure, for that matter). A description of the functional network underlying spatial memory in the rat will require a great deal more research. Not only

must the lesions be more discrete and localized, but the stimulus parameters controlling the behavior must be more carefully monitored. What has emerged from the present study is that what has been called ‘visual’ cortex in the rat clearly participates in more than visual processing.

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