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INFEROTEMPORAL VERSUS COMBINED PULVINAR-PRESTRIATE LESIONS IN THE RHESUS MONKEY: EFFECTS ON COLOR, OBJECT AND PATTERN DISCRIMINATION

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Abstract—Monkeys with inferotemporal lesions were compared to monkeys with combined lesions of the pulvinar and prestriate cortex on discrimination problems and on tests of sensory function. Inferotemporal lesions severely impaired discrimination of colors, objects, and patterns, but did not produce any sensory loss. By contrast, combined pulvinar-prestriate lesions impaired only discrimination of patterns, an impairment associated with sensory deficits and with degeneration of the lateral geniculate nucleus. Thus, inferotemporal lesions affected visual discriminations *per se*, whereas combined pulvinar-prestriate lesions affected discriminations related to specific dimensions of visual stimuli. These qualitatively different effects of inferotemporal and combined pulvinar-prestriate lesions are discussed in terms of current models of central processing of visual information.

INTRODUCTION

THE RESULTS of numerous ablation studies have firmly established that inferotemporal cortex is crucial for normal visual discrimination learning in monkeys (for reviews, see [1–10]). However, the neural pathways through which inferotemporal cortex interacts with primary visual areas is an issue which remains in dispute. While the inferotemporal area does not receive a direct projection from striate cortex, neuroanatomical analysis has demonstrated that these two areas are connected indirectly through several synapses in prestriate cortex [11–18]. Thus, one possibility is that visual information is "serially processed" as it is relayed from striate to prestriate to inferotemporal cortex. The importance of this cortico-cortical pathway to inferotemporal cortex has been emphasized in reports of both behavioral [4, 5, 19] and electrophysiological [20–24] studies.

If prestriate cortex directly relayed information from striate to inferotemporal cortex, then damaging prestriate cortex should yield effects at least as severe as damaging inferotemporal cortex. Yet, extensive removal of prestriate tissue has repeatedly failed to produce an impairment on visual tasks ([5, 25–30]; for reviews, see [4, 31]). MISHKIN [5] reported that total prestriate resections do disrupt pattern discrimination performance, and has argued that any spared remnant of prestriate cortex can mediate sparing of visual function. However, PRIBRAM, SPINELLI and REITZ [32] did not find a pattern discrimination impairment following radical prestriate lesions comparable in extent to those of Mishkin. These

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negative findings thus create a major discrepancy for a serial model of visual processing based upon a striate–prestriate-temporal pathway.

A second potentially important pathway to inferotemporal cortex is a subcortical route from the superior colliculus and pretectal region through the pulvinar [5, 33–36]. However, the interruption of tecto-thalamic inputs to inferotemporal cortex by pulvinar lesions does not disrupt pattern discrimination performance [5, 37]. GROSS [2, 3] has therefore suggested that inferotemporal cortex may integrate visual input, combining the signals from this tecto-thalamic pathway with those of the striate–prestriate pathway. According to such an "input-integration model", deficits similar to those obtained after inferotemporal lesions might only be observed if both afferent pathways were severed.

An attempt to deafferent inferotemporal cortex from its visual input by combined lesions of the pulvinar and prestriate cortex was undertaken by CHOW [38]. Of the two monkeys sustaining lesions, one showed reduced savings but no impairment in the retention of two pattern discriminations, while the other was not impaired at all. Although these results appear to provide strong evidence against an "input-integration model" of inferotemporal cortex, the absence of a behavioral deficit in the Chow study may have been due to incomplete surgery; in both monkeys the pulvinar and prestriate lesions were subtotal.

The purpose of the present study was to examine the effects of combined radical lesions of the pulvinar and prestriate cortex. If these combined lesions do not produce deficits comparable to those produced by inferotemporal lesions, then the neural pathways critical for inferotemporal function in vision are unlikely to involve direct transmission of visual information via afferents. Instead, such a finding would support the accumulating ablation [32, 39–43] and electrophysiological [44–47] data indicating the importance of efferent projections from inferotemporal cortex [48]. This outcome would therefore provide additional evidence for a "parallel processing model" in which inferotemperal cortex modulates visual activity via these cortico-fugal pathways [7–9, 49].

METHOD

Subjects

Sixteen young adult rhesus monkeys (*Macaca mulatta*) of both sexes, ranging in weight from 4·1 to 6·2 kg, served as subjects. Four of these monkeys received bilateral pulvinar lesions followed by additional bilateral prestriate lesions (Group PPS), eight received bilateral inferotemporal lesions (Group IT), and four were unoperated control subjects (Group N). Since not all monkeys participated in all experiments, the specific monkeys who served as subjects are cited under the description of the particular task.

Brain surgery and histology

Preparation for aseptic surgery consisted of Ketamine tranquilization (15 mg/kg i.m.) followed by sodium pentobarbital anesthesia (30 mg/kg i.v.) delivered via saphenous catheter. The catheter was also used for a 5% dextrose-in-saline drip infusion.

Pulvinar lesions were produced by passing radio frequency current through electrodes stereotaxically lowered through small burr holes in the skull. For seven stereotaxic sites on each side of the brain, determined by the Olszewski Atlas [50], 200 mA of current were passed for 30 sec. The electrodes were 1 mm dia stainless steel wire insulated except for 2 mm at the tip; an 18-gauge needle placed in the temporalis muscle of the skull served as the ground.

Both prestriate and inferotemporal lesions were produced by subpial aspiration using a 19-gauge Pribram sucker. For the inferotemporal lesions, the cortex was exposed by openings rongeured in the skull; for the prestriate lesions, bone flaps were made. Bleeding was controlled by means of cottonoid strips and a minimum of electrocauterization. Wounds were closed in anatomical layers with silk sutures, and long-acting bicillin (300,000 U i.m.) was routinely administered.

The inferotemporal lesion was intended to correspond to area TE of VON BONIN and BAILEY [51]: from a point several millimeters anterior to the ascending limb of the inferior occipital sulcus, extending rostrally almost to the temporal pole; dorsally, to include the depth of the inferior bank of the superior temporal sulcus; and ventrally, to the lateral bank of the occipito-temporal sulcus.

The prestriate lesion was intended to remove the entire projection area of striate cortex: caudally, both banks of the lunate sulcus, including the annectant gyrus, and cortex extending rostrally from the lunate over the entire preoccipital gyrus; dorsomedially, the cuneus and precuneus completely; ventrally, both banks of the inferior occipital sulcus; and ventromedially, cortex upward to the calcarine fissure.

Following completion of behavioral testing, the monkeys were perfused intracardially under deep barbiturate anesthesia with saline followed by 10% formalin, and the brains were blocked stereotaxically in the coronal plane. They were then hardened in formalin and 30% sucrose–formalin and, after they were embedded in gelatin–albumin and frozen, 50 μm coronal sections were made. Sections were mounted and stained with cresyl violet for microscopic analysis of the lesions. Cortical lesions were reconstructed from enlarged tracings, using serial sections 1 mm apart. Pulvinar lesions and thalamic degeneration were plotted 0·5 mm apart.

Reconstructions of lateral and ventral views of the brain and representative coronal sections for monkeys sustaining inferotemporal lesions are shown in Fig. 1; retrograde degeneration in the dorsal lateral geniculate nucleus, due to invasion of the geniculo-striate pathway, and in the pulvinar appears in Fig. 2. Reconstructions of lateral and medial views of the brain and representative coronal sections for monkeys sustaining prestriate lesions are shown in Fig. 3; retrograde degeneration in the dorsal lateral geniculate nucleus, due

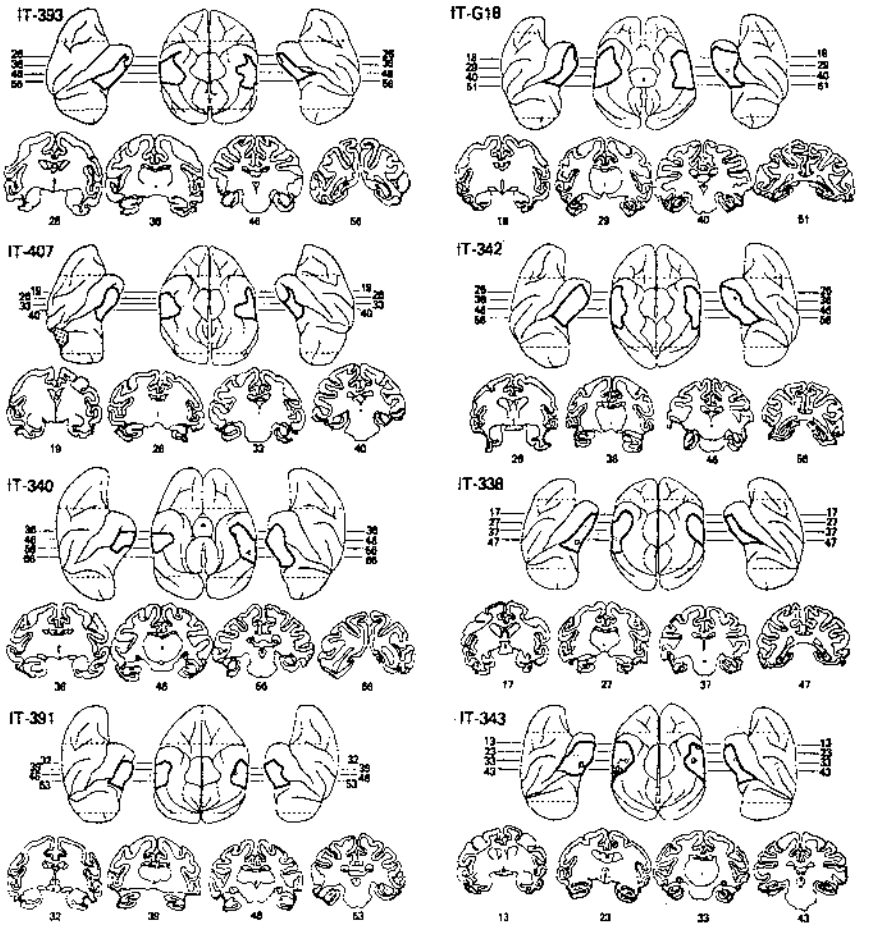

FIG. 1. Reconstructions of lateral and ventral views of the brain with representative coronal sections for monkeys sustaining inferotemporal lesions. On the reconstructions the lesions are shown in black. On the coronal sections the borders of the lesions are indicated by thickened black lines. The dotted area on the right lateral view of IT-407 represents cortical damage, discovered upon autopsy, which was not produced experimentally.

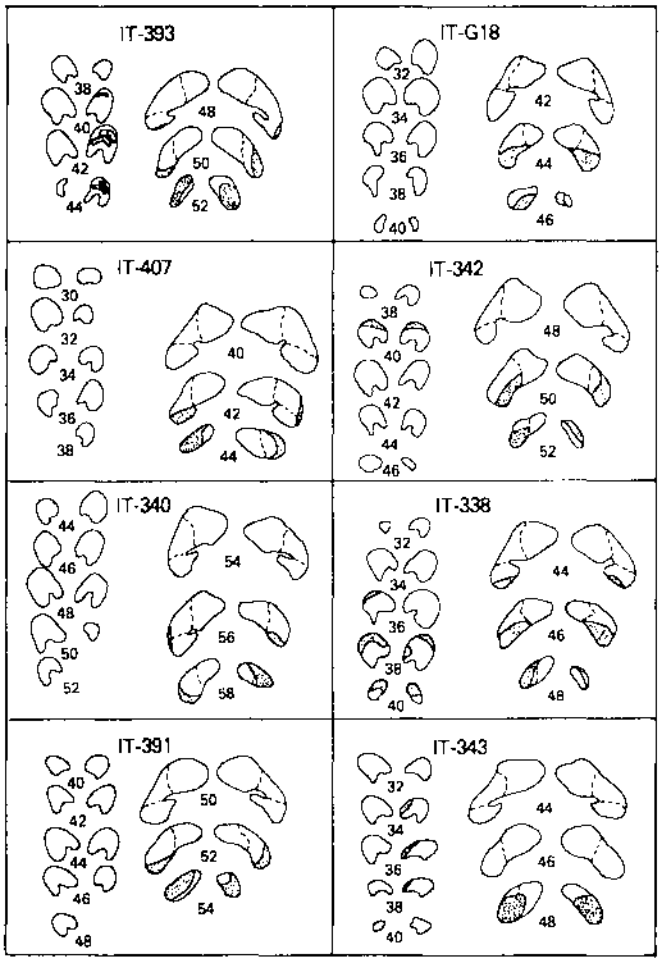

FIG. 2. Tracings of representative coronal sections through the dorsal lateral geniculate nucleus and the pulvinar for monkeys sustaining inferotemporal lesions. Total retrograde degeneration, indicated by complete cell loss and dense gliosis, is shown in stipple.

to invasion of the geniculo-striate pathway, appears in Fig. 4. Tracings of representative coronal sections through the pulvinar lesions are shown in Fig. 5.

The cortical lesions were essentially as intended. There was little variability in the size or locus of any of the four inferotemporal lesions; removals were almost completely confined to the cortex, and damage to the underlying white matter was minimal. The prestriate lesions were massive. There was little remaining prestriate cortex in any of the brains and, in fact, for PPS-G3 damage included a considerable amount of striate cortex as well. Degeneration in the lateral geniculate nucleus for monkeys sustaining prestriate lesions was moderate (PPS-G22) to severe (PPS-G3).

The pulvinar lesions were subtotal, in all cases sparing the anterior portion of the nucleus. In two of the four monkeys (PPS-G17 and PPS-G19) the part of the pulvinar which projects to inferotemporal cortex, the posterior portion of the lateral nucleus [34, 35], was completely destroyed. All four monkeys showed extensive degeneration in the inferior nucleus of the pulvinar as well, most likely a retrograde effect from the subsequent prestriate lesions they received. In addition to pulvinar damage, passage of the electrode consistently produced minimal bilateral damage to the fornix and corpus callosum. There was no detectable damage to either the superior colliculus or pretectum.

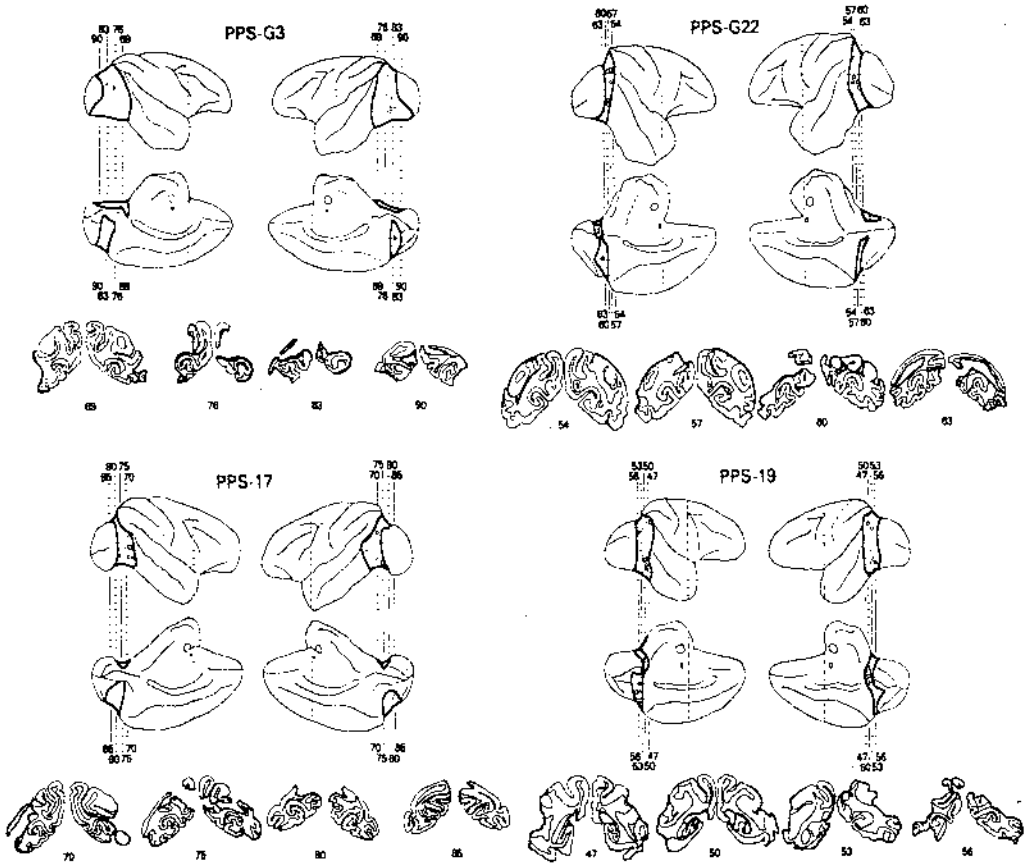

FIG. 3. Reconstructions of lateral and medial views of the brain with representative coronal sections for monkeys sustaining prestriate lesions. On the reconstructions the lesions are shown in black. On the coronal sections the borders of the lesions are indicated by thickened black lines. The grey areas on the medial views of PPS-G22 and PPS-G19 represent cortical damage within the depth of the medial calcarine fissure.

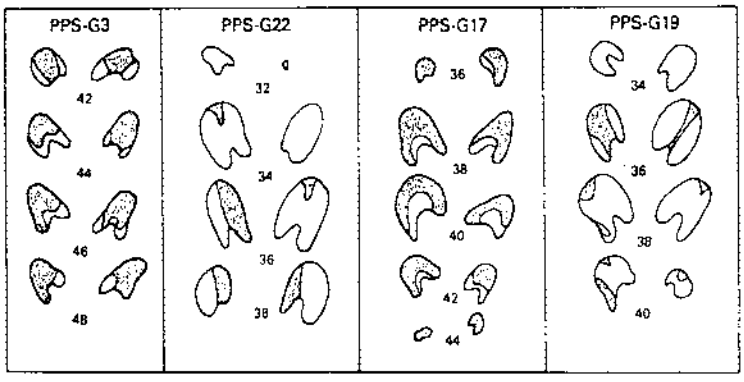

FIG. 4. Tracings of representative coronal sections through the dorsal lateral geniculate nucleus for monkeys sustaining prestriate lesions. Total retrograde degeneration, indicated by complete cell loss and dense gliosis, is shown in stipple.

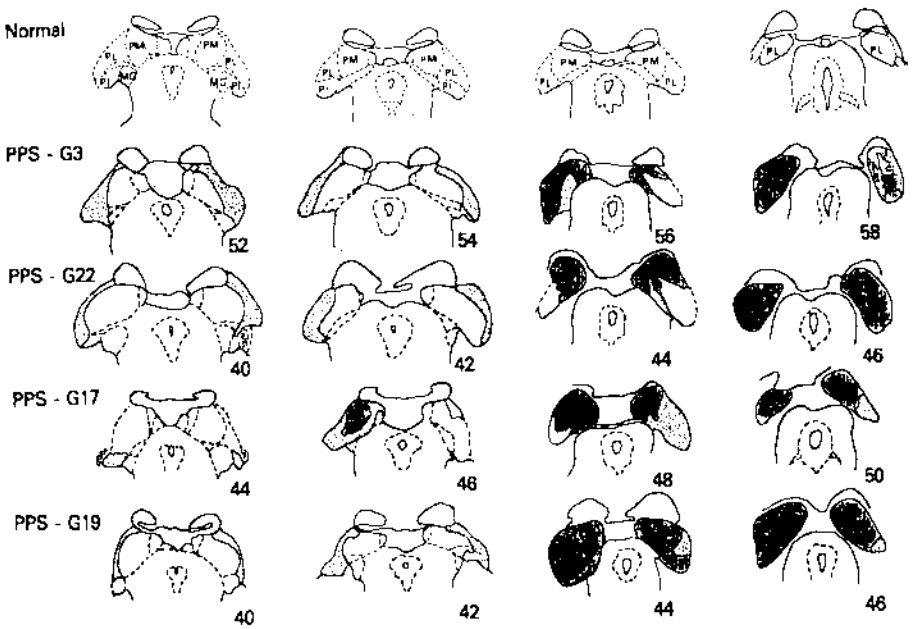

FIG. 5. Tracings of representative coronal sections through the pulvinar lesions. The lesions are shown in black; areas of complete cell loss and dense gliosis are indicated by stipple. Labelling of thalamic nuclei on coronal sections from a normal brain: PI = pulvinar inferior, PL = pulvinar lateralis, PM = pulvinar medialis, MG = medial geniculate.

Apparatus and behavioral procedures

A series of eight tasks was presented to the subjects. Six of the tasks—Color Discrimination Retention, four Pattern Discrimination Acquisitions, Object Discrimination Learning—were included as measures of acquisition or retention of a visual discrimination. The other two tasks—Patterned Strings, Visual Acuity—were included as potential measures of sensory defects. The eight tasks are described below in the order in which they were given.

Experiment 1. *Color discrimination: postoperative retention*

Eight monkeys (Group PPS and four from Group IT) were preoperatively trained to pull in with strings [52] one of two trolleys viewed down a 2·44 m alley, described in detail elsewhere [53]. Mounted upon the front of each trolley was a stimulus plaque, which when pushed over revealed a food well. Following one week of adaptation to the pull-in procedure, the monkeys were trained on the color discrimination. The stimuli were red (pos) and green (neg) 8 cm square metal plaques, presented at distances of 50, 100, or 200 cm from the animal. Discrimination training consisted of 30 noncorrection trials/day, 5 days a week, until a criterion of 90% correct was met on two consecutive days. Each 30-trial session was divided into five 6-trial blocks, such that within each block the three distances randomly occurred twice; on any given trial both stimuli were always at the same distance. The position of the stimuli (left or right) was determined by a pseudo-random sequence [54].

Just prior to surgery the monkeys were tested for retention of the problem. This provided preoperative retention scores for assessing possible postoperative deficits. Four monkeys (Group PPS) then received pulvinar lesions, were tested for retention of the problem, received additional prestriate lesions, and were retested. The remaining four monkeys (Group IT) received one-stage inferotemporal lesions, and were similarly tested for retention. All postoperative procedures were identical to those employed preoperatively.

Experiment 2. *Patterned strings*

Group N, Group PPS, and four monkeys from Group IT participated in this experiment. Monkeys from Group PPS were tested twice, once after receiving pulvinar lesions and again after receiving prestriate lesions; monkeys from Group IT were tested once postoperatively. To control for possible training effects resulting from testing Group PPS twice, monkeys form Group N were also given a second test three months after their first test. The data from Group N indicated no change in performance from the first to second test on any of the patterned string arrangements.

The stimuli consisted of patterns formed from two white strings, 1·59 mm in dia, arranged on a matte black plywood board. The board (0·61 × 0·46 m) was placed in front of and level with the floor of the monkey's cage (0·46 × 0·66 × 0·51 m). The two strings were attached at their near ends 12·70 cm apart and 7·62 cm from the monkey's cage. The far ends of the strings were tied to 0·64 cm white plastic receptacles that served to hold raisin rewards. Each of the eight patterned string arrangments was 45·72 cm in length, the width varying for different patterns. The far ends of the strings were either 12·70 or 3·81 cm apart, depending on the pattern. Once a pattern had been formed, the experimenter rolled the horizontal testing board toward the monkey, who could then reach through the vertical bars of his cage, spaced 4·44 cm apart, to reach the string.

All monkeys were pretrained to reach through the bars and pull strings from their attached ends to obtain raisin rewards located at their far ends. Following pretraining, the monkeys were tested for 32 trials/day for 15 days. Each day's test consisted of four presentations of eight patterns, such that each pattern randomly occurred once every eight trials. Asymmetrical patterns were tested as two sets of mirror images presented twice each. The reward position was assigned to ensure an equal occurrence of rewards on the right and left of each pattern. A noncorrection procedure was used. The configuration of the eight patterns is shown in Fig. 7. They were selected from those used in prior studies [55, 56].

Experiment 3. *Pattern discriminations: postoperative acquisition*

Group N, Group PPS, and four monkeys from Group IT were trained postoperatively on a series of four two-choice pattern discrimination problems, selected for varying degrees of difficulty. The four pairs of patterns in the order in which they were learned are shown in Fig. 8; in all problems they appeared as white patterns against a dark field.

The first pattern discrimination was trained in a computer-controlled (PDP-8) automated apparatus that allowed discrete trial analysis (DADTA), described in detail elsewhere [57]. Briefly, the apparatus consisted of a 0·51 × 0·51 m square panel in which there was embedded a 4 × 4 regular array of clear round plastic push-panels, 2·54 cm in dia. Microswitches mounted behind each of the response panels signalled the presses to the computer; the stimuli were rear-projected through these panels by IEE digital display projectors. The trial sequence, intertrial interval, and rewards were all controlled by a computer program, and the responses recorded by teletype. A correct response caused a banana pellet food reward to be delivered by a mechanical feeder to a single food well centered just below the array of response panels.

During training the monkey faced the response panel from a containing cage identical to the one described in Experiment 2. The two stimuli were only presented on 2 of the 16 possible response panels—the middle 2 panels of the second row from the top. The position of the stimuli on these panels was determined by a pseudo-random sequence [54]. Fifty noncorrection discrimination training trials were run each day, 5 days a week, until a criterion of 90% correct was met on two consecutive days. During training, except for a constant intertrial interval of 8 sec, the monkey paced the trials as there was no time limit imposed for responding.

The subsequent three pattern discrimination problems were trained in an automated apparatus controlled by a digital logic system, which also tabulated the data and delivered banana pellet food rewards for correct responses. During training the monkey faced a response panel consisting of three clear round plastic push-panels, 2·54 cm in dia, spaced 7·62 cm apart. The stimuli were rear-projected through the two side panels by IEE digitial display projectors; located beneath the center panel was the food well. The monkey sat in a metal containing cage (0·56 × 0·46 × 0·51 m), the front side of which was a plexiglass window. The monkey responded by reaching through holes in the plexiglass window and pressing one of the two lit panels; the stimulus position on the panels was automatically programmed in a pseudo-random sequence [54]. One hundred noncorrection trials were run each day, 5 days a week, until a criterion of 90% correct was met within a single 100-trial session. A constant 8 sec intertrial interval was maintained, but the monkey himself paced the trials as in the first pattern discrimination. Training was terminated on each pattern discrimination if criterion was not met by 5000 trials.

Experiment 4. *Object discrimination: postoperative acquisition*

Group N, Group PPS, and eight monkeys from Group IT were trained postoperatively on an object discrimination problem in a Wisconsin General Test Apparatus (WGTA). The testing cage was identical to the one used in the automated DADTA. The monkey's compartment was illuminated by a fluorescent light bulb which rendered opaque to the monkey a one-way vision door, separating the monkey from the experimenter and testing board. The opaque door was raised by the experimenter during testing, allowing the monkey free access to the testing board which contained two food wells. The testing board measured 0·71 × 0·20 m and the food wells were spaced 38·10 cm apart from center to center. The two stimulus objects were directly affixed to 7·62 cm square matte grey plywood plaques which covered the food wells during testing. The objects were a red and yellow plastic tug-boat, 10·16 cm in length, mounted diagonally on one plaque, and a yellow and white pair of soap bubble pipes, 10·16 cm in length, mounted criss-crossed together on the other plaque. Displacement of the plaque with the positive stimulus (tug-boat) was rewarded with a raisin.

Following a single day's shaping in the WGTA, training of the two-choice object discrimination was begun. Discrimination training consisted of 30 trials/day, 5 days a week, to a criterion of 90 correct out of 10 consecutive sets of 10 trials. An intertrial interval of 7 sec was maintained, and the position of the stimuli was varied pseudo-randomly according to a Gellermann sequence [54]. A modified correction procedure was used in which an incorrect trial was followed by another trial with the stimuli in the same position. However, on the fourth incorrect correction trial the monkey was permitted to self-correct, and the following trial continued the Gellermann sequence. Training was discontinued if criterion was not met by 1000 trials.

Experiment 5. *Visual acuity*

In this final task, an adaptation of one used previously [56], Group N, Group PPS, and five monkeys from Group IT were tested for impaired visual acuity. A matte black plywood testing board (0·61 × 0·46 m) was placed in front of the monkey's cage, level with its floor; the cage was identical to the one used in Experiment 2. The monkeys were first trained to pull in a coarse white string (1·59 mm dia) placed on the testing board to obtain a raisin attached to the far end of the string. In formal testing, black surgical threads of four sizes (USP 6-0, 0·08 mm dia; USP 5-0, 0·15 mm dia; USP 3-0, 0·25 mm dia; USP 1, 0·45 mm dia) were introduced in addition to the white string. One end of the string or thread was 7·62 cm from the monkey's cage, either in the center of the testing board or 10·16 cm to the right or left of center; the end with the raisin attached was always in the center 38·10 cm from the monkey's cage. A schedule was followed which balanced the order of trials with respect to size of thread and side of presentation. The test session consisted of 30 noncorrection trials, 2 trials for each size/side combination.

Statistical analysis

Mann–Whitney *U* Tests (two-tailed) were employed to test for significant differences between paired comparisons of the groups [58]. On retention tests, savings scores were computed according to the formula: (Trials to Learn − Trials to Relearn) / (Trials to Learn + Trials to Relearn).

RESULTS

Experiment 1. *Color discrimination*

All monkeys easily learned the red/green discrimination, requiring from 60 (IT-G18) to 330 (PPS-G17) trials. On the preoperative retention test all but one monkey (IT-G18) performed at criterion (0 trials), and this monkey required only one additional day of training (30 trials) to do so.

Following pulvinar surgery, all four monkeys in Group PPS were either at criterion, or required only one additional day of training to perform at 90% (PPS-G17). A prestriate lesion added to pulvinar damage also had little effect, except in one subject. For two monkeys (PPS-G22 and PPS-G19) there was no effect at all; they performed at criterion following prestriate surgery. A third monkey (PPS-G17) showed a minimal deficit by requiring 4 days of postoperative training (120 trials) to reach criterion. The fourth monkey (PPS-G3) demonstrated an impairment; she required three times as many trials (390 trials) to relearn the color discrimination as she had required to learn it originally (120 trials). This same monkey showed extensive damage to striate cortex compared to the others in Group PPS, and had practically complete lateral geniculate degeneration (Fig. 4). She was also the only operated animal who appeared blind postoperatively.

In contrast to the effects of combined pulvinar-prestriate lesions, inferotemporal lesions produced striking impairments. For three monkeys (IT-342, IT-338, and IT-343) the impairments were severe; they required from 270 to 840 trials to relearn the problem. The fourth monkey (IT-G18) showed no improvement over his preoperative retention score (30 trials), which was below that of all other normal monkeys.

Mean postoperative savings scores following pulvinar, prestriate, and inferotemporal lesions are shown in Fig. 6. The control bar shown in the figure represents mean savings on the preoperative retention test; since there were no differences between Groups PPS and IT preoperatively, this score was computed on the basis of all eight monkeys. The

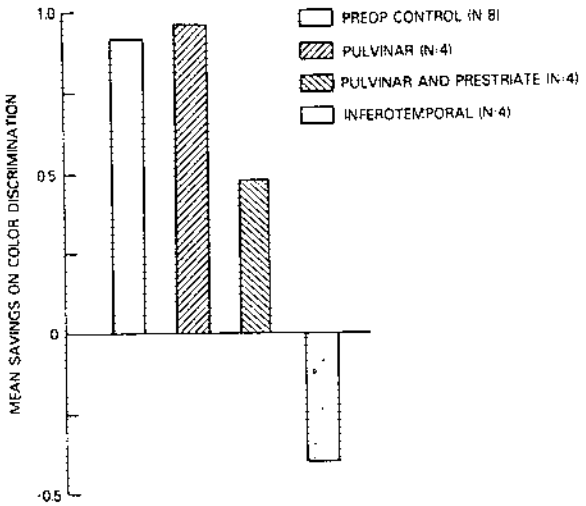

FIG. 6. Color discrimination retention following lesions of the pulvinar, prestriate cortex, and inferotemporal cortex. The control bar is based on the preoperative retention scores of all eight monkeys. Mean savings scores were computed according to the formula: (Trials to Learn −Trials to Relearn) / (Trials to Learn + Trials to Relearn).

results indicate, first, absolutely no effect of the pulvinar lesion. Second, although the addition of a prestriate lesion to a monkey with pulvinar damage did have an effect, it was slight compared to the deficit produced by the inferotemporal lesion. The difference between the effects produced by prestriate and inferotemporal lesions is significant provided PPS-G3 (the monkey with practically complete lateral geniculate degeneration) is excluded from the analysis ($U = 0$, $P = 0{\cdot}056$); when PPS-G3 is included $U = 2$, $P = 0{\cdot}114$.

Experiment 2. *Patterned strings*

Patterned string performance on each of the eight problems is given in Fig. 7. The problems are ordered in increasing difficulty according to the mean per cent correct for Group N. The performance of Group N fell below 80% on all but the two easiest problems, and did not differ significantly from chance on the four most difficult. Monkeys in Group PPS showed normal behavior on all problems following their initial pulvinar surgery. The performance of these monkeys was affected, however, by their subsequent prestriate lesions; they were impaired relative to normal monkeys on the three easiest problems ($U = 0$, $P = 0{\cdot}028$), and also impaired relative to monkeys with inferotemporal lesions on the two easiest problems ($U = 1$, $P = 0{\cdot}058$; $U = 0$, $P = 0{\cdot}028$ respectively). The behavior of monkeys in Group IT did not differ from normal monkeys, except on the third easiest problem ($U = 1$, $P = 0{\cdot}058$), where their performance resembled that of monkeys with combined pulvinar-prestriate lesions. The poor performance of Group IT on this problem was primarily due to the low score of IT-393, the only monkey with lateral geniculate degeneration from Group IT who was tested in this experiment. When his score (53%) is excluded from the data analysis, Groups IT and N do not differ significantly ($U = 1$, $P = 0{\cdot}114$).

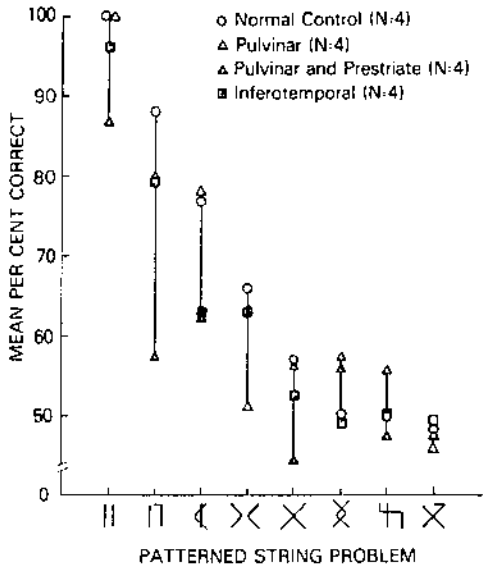

FIG. 7. Patterned string performance of monkeys with pulvinar lesions, combined pulvinar-prestriate lesions, inferotemporal lesions, and unoperated controls.

Experiment 3. Pattern discriminations

The number of trials to acquire each of the four pattern discrimination problems for individual monkeys is shown in Table 1, with group means graphed in Fig. 8. The results indicate that monkeys with inferotemporal lesions were significantly impaired relative to normal monkeys on all four pattern discriminations learned ($U = 0$, $P = 0{\cdot}028$). Monkeys with combined pulvinar-prestriate lesions were impaired only on the first ($U = 1$, $P = 0{\cdot}058$) and third ($U = 0$, $P = 0{\cdot}028$) discrimination learned. There were, however, no statistically significant differences between the two operated groups. On each problem the number of trials to criterion for monkeys in Groups PPS and IT showed considerable overlap, and for both groups there was always one monkey who failed each problem by

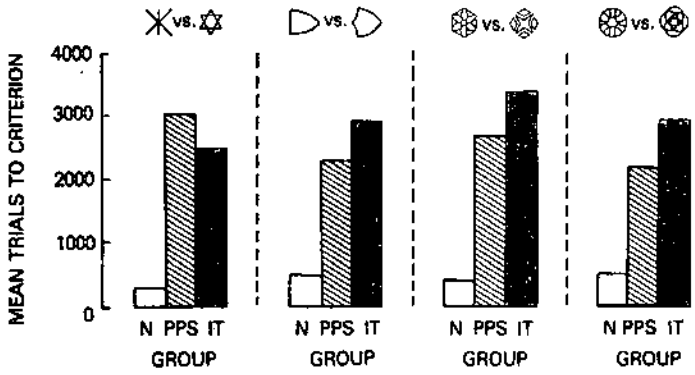

FIG. 8. Pattern discrimination learning by monkeys with combined pulvinar-prestriate lesions (Group PPS), inferotemporal lesions (Group IT), and unoperated controls (Group N).

Table 1. Postoperative acquisition of four pattern discrimination problems: Trials to criterion* for normal monkeys (N), monkeys with combined pulvinar-prestriate lesions (PPS), and monkeys with inferotemporal lesions (IT)

Monkey	Pattern Discrimination†			
	First	Second	Third	Fourth
N — G14	156	200	200	200
N — G23	150	400	200	200
N — 389	502	909	733	1353
N — 410	350	510	624	360
Group N Mean	289·5	505	439	528
PPS — G3	473	5000	1022	732
PPS — G22	3589	400	1066	200
PPS — G17	5000‡	2702	5000	5000
PPS — G19	3148	1080	3698	2832
Group PPS Mean	3052·5	2295·5	2696·5	2191
IT — 393	886	2329	2416	2544
IT — 407	1544	5000	2382	2522
IT — 340	5000	1376	3911	1600
IT — 391	2842	3030	5000	5000
Group IT Mean	2568	2934	3427	2916·5

*Criterion trials are not included.
†The patterns are shown in Fig. 8.
‡Training was discontinued at 5000 trials.

attaining a score of 5000 trials. On the other hand, monkeys in Group PPS did perform within the normal range on the first (PPS-G3), second (PPS-G22) and fourth (PPS-G3 and PPS-G22) problem.

Experiment 4. *Object discrimination*

The mean number of trials to learn the object discrimination for Groups N, PPS, and IT is shown in Fig. 9. Group PPS acquired the discrimination as easily as Group N. Group IT, however, was severely impaired relative to both Group N ($U = 4$, $P = 0{\cdot}048$) and Group PPS ($U = 1$, $P = 0{\cdot}008$). In fact, one monkey with an inferotemporal lesion (IT-343) was performing at only 70% after 1000 trials.

Experiment 5. *Acuity test*

The results of the acuity test are shown in Fig. 10. The data indicate, first, that monkeys in Group N made errors when tested with the thinnest thread. Although an increase in errors on the thinnest thread was even more apparent for monkeys in Group IT, their drop in performance on this thread was not significantly greater than that demonstrated by Group N ($U = 4$, $P = 0{\cdot}190$). By contrast, Group PPS showed significant acuity deficits relative to Group N on all threads but the two thickest ($U = 0$, $P = 0{\cdot}028$). Group PPS was also significantly impaired relative to Group IT on the third ($U = 0$, $P = 0{\cdot}016$) and fourth ($U = 0$, $P = 0{\cdot}016$) thinnest thread.

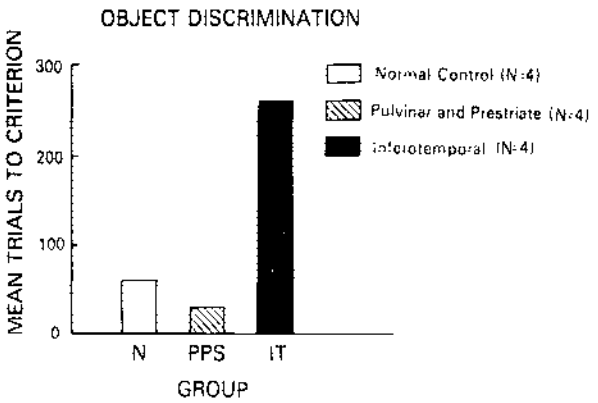

FIG. 9. Object discrimination learning by monkeys with combined pulvinar-prestriate lesions (Group PPS), inferotemporal lesions (Group IT), and unoperated controls (Group N).

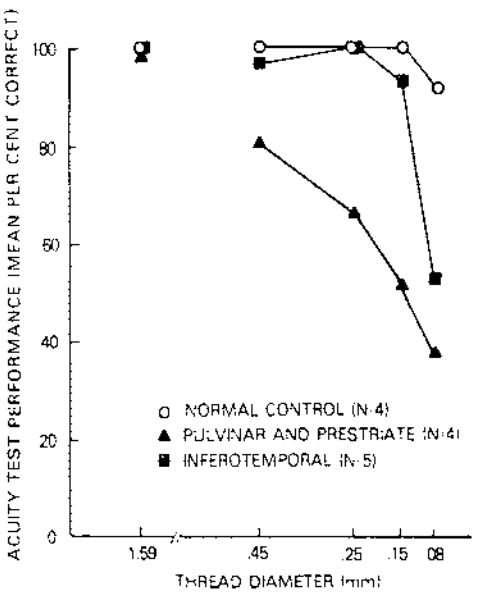

FIG. 10. Acuity test performance of monkeys with combined pulvinar-prestriate lesions (Group PPS), inferotemporal lesions (Group IT), and unoperated controls (Group N).

DISCUSSION

The outcomes of the present series of experiments help considerably in clarifying prior discrepant results and interpretations concerning central processing of visual information. In addition, they confirm once again the major findings that have been consistently obtained in all investigations: bilateral lesions of inferotemporal cortex produce severe impairments on a variety of visual discriminations (e.g. [6, 56, 59, 60]), and bilateral massive damage to that portion of the pulvinar which projects to inferotemporal cortex fails to replicate those impairments [5, 37]. Further, impairments on visual discrimination following lesions of inferotemporal cortex are neither attributable to a sensory loss [56, 61–64] nor related to the physical dimensions of the cues as such [6], but are a function of the discrimination situation in which the cues are embedded.

The discrepancies and controversies in the literature revolve around the effects of lesions of prestriate cortex. In the current experiments radical prestriate resections were superimposed on existing pulvinar damage. The results showed that the effects of these combined lesions were no more severe than those previously reported to follow prestriate lesions alone [5, 65–69]. However, the effects of damage to prestriate cortex were not negligible. While the overall performance of monkeys with prestriate lesions on the color and object discriminations was comparable to that of unoperated controls, one monkey (PPS-G3, who had extensive lateral geniculate degeneration and appeared blind postoperatively) did show a deficit on the color discrimination, the first task administered postoperatively.

But it is the results from pattern discriminations which have provided the greatest discrepancies and thus fuelled controversy. In the present study the overall performance on pattern discriminations of monkeys with prestriate lesions was indistinguishable from that of monkeys with inferotemporal lesions. Still, on three of the four discrimination problems at least one of the four monkeys with prestriate damage performed within the normal range. Further, in contrast to monkeys with inferotemporal lesions, the performance on pattern discriminations of monkeys with prestriate lesions correlated with their performance on the patterned string and acuity tests, which in turn correlated with the extent of degeneration in the lateral geniculate nucleus. With the exception of one monkey (PPS-G3), the acuity scores and the extent of lateral geniculate degeneration were predictive of how much difficulty the monkey with prestriate lesions would have on the pattern discrimination problems. The lesion in the excepted monkey failed to extend into the depth of the superior temporal sulcus bilaterally, and this may be related both to her poor performance on the color discrimination and to her relatively good performance on the pattern discriminations, despite considerable degeneration in the lateral geniculate nucleus. Finally, for monkeys with prestriate lesions the patterns that were the hardest to learn were generally those that were the most detailed and thus required the greatest acuity. This relationship did not hold either for normal monkeys or for those with inferotemporal lesions. The data therefore suggest that there is a sensory component in the discrimination deficit produced by prestriate removal which is most likely related to, but not completely dependent on, involvement on the geniculo-striate system.

The data from the current series of experiments thus agree substantially with those previously reported: lesions of prestriate cortex can [5, 65–69], though they do not necessarily ([25–30, 32]; for review, see [4]), impair visual discrimination performance, and in many instances (for review, see [31]), though not all (e.g. [5]), such impairment is considerably less than that obtained from more restricted lesions of inferotemporal cortex. Further, when restricted lesions are made which divide the reach of cortex from the occipitally located striate region forward to the temporal pole, the posterior lesions (whether called "prestriate" or "foveal prestriate" or "posterior inferotemporal")* and the more anteriorly placed lesions have different effects. When lesions are placed posteriorly there is a correlation

*The literature is confusing with respect to nomenclature. IWAI and MISHKIN [70] showed that a visual discrimination deficit of the type obtained from prestriate resections in the present study results from cortical lesions anterior to the inferior occipital sulcus and ventral to the posterior extent of the superior temporal sulcus; they termed this cortical area "posterior inferotemporal". COWEY and GROSS [71] also found a visual discrimination deficit following lesions of this cortex, but labelled the area "foveal prestriate". Further, when prestriate lesions are made that spare the posterior extent of the superior temporal sulcus, the resections are said to be incomplete because there is a projection from striate cortex to the posterior bank of this sulcus [13–15]. There may be tissue which does not receive such a projection and still produces the type of deficit found in the present study, but this has not as yet been fully established.

between discrimination deficits, if they occur, and the physical dimensions of the cues; no such correlation holds for the effects of more anteriorly placed lesions [5, 71, 72].

There appears, therefore, little ground for controversy on the question of whether the effects of posterior (prestriate) and anterior resections of occipito-temporal cortex differ. The controversy is generated when claims are made that the effects of the posterior resections are nil, and that such findings preclude an orderly progression of visual processing from primary (striate) through secondary (prestriate) to tertiary (inferotemporal) cortical systems. Let us examine each of these controversial statements in the light of the results obtained in the current study.

(1) In the current study, as in several previous ones, extensive resection of prestriate cortex (even when coupled with massive destruction of the pulvinar) failed to impair the performance of some, though not all, visual discriminations. (2) Further, as in all previous studies, whenever impairments occurred they could be related to the physical dimensions of the cues (and to some extent degeneration of the lateral geniculate nucleus due to invasion of the primary visual system). (3) The failure to obtain a deficit on some discrimination tasks has been attributed to sparing of prestriate tissue, but this does not account for the difference between the effects of prestriate and inferotemporal lesions. This difference has been emphasized by two recent demonstrations: monkeys with prestriate lesions are impaired in their ability to respond to distance cues in a size constancy task, while monkeys with inferotemporal lesions simply fail to discriminate [53], and anterior but not posterior lesions impair discrimination reversal learning [73, 74].

These data clarify the issue. The question becomes not only whether signal transmission critical to the functions of inferotemporal cortex demands an intact prestriate cortical pathway between striate and inferotemporal cortex, but whether the functions of striate, prestriate, and inferotemporal cortex are hierarchically ordered. The answer to this question depends on what is meant by "hierarchically ordered". If what is meant is simply that a scotoma is more like a retinal sensory impairment and that discrimination involves decision, learning, and memory, which are clearly central processes, and that perceptual constancy lies somewhere in between, the answer is that of course the functions of striate, prestriate, and inferotemporal cortex are hierarchically ordered. If, however, one asks whether this hierarchy is an anatomically dependent one, as it is between retina and striate cortex, the answer is more difficult to attain. Largely, this is due to the fact that anatomical dependency can take two forms: a direct cascade of connections between striate, prestriate, and inferotemporal cortex may be critical—this is the classical view—or a set of cortico–subcortical relays involving feedback and feedforward circuits may be operative—a view which has been termed, not completely correctly, the "parallel processing model".

The dissociation between the effects of lesions of striate, prestriate, and inferotemporal cortex supports the hypothesis that *separate* visual systems are being addressed [75]. This, however, does not preclude an hypothesis that one system is critically dependent for its visual functions on one or more of the others. The current experiments were undertaken to test this hypothesis and the results can be interpreted, as have previous such results, as confirming or infirming depending whether the prestriate resections are considered "complete". Three regions are especially vulnerable to failures in extirpation: (1) that within the inferior occipital sulcus; (2) that surrounding the anterior extent of the medial calcarine fissure; and (3) that buried in the depth of the posterior extent of the superior temporal sulcus.

We believe that the resections performed in the current experiments and those in previous

studies performed in this [32, 74, 76] and other laboratories [73, 75] disconfirm the hypothesis that removal of any one of these regions is singly responsible for producing as consistent and severe a deficit in visual discrimination as does the inferotemporal lesion. The question remains whether complete removal of all of them together produces such a deficit. Considerable progress towards such a complete removal was made in the current study, but there continues to be some sparing of critical tissue in all subjects, though in different locations in different monkeys. We are especially concerned with sparing in the posterior portion of the superior temporal sulcus, which one of us (Ungerleider) now finds receives a direct input from the entire striate cortex. However, the cortex on the banks of the posterior extent of the superior temporal sulcus has not been shown to project to inferotemporal cortex and, in view of its proximity to parietal cortex, is more likely to be part of the occipito-parietal mechanism involved in the visual guidance of prehension.

In conclusion, the results of the current experiments confirm once again the severe impairment of visual discrimination performance that follows lesions of inferotemporal cortex. They also confirm the independence of this impairment from the physical dimensions of the cues as such. Further, the results confirm prior failures to reproduce this impairment with massive lesions of the thalamic (pulvinar) input to inferotemporal cortex. Finally, in contrast to inferotemporal lesions, extensive prestriate lesions (even in combination with extensive pulvinar damage) have been shown once again to produce either no effect on visual discriminations or an effect that is related to the physical dimensions of the cues, which is partially but not completely correlated with invasion of the geniculo-striate system. These results disconfirm an "input integration model" of the function of inferotemporal cortex, and support the hypothesis that striate, prestriate, and inferotemporal cortex are parts of three separate visual systems which, from other data, are known to be hierarchically related with respect to retinal vs cortical contributions to visual function. Our evidence does not, however, support the hypothesis that the hierarchy is established by a direct cascade of connections between striate, prestriate, and inferotemporal cortex, although this "serial processing model" remains viable. Rather, taken together with the results of earlier experiments, the present data affirm the hypothesis that a set of more or less parallel operations of cortico-subcortical feedback and feedforward circuits is involved in establishing the hierarchy.

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Résumé :

On a comparé les performances des singes avec lésions infero-temporales avec celles des singes avec des lésions combinées du pulvinar et du cortex pré-strié sur des problèmes de discrimination et sur des épreuves de fonctionnement sensoriel. Les lésions infero-temporales perturbaient gravement la discrimination des couleurs, des objets et des patterns mais ne déterminaient aucun déficit sensoriel. En revanche, les lésions combinées pulvinar et prestrié ne perturbaient que la discrimination des patterns c'est-à-dire un trouble associé avec les déficits sensoriels et avec la dégénérescence du corps genouillé latéral. Ainsi, les lésions infero-temporales affectaient les discriminations visuelles per se, tandis que les lésions combinées pulvinar pré-strié affectaient les discriminations liées aux dimensions spécifiques des stimulus visuels. On discute ces effets qualitativement différents des lésions infero-temporales et des lésions combinées pulvinar pré-strié d'après les modèles actuels du traitement central de l'information visuelle.

Deutschsprachige Zusammenfassung:

Affen mit infero-temporalen Läsionen wurden anhand von Diskriminationsaufgaben und von Tests für sensorische Leistungen mit solchen verglichen, die kombinierte Läsionen des Pulvinar und des prästriären Cortex aufwiesen. Die infero-temporalen Läsionen beeinträchtigten die Diskrimination von Phasen, von Objekten und Mustern in erheblichem Maße. Sie verursachten jedoch keine Leistungsstörung im sensorischen Bereich. Im Gegensatz dazu wurde durch die kombinierte Läsion von Pulvinar und Prästriatum nur eine Schädigung der Diskrimination von Mustern hervorgerufen, welche mit sensorischen Mängeln und einer Degeneration des lateralen Corpus geniculatum einhergingen. Daraus folgt, daß die infero-temporalen Läsionen die optische Diskrimination an sich tangieren, während die kombinierten Läsionen von Pulvinar und Prästriatum die Diskriminationsleistungen hinsichtlich spezifischer Dimensionen der optischen Leistung beeinträchtigen. Diese qualitativ unterschiedlichen Auswirkungen der verschiedenen Läsionsorte werden im Bezug auf derzeitige Modelle über die zentrale Verarbeitung optischer Eindrücke diskutiert.