THE EFFECT OF D – AMPHETAMINE AND TOTAL STRIATE CORTEX LESIONS ON A VARIETY OF VISUAL TASKS IN THE HOODED RAT

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Partial deficits of visual cliff performance when motivated by escape from electrical shock, and severe deficits on the classical visual cliff and visual placing tasks were found in male hooded rats with total ablation of the striate cortex. However, injection of d-amphetamine was able to partially reverse these deficits. Optokinesis was not affected by the striate ablation. The results were interpreted as evidence that ablation of the striate cortex disrupts the animal's ability to perceive pattern, but the animal can still respond to the particular features of the pattern (eg. brightness) and thus still be able to perform the visual tasks. A neural model was hypothesised to help explain the results.

Braun (1966), and Meyer, Horel and Meyer (1963) found that rats with total ablation of the striate cortex could not visually place; that is, if the lesioned Ss were lowered to a horizontal surface, they did not extend their paws to the surface in anticipation of the landing. However, these same studies also showed that visual placing in such lesioned animals was recovered after injection of amphetamine. The purpose of this study was to extend these findings to other visual tasks which are lost after striate cortex lesioning.

It is only in the visual placing task that the literature is consistent in showing that striate lesions produce visual deficits. Early findings have yielded no evidence that striate lesions interfere with optokinesis (Smith, 1938a; 1938b) but very little subsequent research has been conducted using this procedure. Although it is not a direct concern of this thesis to attempt an explanation of inconsistent results, it should be noted that different pattern and depth discrimination studies of striate lesioned animals do not agree in their findings. Bauer and Hughes (1970), Bland and Cooper (1970), Horel, Bettinger, Royce and Meyer (1966) and Meyer, Anderson and Braun (1966) using a Yerkes discrimination apparatus, and Lashley (1930) using the jumping stand, found that adult rats with lesions in the striate cortex showed a deficit in the discrimination of patterns. These workers proposed that this may be due to the loss of the animal's ability to perceive patterns. Jonason, Lawler, Robbins, Meyer and Meyer (1966) also reported a deficit in pattern discrimination and showed further that injections of amphetamine did not facilitate the relearning of a pattern discrimination lost after striate cortex ablation. However, Lewllyn, Lowes and Isaacson (1969)

were able to reverse a similar discrimination deficit after extensive discrimination training without the use of the drug. Meyer (1963) using cats, and Diamond and Hall (1969) using tree shrews found little deficit it the animals' ability to discriminate certain patterns after lesioning.

Inconsistent findings have also been found in different depth discrimination studies. Meyer et al. (1966) and Meyer (1963) found that adult lesioned rats did not discriminate depth on a visual cliff. On the other hand, Cheney and Crow (1969) and Lewllyn et al. (1969) demonstrated depth avoidance by lesioned rats. The latter study obtained these results only after extensive pattern discrimination training.

The present experimentation was concerned with the possibility of generalizing the Braun (1966) and Meyer et al. (1963) results from visual placing to other visual tasks which show deficits due to striate lesioning. In order to do this, however, it was first necessary to determine if a striate lesion disrupts a particular visual behavior. The tasks chosen for this experiment were: visual placing, optokinesis, and visual cliff performance. In addition to the classical single trial visual cliff procedure (see Walk, 1965), a discrete trial procedure was employed in order to establish if differential responding could be accomplished when the cues associated with depth were used as discriminative stimuli in a learning situation. If a visual behavioral deficit were found in any fo the tasks, the amphetamine would be used to determin if a reversal comparable to that demonstrated by Braun (1966) could be achieved.

METHOD

Subjects

Sixty adolescent male Long-Evans hooded rats obtained from Canadian Breeding Laboratories, La Prarie, Quebec, weighing 120-200 gms. were separated randomly into four groups; the lesion drug (L.D.), n = 19; the lesion no drug (L.Nd.), n = 19; the sham drug (S.D.), n = 10; and the sham no drug (S.Nd.), n = 10.

Apparatus

Figure 1 shows the visual cliff apparatus used in this experiment. It measured 39.9 cm. by 43.14 cm. and was 25.38 cm. deep. Two panes of clear glass rested on the top of the apparatus and a sheet of milky lucite formed the translucent bottom. A white centerboard 7.6 cm. wide and 5.0 cm. high was placed on the glass across the width of the apparatus. The depth stimulus was a black and white 1.26 cm. checkered pattern which covered the walls and the lucite bottom on the deep end of the apparatus, and was sandwiched between the two panes of glass on the shallow side.

Seven inches below the lucite floor, on both sides of the cliff were located seven 15 watt and two 100 watt light bulbs. On the deep side, the lights were connected directly to a wall power socket, and under the shallow side the lights were attached to a variac, thus allowing the intensity to be varied so as to equate the luminosity of the checkered pattern at the level of the glass on either side. This light intensity was maintained at 1.5 ft. lamberts (S.E.I. photometer).

The visual cliff was placed in a dark room, 21 in. away from a one way mirror. During the visual cliff and the reinforced depth

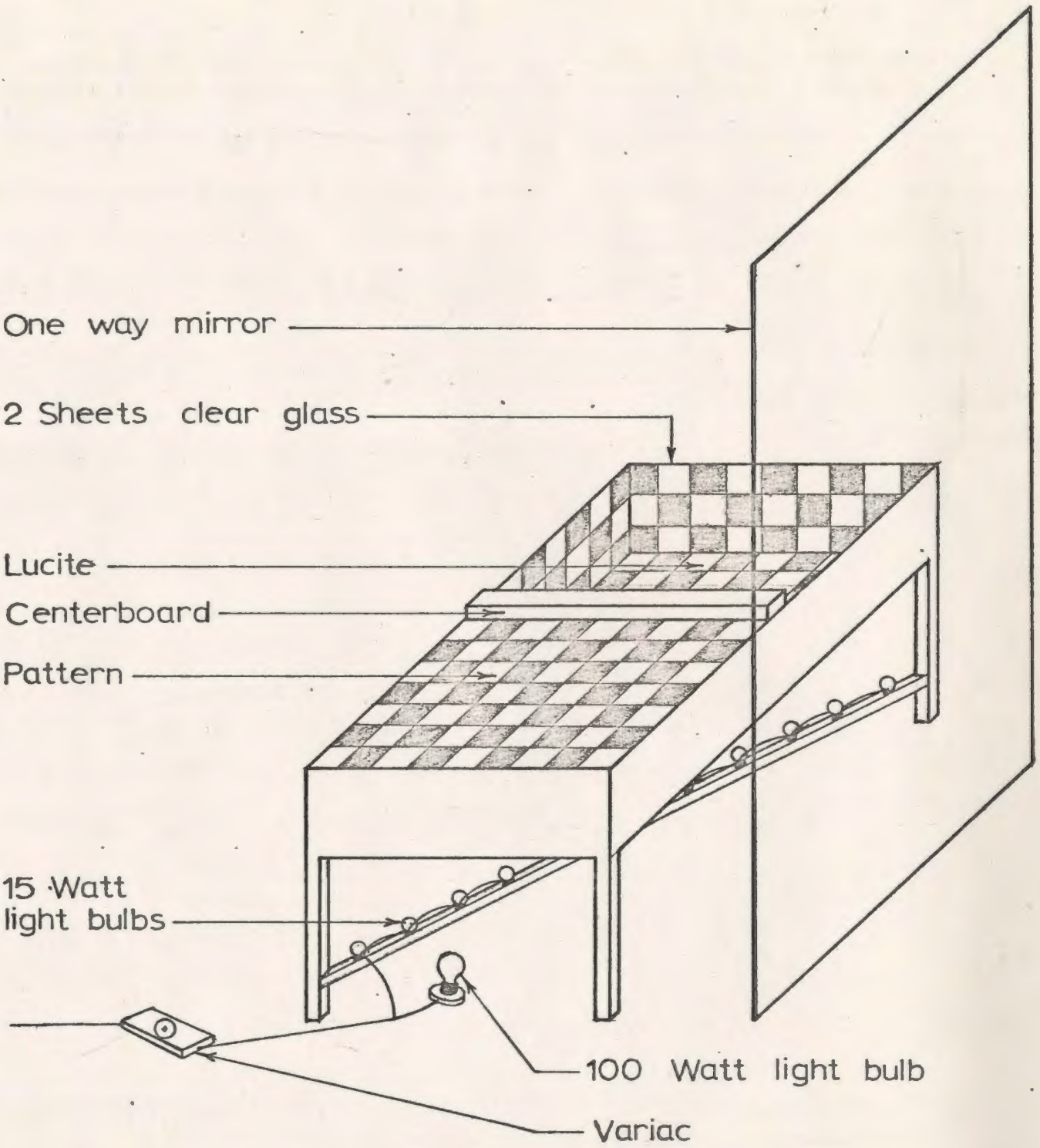

Fig. 1 The visual cliff apparatus

discrimination experiments, white noise was used for masking.

The modified T-maze used to test discrimination learning is shown in Figure 2. The apparatus had no bottom, but rested on the visual cliff apparatus so that the arms rested on each stimulus side, and the stem rested on the centerboard. The walls of the arms and the stem were 13 cm. and 7.6 cm. high respectively, the difference being made up by the height of the centerboard. The combined length of the arms was 71.6 cm. and the stem was 58.4 cm. long. A plexiglass door between the stem and the arms prevented the animal from returning to the stem once the choice point had been reached. Aversive stimulation was used in this procedure and was generated by a LaFayette Instrument Co. shock generator (Model 5226) and administered to the base of the animal's tail by wire attached to two copper rings.

The visual placing apparatus consisted of a brown masonite fork edged with 1.3 cm. black tape and brown masonite backdrop (see Figure 3). The two prongs of the fork were 12.7 cm. apart, extending 7.6 cm. parallel to the floor. This fork rested on a chair, against which the backdrop was propped at a 70° angle to the fork. Thus, when looking down at the apparatus, the black edge of the prongs was all that was readily visible. The entire apparatus reflected .9 ft. lamberts in a diffusely lit room.

The device used to measure the optokinetic effect was a cylinder 71.7 cm. deep and 35.5 cm. in diameter with 1.9 cm. vertical black and white stripes on its interior surface (see Figure 4). This cylinder rotated at a speed of 3.5 rpm. Inside this cylinder a clear glass jar 17.8 cm. in diameter was suspended. Lighting was provided

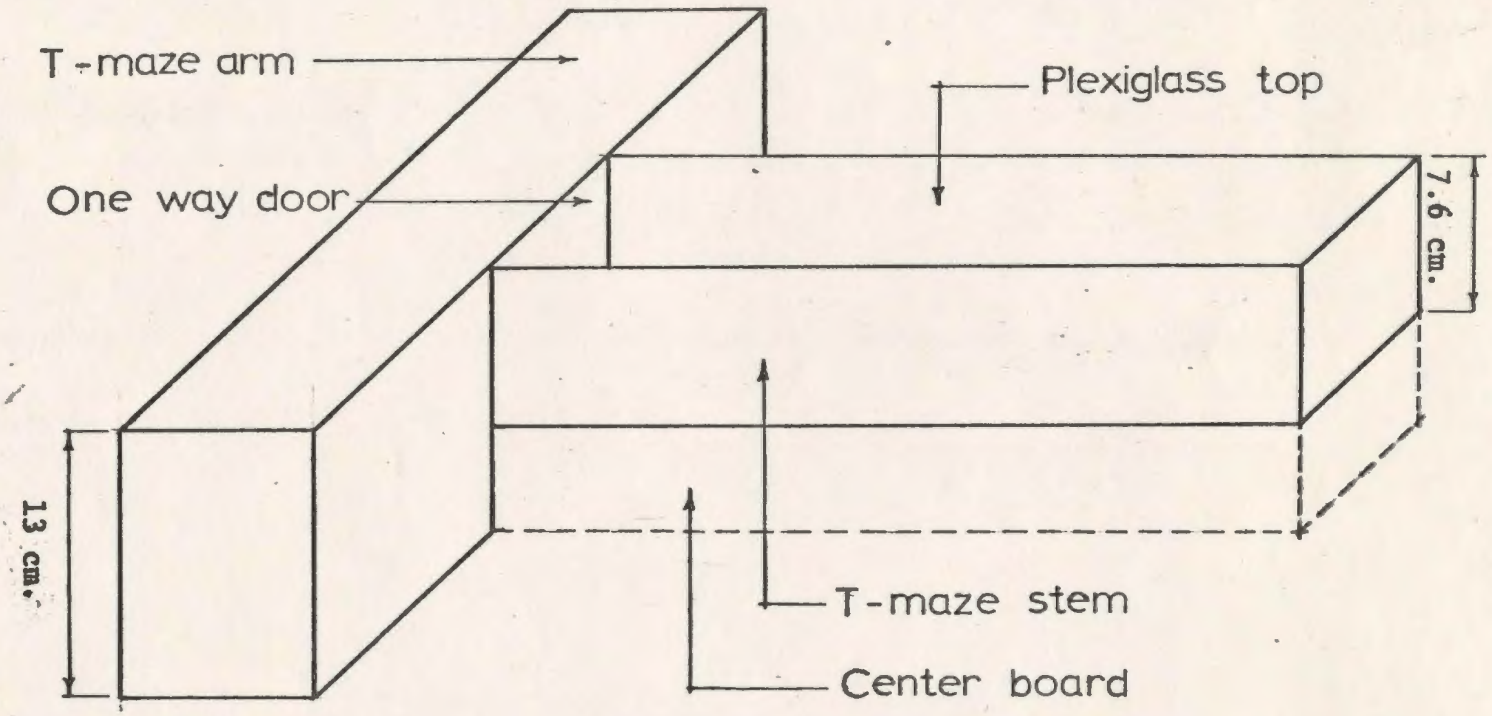

Fig. 2 The T-maze forced choice depth perception apparatus

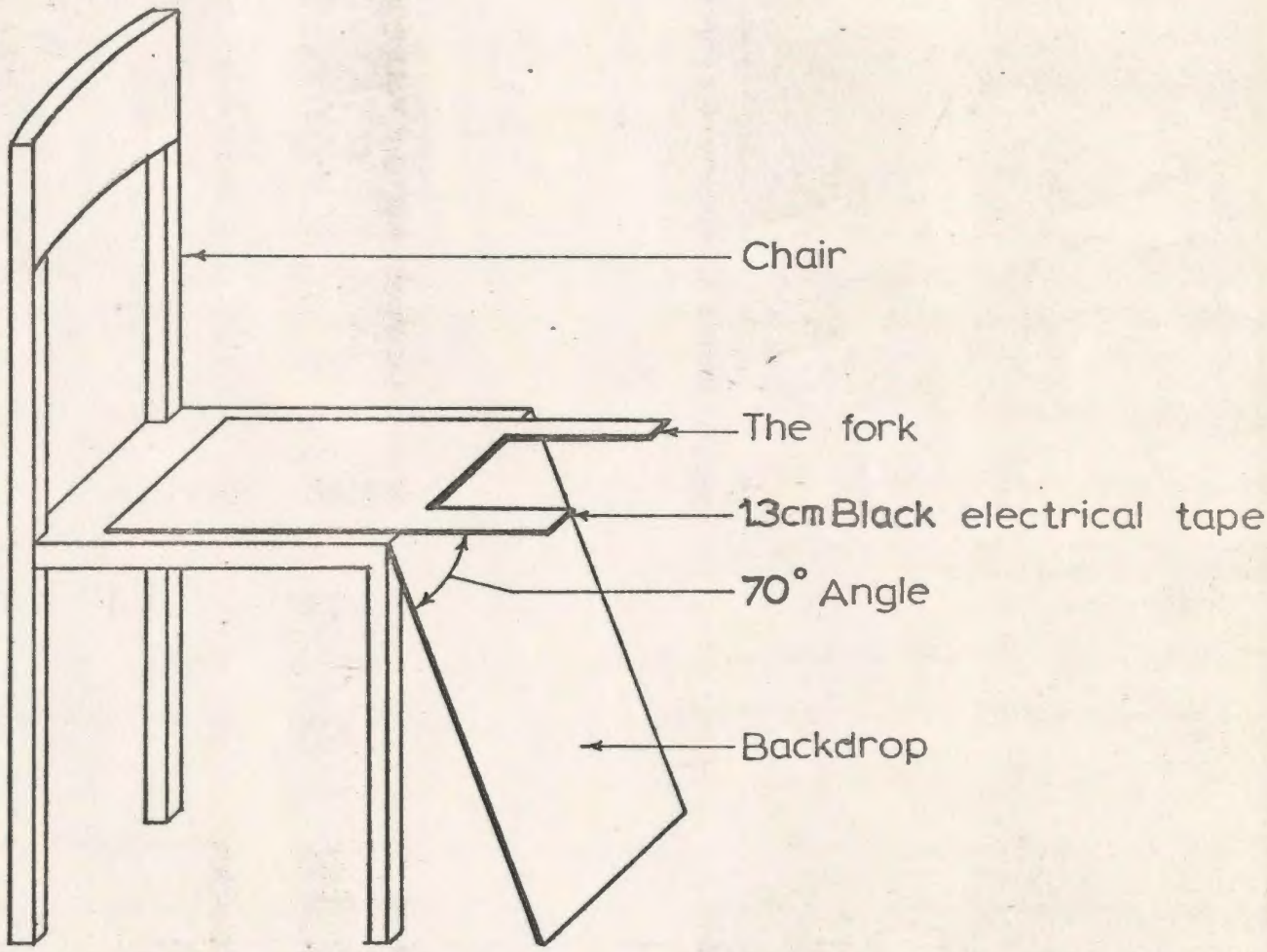

Fig. 3 The visual placing device

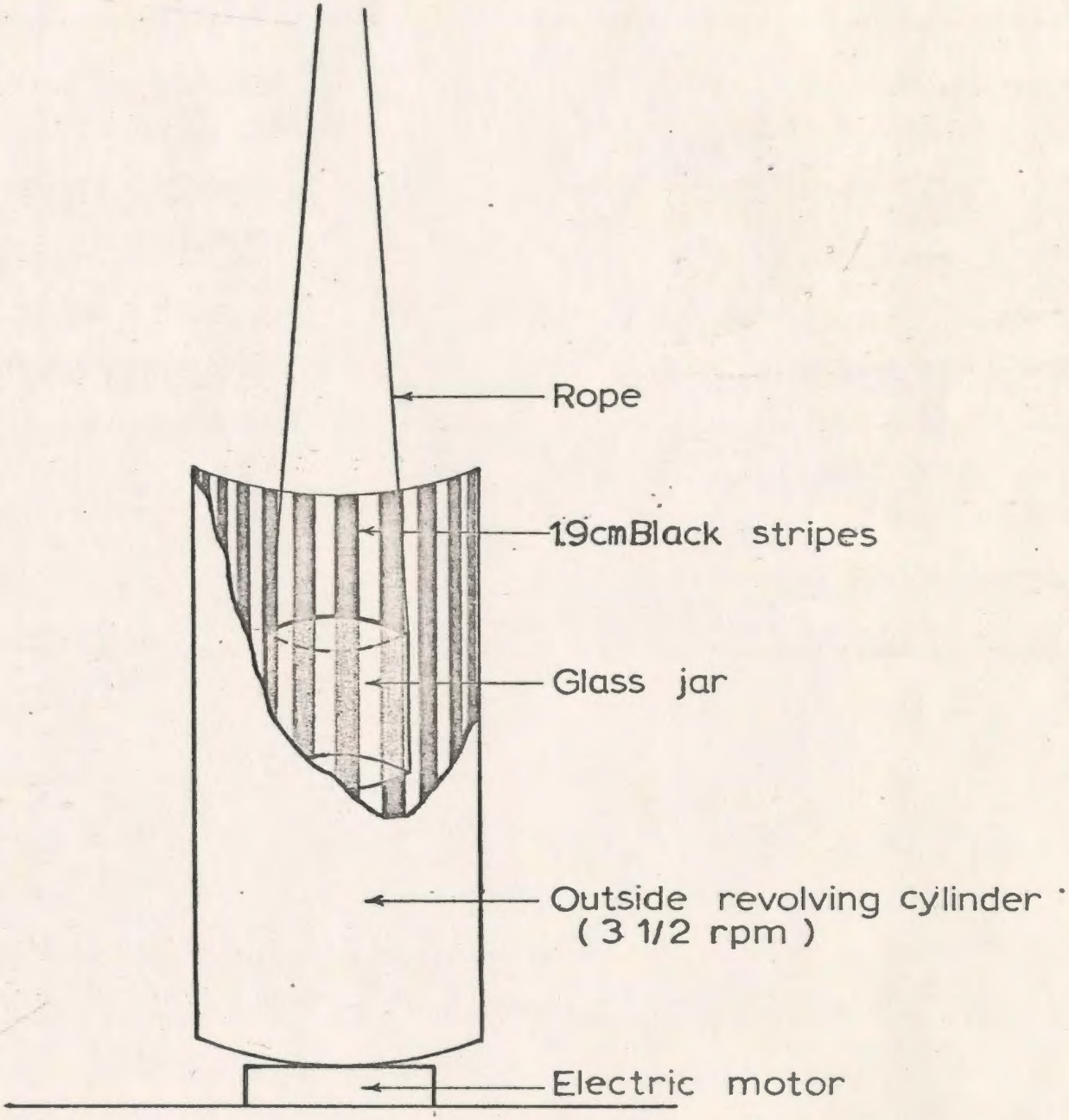

Fig. 4 The optokinetic device

by an overhead incandescent lamp.

Procedure

Under Penthrane gas anesthesia (Registered Tradename), the L.D. and the L.Nd. Ss had their entire striate cortex, including Brodman's area 17, removed by aspiration. The S.D. and the S.Nd. groups had only the dura-mater punctured over the striate area.

Placement of the lesion in the operated groups was primarily determined by landmarks on the skull. The scalp was trimmed of fur and an incision was made along the midline surface from anterior to the ears, to the nape of the neck. Four clamps held the skin and muscle in place while an opening in the skull was drilled with a dental burr. The opening was about 2 mm. left of bregma, going antero-posterally 1 to 2 mm. parallel to the midline. The opening was completed by drilling along the ridge and then cutting back towards the original starting point. The same area was removed from the other half of the skull. Once the openings were made, the dura-mater was pierced with a needle and the appropriate area of the cortex was suctioned out using a drawn out pipette (diameter of about ½ mm.) which was attached to the rubber hose of the machine (Sklar Suction, Model 100-560). Suction level was maintained relatively low. Following the operation, the animals were first placed in plastic cages under heat lamps until they recovered from anesthesia, and then were placed in pairs into smaller cages (20.3 x 17.8 x 22.9 cm.). The rats were fed Purina rat chow pellets ad lib., and had free access to water.

After about six weeks the Ss were tested and sacrificed the

same day. They were perfused with saline and 10% Formaline. The brains were stored in 10% Formaline, and were later embeded in paraffin. Before embedding, the brains were photographed and Lashley type diagrams were made of the lesioned groups. The wax blocks were then sectioned (15u) and stained using the Kluver-Barerra (1953) cell and fiber stain. From the slides, the extent of lateral geniculate degeneration was established.

Following recuperation, the rats were tested in a double blind study, on each of the tasks described below. Before testing, all Ss were injected intraperitoneally with either d-amphetamine sulfate (USP) (0.5 mg./kg.), or with saline and placed in a holding cage. The 0.5 mg./kg. dose had been established by an earlier pre-test on animals of similar age and strain at a level that increased motor activity yet produced no deficit in balance. The increased activity was measured by the number of line crossings of a grid painted on the floor of an open field and the balance performance was measured on a 5 cm. wide board.

After 15 min. the animals were removed from the holding cage and tested on the various tasks in the order in which they were discussed below. The entire testing session lasted 20 min. to one hour for each animal.

Visual cliff. Each S was placed on the centerboard of the visual cliff and the side to which the rat first descended with all four paws was recorded. The experiment had no time limit, and terminated only with the rats descent.

Optokinetic effect. The rat was placed into the jar, and left

there for 3 min. or until it responded. The criterion for response was head movement following the contour of seperation between the moving stripes in front of the animal (Smith, 1940; Smith and Bojar, 1938; Smith and Kappauf, 1940).

T-maze experiment. The T-maze was placed over the visual cliff, and the animal, with electrodes attached to its tail, was placed in the stem of the maze. If after 30 sec. the animal did not respond by descending to either arm, it was shocked with 0.5 ma. current for 1 sec. and removed from the maze and held by E for 30 sec. It was then replaced in the start box and run again. This procedure was continued until the animal descended. A correct response was considered to be a descent with all four paws onto the shallow side of the cliff. If the animal responded by entering the deep side, it was shocked at the same level in 1 sec. bursts until it entered the shallow side of the maze which was randomly altered from the left to the right.

The criterion for learning was eight out of ten consecutive shallow descents. If the animal did not reach criterion after eighty trials, the procedure was discontinued.

Visual placing. The rat was picked up by the base of its tail and held approximately 45 cm. above the apparatus. The E then lowered the animal down to the level of the fork. The S head was kept about 8 cm. away from the fork so that the animal could not feel the fork with it's whiskers. The S was given a maximum of five trials to respond, or until it started to climb on it's own tail. If after this time the S did not visually place, then a negative response was recorded.

RESULTS

Anatomical Results

Two rats did not survive surgery. Of those that did survive, the maximum and minimum extents of cortical lesioning are demonstrated by Lashley diagrams (Figure 5 shows the L.D. group: Figure 6 shows the L.Nd. group). It should be noted that some animals had lesions extending into anterior portions of the cortex. The Kluver-Barrera myelin and nissle body stain was used to determine the amount of lateral geniculate degeneration in the lesioned animals. Marked gliosis and degeneration was found in all the operated animals while no sign of deterioration could be seen in the sham operated animals.

Behavioural Results

The optokinetic response did not seem to be affected by any of the treatments (see Table 1). The percentages of animals in each group showing the response were 90% for the S.Nd. rats, (n-10), 100% for the S.D. rats, (n=10), 95% for the L.Nd. group, (n=19), and 89% for the L.D. group, (n=19). Since the lesioned animals which did not receive the drug exhibited optokinesis about as frequently as the S.Nd. controls, it seems certain that optokinesis cannot be strongly affected by the striate lesion. Obviously then, the original plan to attempt to reverse a defect in optokinesis by administration of amphetamine was not scientifically meaningful because there was no effect to reverse.

The three other tasks, however, were severely disrupted by the striate lesion, so that it was meaningful to attempt to determine the effects of amphetamine on the performance of the lesioned animals.

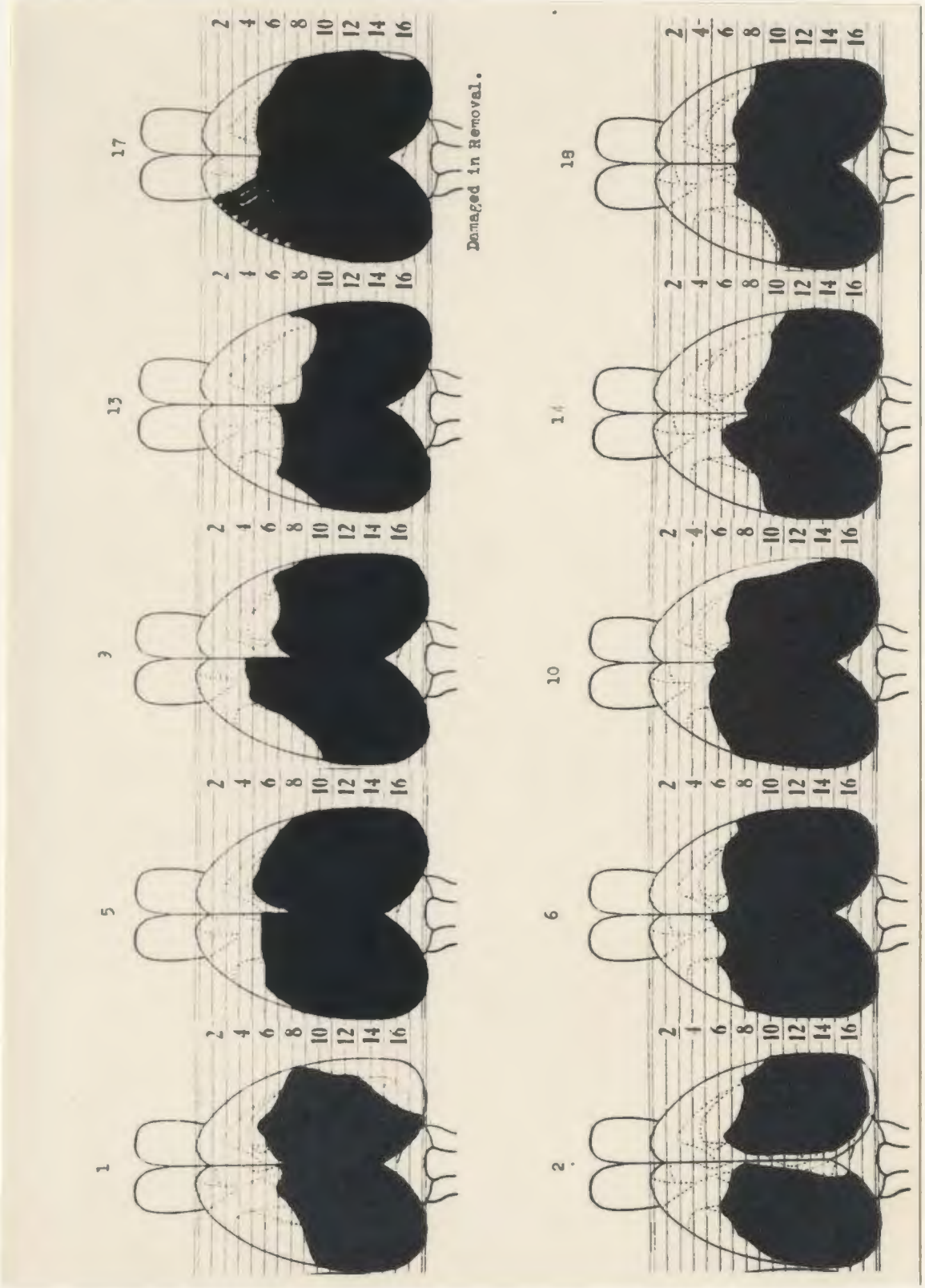

Fig. 5 Schematic representations of the lesion sizes of the L.D. group through Lashley-type diagrams.

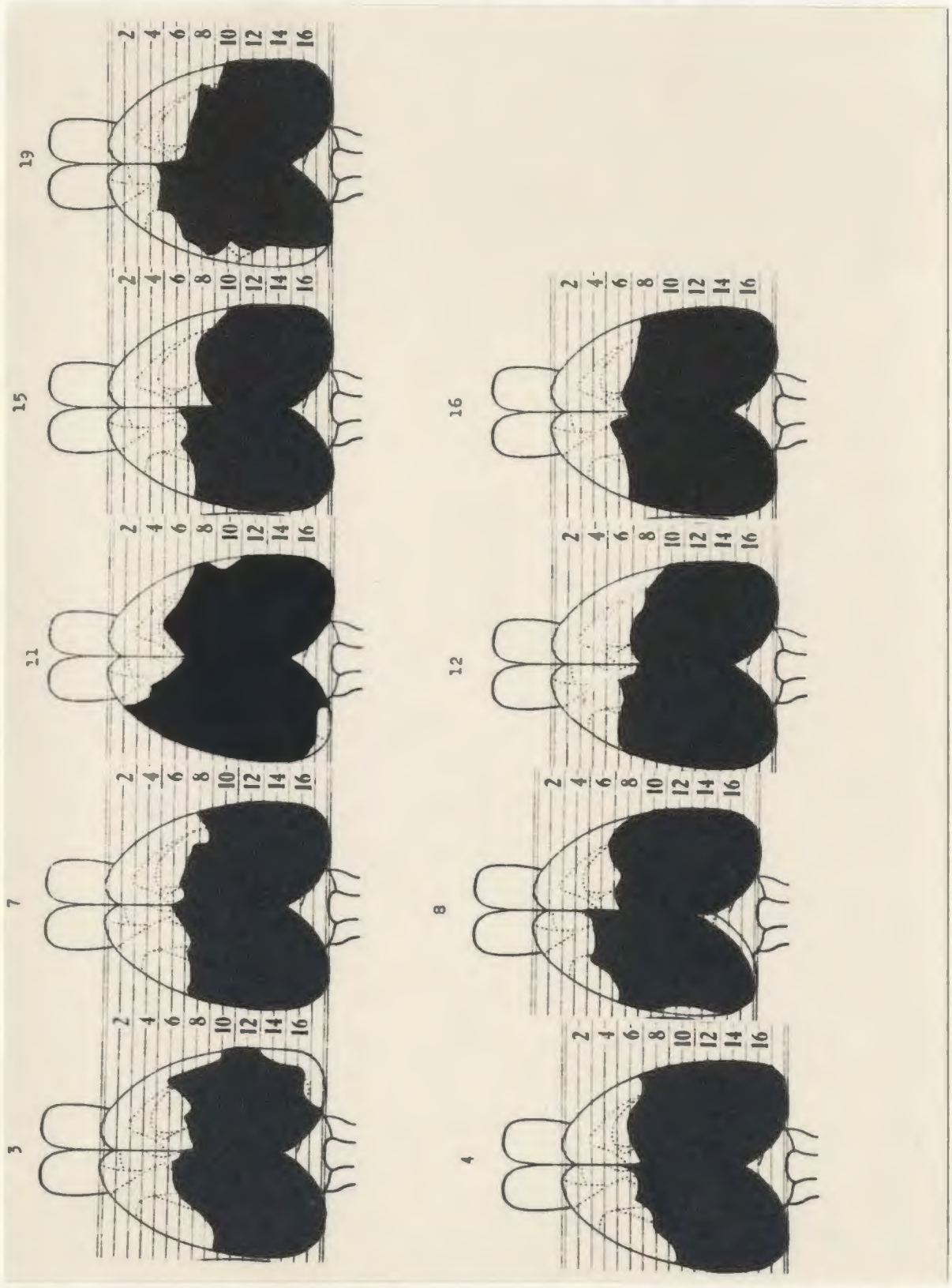

Fig. 5 (cont'd.)

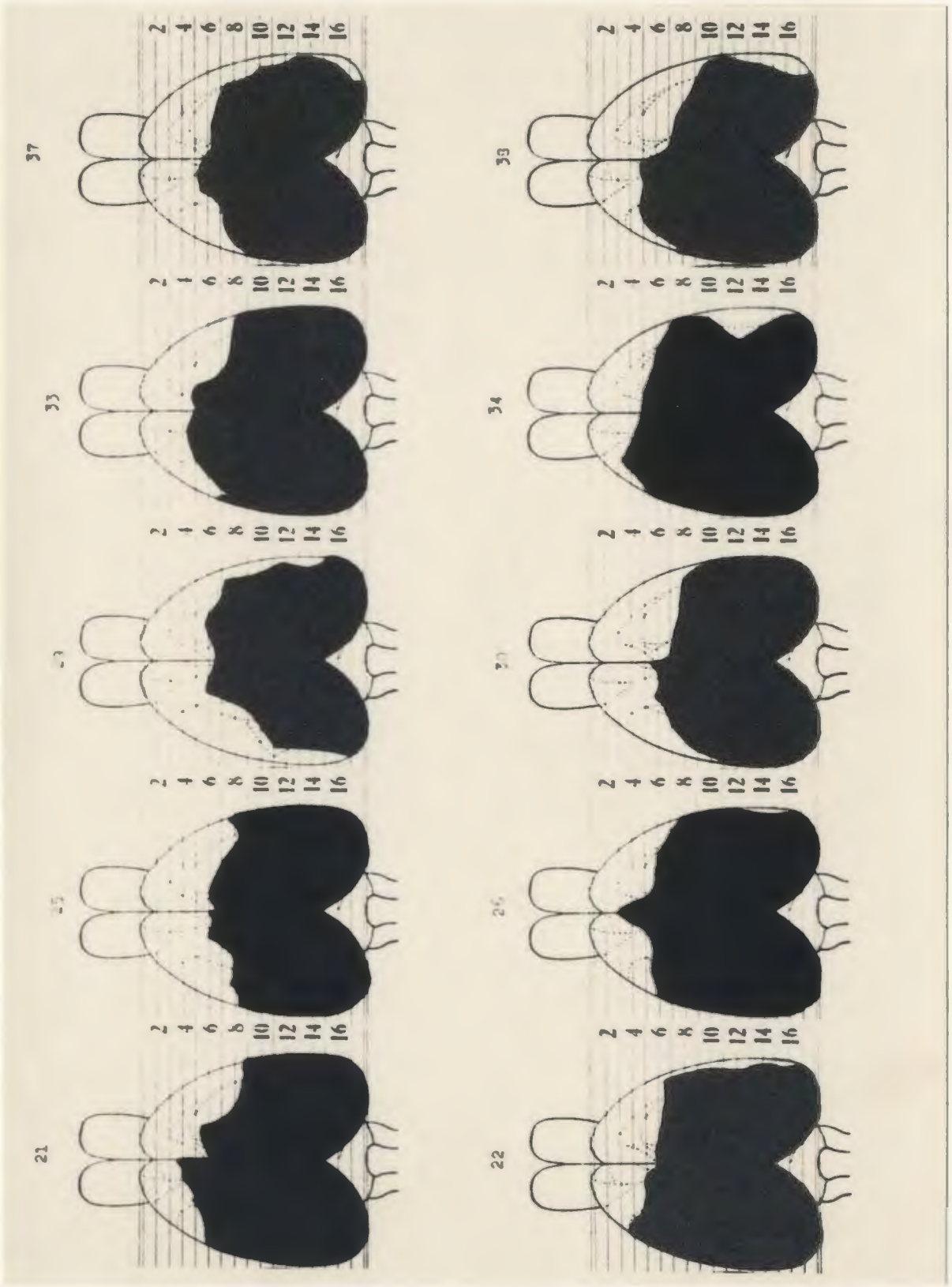

Fig. 6 Schematic representations of the lesion sizes of the L.Nd. group through Lashley-type diagrams.

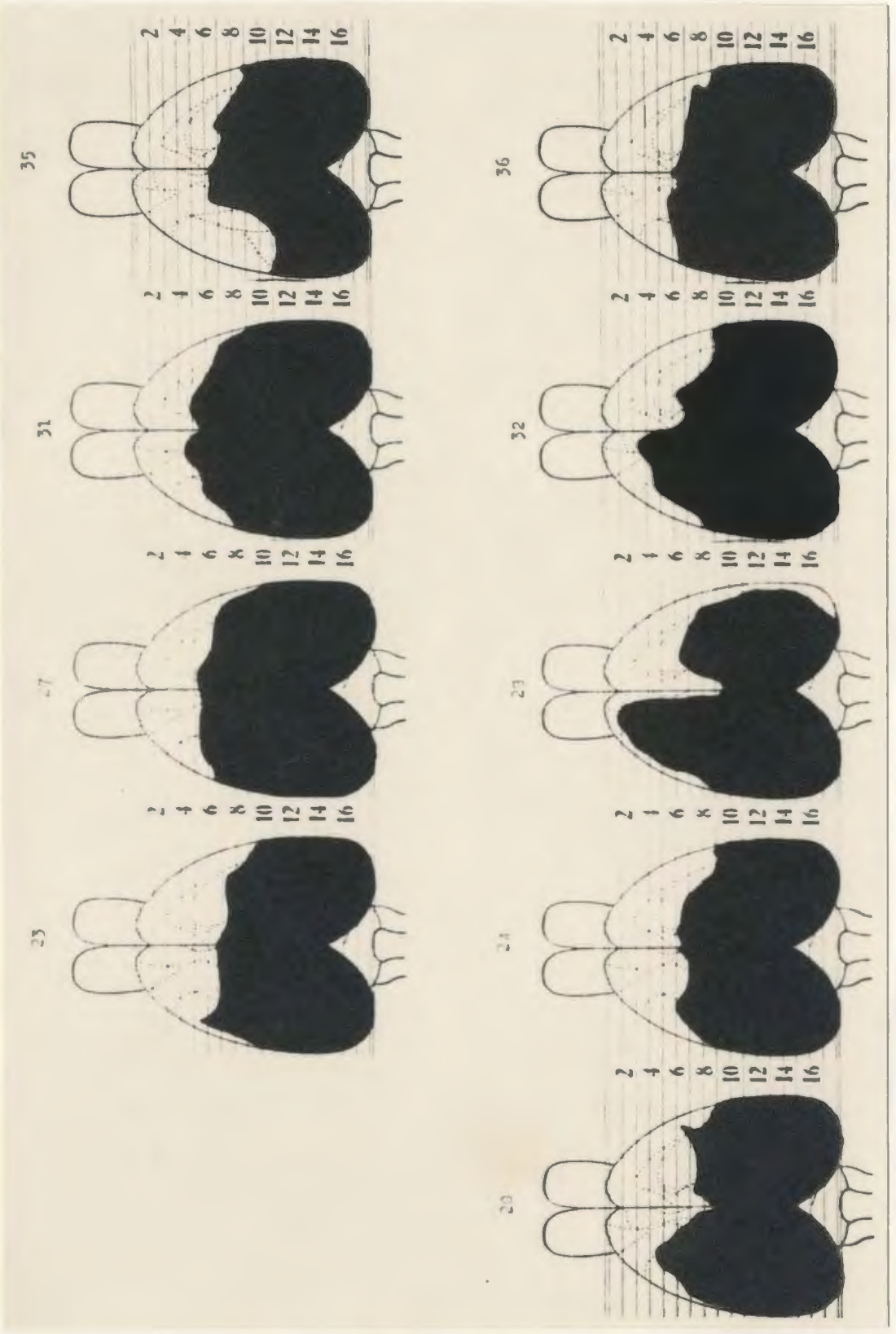

Fig. 6 (cont'd.)

Table 1 shows the results for each type of performance for each of the four groups of rats.

The visual cliff performance is reported as the number of animals that descended to the deep side divided by the number in the group and the visual placing results are expressed as the number of animals that showed no visual placing divided by the number in the group. On the visual cliff, all animals descended either to the deep or the shallow side. The T-maze data were expressed as the mean number of trials it took the animals to reach learning criterion. One rat in each lesion group did not reach criterion, and they were given a score of 80. Thus, the higher the score in Table 1, the poorer the visual performance.

Each row in Table 2 tests a specific a priori hypothesis using x^2. For this reason, the comparisons between S.D. vs L.D. and S.D. vs L.Nd. had not been made, as they were scientifically not meaningful to this study. The first row tests the hypothesis that striate lesions disrupt visual performance. This hypothesis was confirmed for each of the visual tasks shown. The second row tests the hypothesis that administration of amphetamine tends to reverse the disruption in performance produced by the lesion. This hypothesis also was confirmed in each of the tests. The third row tests the hypothesis that the improvement in visual performance in lesioned rats produced by amphetamine does not raise them to the level of non-lesioned undrugged rats. This hypothesis was not confirmed for the visual placing and the visual cliff (first choice) tasks, but was confirmed in the reinforced visual cliff performance, and thus

TABLE 1

Mean results on each task for each group of rats

		$\bar{X}$ Proportion Deficit Performance			$\bar{X}$ Trials to Criterion
Groups	N	Visual Cliff	Visual Pl.	Optokinesis	T-Maze
Sham Drug	10	.20	.10	.00	14.0
Sham No Drug	10	.10	.10	.10	12.0
Lesion Drug	19	.21	.11	.14	16.0
Lesion No Drug	19	.53	.84	.05	25.0

TABLE 2

Results of statistical tests between specific group of rats

Groups	$\bar{X}$ Proportion Deficit Performance		$\bar{X}$ Trials to Criterion
	Visual Cliff	Visual Pl.	T-Maze
Sham No Drug	.10	.10	12.0
Lesion No Drug	.53	.84	25.0
χ^2	3.36	11.88	v 22.0
p<	.05	.001	p< .001
Lesion No Drug	.53	.84	25.0
Lesion Drug	.21	.11	16.0
χ^2	2.83	17.84	v 85.0
p<	.05	.001	p< .01
Sham No Drug	.10	.10	12.0
Lesion Drug	.21	.11	16.0
χ^2	.05	.33	v 40.0
p<	n.s.	n.s.	p< .01
Sham No Drug	.10	.10	12.0
Sham Drug	.20	.10	14.0
χ^2	.004	.55	v 56.0
p<	n.s.	n.s.	p< n.s.

no certain conclusion can be reached. The last row tests the hypothesis that the drug improves visual performance in unlesioned rats. This hypothesis also was not confirmed. Thus, it seems that amphetamine tends to reverse disruption in visual performance produced by striate lesions, although it does not markedly affect visual performance in unlesioned rats.

DISCUSSION

These results confirm the earlier findings that amphetamine can reverse deficits in visual placing produced by striate cortex lesions. They also show that when deficits do occur on the visual cliff task, the drug also tends to reverse these effects.

Lashley's studies of the involvement of the cerebral cortex in vision revealed that destruction of the lateral border of the striate area eliminated pattern and depth vision (Lashley and Frank, 1934; Lashley, 1939). A generalization of this magnitude is misleading, however, since 18 out of 19 of the L.Nd. rats did learn the visual cliff task with appropriate reinforcement procedures.

A qualification is therefore necessary. Sharpless and Jasper (1956) proposed a neural model of the auditory system in which the cortex is considered to be involved in the combination and integration of the separate features of the pattern stimulus. Nevertheless, particular features of the pattern may be discriminated at the thalamic and collicular levels. Thus, cats lesioned in the auditory cortex could no longer habituate to a pattern of tones, but did habituate to the specific

frequencies of the tones used in the pattern. These findings are corroborated by Diamond (1967) using lesioned tree shrews.

Evidence that this model can be applied to the visual system has been supplied by Diamond and Hall (1969) when they ablated the straite cortex of the tree shrew and found a total incapacity of the animals to abstract figures imbeded in a larger pattern, but found no loss in discrimination of simple, nonembeded patterns. From these results, they concluded that the geniculo-striate system is not necessary for lower levels of integration. In the same view, Ganz (1971) distinguished two types of visual tasks: simple brightness and contour orientation which demand only that the animal respond in terms of reaction to the majority of his feature detectors, and which are not lost after striate cortex ablations (Bauer and Cooper, 1964; Lashley, 1935) and discrimination of complex pattern by combining the stimulus features into a complex whole, such as depth.

These theories describe two systems. One involves inputs from the feature detectors (eg. brightness, motion, etc.) in centers other than the striate cortex to cells within the cortex which respond to multiple inputs. The outputs of these "complex and hypercomplex" cells (Hubel and Wiesel, 1962) correspond to the total pattern (see Figure 8, system A). This description corresponds to the interpretation of the single cell micro-electrode findings of Hubel and Wiesel (1961; 1962), Hubel (1960) and Bishop, Burke and Davis (1959). However, the animals in this experiment were still able to perform the visual tasks after total striate ablation. To explain this finding, it would seem necessary to hypothesise the existence of connections between lower levels and the

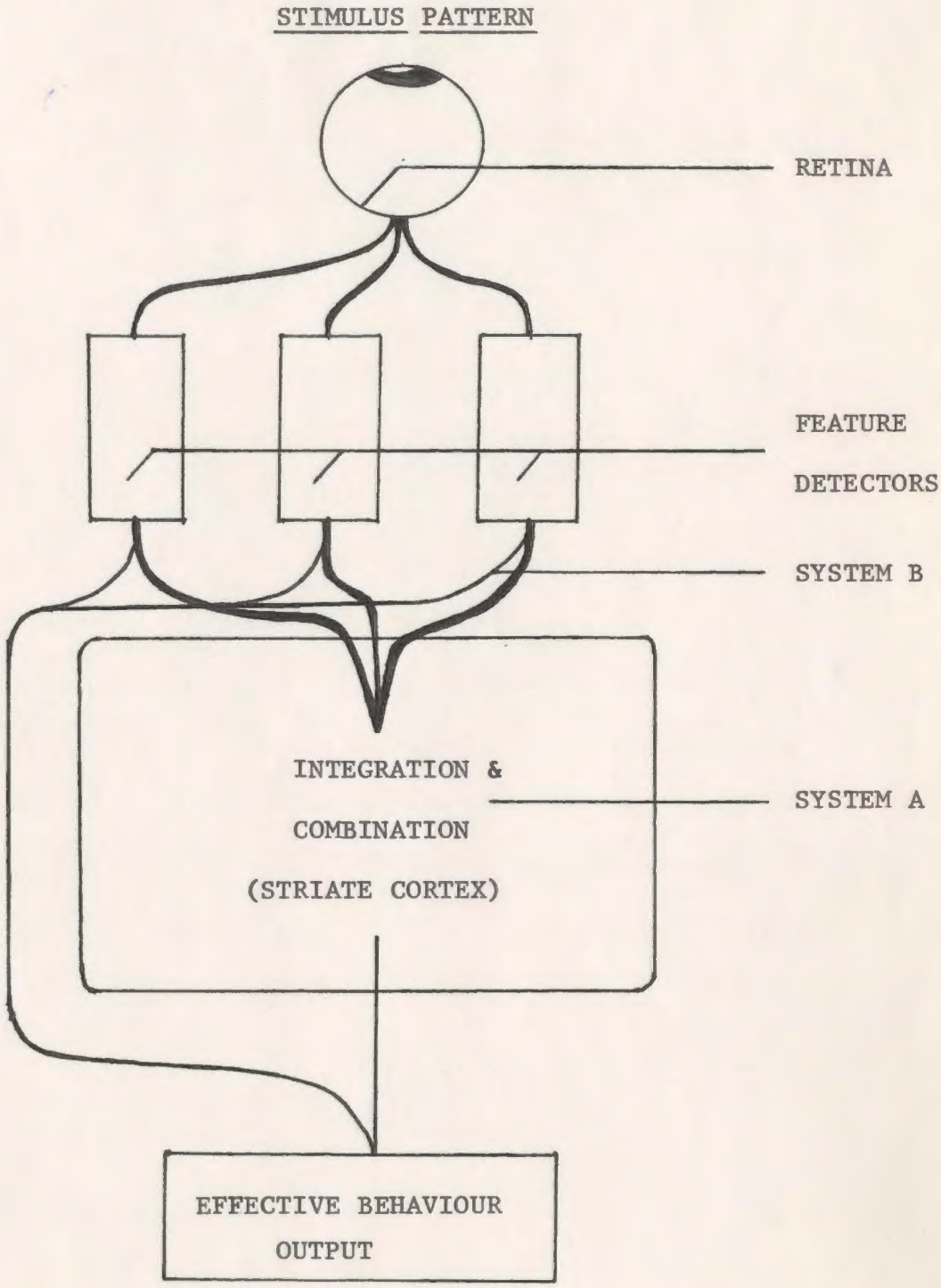

Fig. 8 Diagram to illustrate the proposed model of feature and pattern discrimination

response system which by-pass the striate cortex (see Figure 8, system B). The difference between the two systems is that when the animal uses, or is forced to rely exclusively on system B, it responds solely to features, and not to total pattern. This would explain why striate lesioned rats can discriminate brightness (Bauer and Cooper, 1964; Jonason et al., 1970) and display optokinesis (Smith, 1938; 1940), but cannot learn complex patterns composed of these features (Bauer and Hughes, 1970; Bland and Cooper, 1970; etc.). A non-lesioned animal, however, would be expected to utilise both systems A and B and thus be able to discriminate features and total pattern, if necessary, independently.

The role of amphetamine in restoring the visual behaviors that were lost after striate cortex ablations is unknown, but certainly cannot lie in an ability to restore the combinatorial integrative mechanism (System A), of the striate cortex. Therefore, it can only serve to make better use of the remaining mechanisms. It is suggested, therefore, that the remaining portion of the nervous system, including the function of system B is enhanced by amphetamine. An experiment by Jonason et al. (1970) lends more support for this interpretation. These workers showed improved speed of acquisition a feature discrimination (brightness) in lesioned animals with amphetamine. However, no effect upon pattern discrimination was evident by the drug.

Conclusion

The following conclusions can be made from this study:

1) Total ablation of the rat's striate cortex does not affect the optokinetic response, but does produce a deficit in visual placing and performance on the visual cliff tasks.

2) Injecting d-amphetamine into striate lesioned rats reverses these deficits.

3) Training using electric shock procedure improves performance on the visual cliff in lesioned animals.

These findings are interpreted as suggesting that the lesioned animals may still be able to perform a pattern and/or depth discrimination task, but the response is determined by discrimination of particular features rather than the entire pattern.

It is also suggested that neural pathways, other than through the geniculo-striate system, allow the animal to discriminate features of a pattern, but not the whole pattern itself.

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