Size Constancy in Rhesus Monkeys: Effects of Pulvinar, Prestriate, and Inferotemporal Lesions

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Summary. The present study tested the theory that inferotemporal cortex integrates 1) distance information transmitted via superior colliculus-pulvinar afferents, with 2) form information transmitted via striate-prestriate cortex afferents (Gross, 1973a, 1973b). Monkeys were trained to choose the larger of two objects, independent of distance, to obtain a reward. Based on the integration theory, the following predictions concerning this size constancy discrimination were made: 1) monkeys with pulvinar lesions, unable to code distance, should be impaired and adopt strategies based on retinal image size; and 2) monkeys with prestriate lesions, unable to code retinal image size, should be impaired and adopt strategies based on distance. Contrary to these predictions, pulvinar lesions produced no deficit; and although prestriate lesions did produce an impairment, it was due to a failure to code distance in assessing the true size of the object. Thus, monkeys with prestriate lesions consistently responded to retinal image size instead of object size. Replicating an earlier report (Humphrey and Weiskrantz, 1969), inferotemporal lesions also produced an impairment; however, errors made by monkeys with inferotemporal lesions were random and could not be attributed to any consistent strategy. All monkeys reacquired the discrimination postoperatively, indicating that there are multiple mechanisms available to the brain-damaged animal for the perception of size constancy.

Key words: Size constancy – Pulvinar – Prestriate Cortex – Inferotemporal Cortex – Vision

Introduction

Size constancy is the ability to correctly judge an object's size despite changes in its distance and hence changes in its retinal image size. Size constancy has been studied in man by requiring subjects to adjust the size of a variable stimulus to match the size of a standard stimulus placed at different distances within a visual alley (e.g., Holway and Boring, 1941). Such studies indicate that under normal binocular viewing conditions subjects show perfect size con-

stancy by adjusting the variable stimulus to the physical size of the standard; only a slight reduction in constancy results when one eye is occluded. However, when depth cues are systematically eliminated there is a dramatic loss of size constancy, resulting in matches between the variable and standard stimuli that are based almost entirely on retinal image size. Thus, the ability to make accurate estimates of the real size of an object appears to be dependent upon the availability of depth cues.

In studies of size constancy in animals, a size discrimination instead of a size match has been required. Initially, the animal is trained to choose the larger of two stimuli that are placed at equal distances. During subsequent tests, the larger stimulus is moved away from the animal, thereby making its retinal image size equal to or less than that of the smaller stimulus. Continued responding to the previously rewarded and objectively larger stimulus indicates the presence of size constancy. Size constancy has been demonstrated with this paradigm in the monkey (Locke, 1937), cat (Gunter, 1951), rat (Heller, 1968), duck (Pastore, 1958), and carp (Herter, 1930, 1953).

Humphrey and Weiskrantz (1969) have reported a severe impairment on size constancy problems by monkeys with lesions of inferotemporal cortex. An interesting aspect of this study was the following: the monkeys' incorrect responses appeared to alternate between two strategies, one based on retinal image size, the other based on distance. Veridical perception of object size depends upon a combination of information about both the retinal image size of an object and its distance from the viewer according to the rule: Perceived Size = k (Retinal Image Size × Distance). Therefore, it seemed that the monkey with an inferotemporal lesion was capable of processing both necessary cues, but could not integrate them to achieve size constancy.

These findings are especially intriguing in the context of Gross' (1973a, 1973b) proposal that inferotemporal cortex functions to integrate inputs that are serially processed from two visual systems: one, a geniculo-striate-prestriate pathway to inferotemporal cortex, transmitting information for form perception; the other, a superior colliculus-pulvinar pathway to inferotemporal cortex, transmitting information for visual space. According to this analysis, retinal image size is coded by the geniculo-striate-prestriate pathway and distance by the superior colliculus-pulvinar pathway. Thus, in a size constancy task where the integration of both systems is required, a monkey with an inferotemporal lesion might be expected to demonstrate response strategies based alternatively on retinal image size and distance.

The present study was designed to test Gross' integration theory of inferotemporal cortex by comparing size constancy deficits produced by inferotemporal lesions to those produced by lesions of brain structures which project to inferotemporal cortex from the two visual systems, namely, prestriate cortex and the pulvinar. The predictions generated by this theory were that: 1) monkeys with prestriate lesions, unable to process retinal image size information, should fail size constancy by adopting response strategies based on distance; and 2) monkeys with pulvinar lesions, unable to process distance information, should fail size constancy by adopting response strategies based on retinal image size.
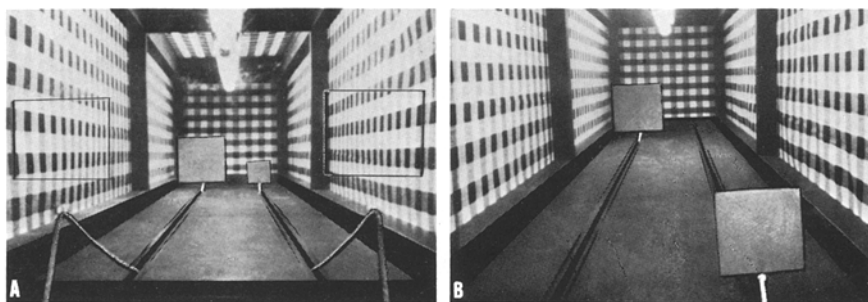
Fig. 1A and B. Apparatus for testing size constancy. **A** The two stimuli are shown at equal distances; thus, the larger stimulus (left) produces a larger retinal image size than the smaller stimulus (right). **B** The larger stimulus is shown at four times the distance of the smaller stimulus; thus, the larger stimulus (left) produces a smaller retinal image size than the smaller stimulus (right). **A** and **B** were photographed at different distances: **A** shows portions of the monkey's compartment in addition to the visual alley; **B** shows the visual alley from the monkey's testing position

Methods

Subjects

Eight young adult rhesus monkeys (*Macaca mulatta*) of both sexes, ranging in weight from 4.1–6.2 kg, served as subjects.

Apparatus

The stimuli were square (16-cm, 8-cm, or 4-cm), red, metal plaques. They were mounted on the front of movable trolleys, viewed down an alley (2.44 m × 0.46 m × 0.46 m). The walls and roof of the alley were checkered black and white, and it was illuminated overhead by fluorescent lights (Fig. 1A). The monkey sat in a transport cage (0.56 m × 0.46 m × 0.51 m) in front of a plexiglass window facing the alley. The trolleys ran along two tracks 0.2 m apart within the alley; thus, the distances of the stimuli from the monkey could be varied. Attached to the front of each trolley was a string which extended through the plexiglass window into the monkey's cage. The monkey responded by pulling in with a string one of the two trolleys, reaching through one of two square holes in the plexiglass window, and pushing over the stimulus plaque, thereby revealing a food well within the trolley. A correct choice was rewarded with a raisin, an incorrect choice terminated the trial. During the 15-sec intertrial interval an opaque door was lowered in front of the plexiglass window.

The stimuli were paired as 16-cm vs. 8-cm and 8-cm vs. 4-cm, so that the larger was always twice the size of the smaller. The distances used were 50, 100, or 200 cm from the monkey; for purposes of computing retinal image size it was assumed that the monkey sat with his head almost at the plexiglass window.

Procedure

Prior to preoperative training, monkeys were adapted for approximately one week to the test situation. This included training the monkey to pull in the trolley (Klüver, 1933) and recover the raisin from the hidden food well. After an initial color discrimination in the apparatus (Ungerleider and Pribram, in press), training was begun on the size constancy problem. The task was to choose the larger of the two stimulus plaques. Discrimination training consisted of 30 noncorrec-

tion trials per day, 5 days a week, until a criterion of 90% correct was met on two consecutive days.

In Stage I of the problem both stimuli were presented at equal distances, 50, 100, or 200 cm. The 30 trials of each session were divided into five 6-trial blocks such that within each block the three distances randomly occurred twice, and for each distance both stimulus pairs (16-cm vs. 8-cm and 8-cm vs. 4-cm) occurred once. The position of the correct stimulus (left or right) was determined according to a pseudorandom sequence (Gellermann, 1933).

When criterion was met in Stage I of the problem, unequal distance trials were introduced such that the larger (or correct) stimulus sometimes produced a smaller retinal image size. Specifically, Stage II consisted of five different trial conditions: i) L1/4dS trials – the larger stimulus was one quarter the distance of the smaller; ii) L1/2dS trials – the larger stimulus was one half the distance of the smaller; iii) L = dS trials – both stimuli were at equal distances; iv) L2dS trials – the larger stimulus was twice the distance of the smaller; and v) L4dS trials – the larger stimulus was four times the distance of the smaller. Since the larger stimulus was twice the size of the smaller, the larger produced a smaller retinal image size on L4dS trials (Fig. 1B), and the retinal image sizes of the two stimuli were equal on L2dS trials; the larger stimulus produced a larger retinal image size on L1/4dS, L1/2dS, and L = dS trials.

The schedule of trials within each 30-trial session of Stage II is given in Table 1. Within each session each trial condition occurred an equal number of times, once in each 5-trial block. Over two consecutive test sessions both stimulus pairs occurred an equal number of times for each of the distance positions. Training continued on Stage II until a criterion of 90% correct was again met on two consecutive days.

All monkeys received surgery (described below) 3–4 weeks following training. Just prior to surgery the monkeys were tested for retention. This provided preoperative retention scores for assessing possible postoperative deficits. Following a 3-week postoperative recovery period, testing was resumed using identical procedures to those employed prior to surgery.

Table 1. Schedule of Trials Within Each 30-Trial Session in Stage II of the Size Constancy Problem

Trial Condition	Distance (cm) of Stimuli from Monkey		Number of Trials
	Large Stimulus	Small Stimulus	
L1/4dS	50	200	6
L1/2dS	50	100	3
	100	200	3
L = dS	50	50	2
	100	100	2
	200	200	2
L2dS	200	100	3
	100	50	3
L4dS	200	50	6

Brain Surgery and Histology

Following completion of preoperative training, four monkeys were assigned to Group PPS and four monkeys were assigned to Group IT. Group PPS received bilateral pulvinar lesions, was tested for retention of the problem, received additional bilateral lesions of prestriate cortex, and was retested. Group IT was tested for retention following a one-stage bilateral lesion of inferotemporal cortex.

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

INFEROTEMPORAL
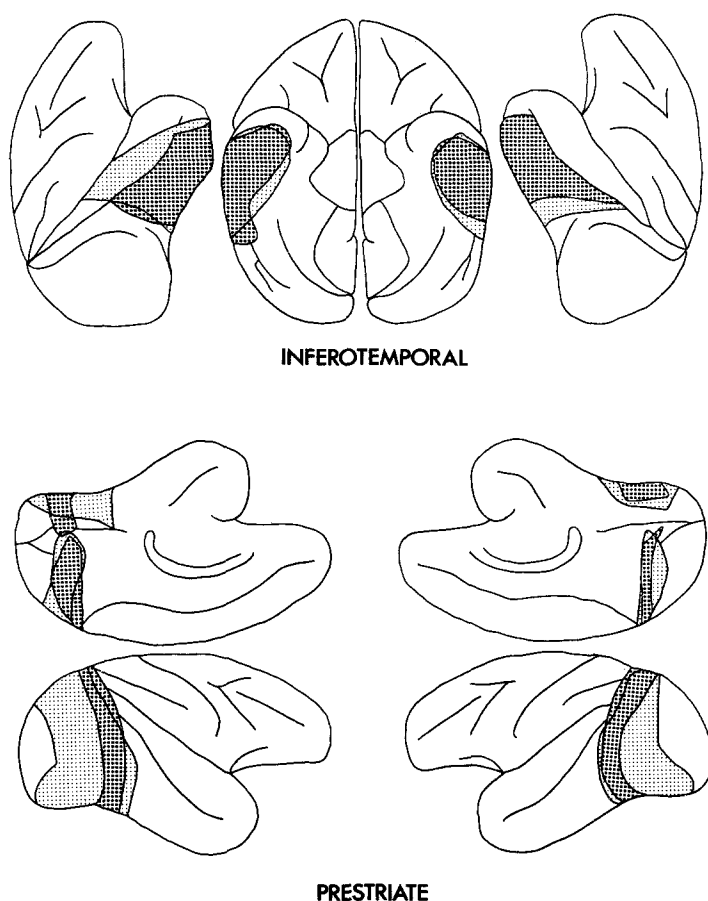
PRESTRIATE

Fig. 2. Minimum and maximum extent of cortical damage for monkeys sustaining inferotemporal and prestriate lesions. Minimum and maximum inferotemporal lesions were produced in IT-342 and IT-343 respectively; minimum and maximum prestriate lesions were produced in PPS-G22 and PPS-G3 respectively. Reconstructions of the individual brains, representative coronal sections through the lesions, and thalamic degeneration are published elsewhere (Ungerleider and Pribram, in press)

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, 1952 atlas), approximately 200 mA of current were passed for 30 sec. The electrodes were 1-mm-diameter 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 lesion, the cortex was exposed by openings rongeured in the skull; for the prestriate lesion, 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.
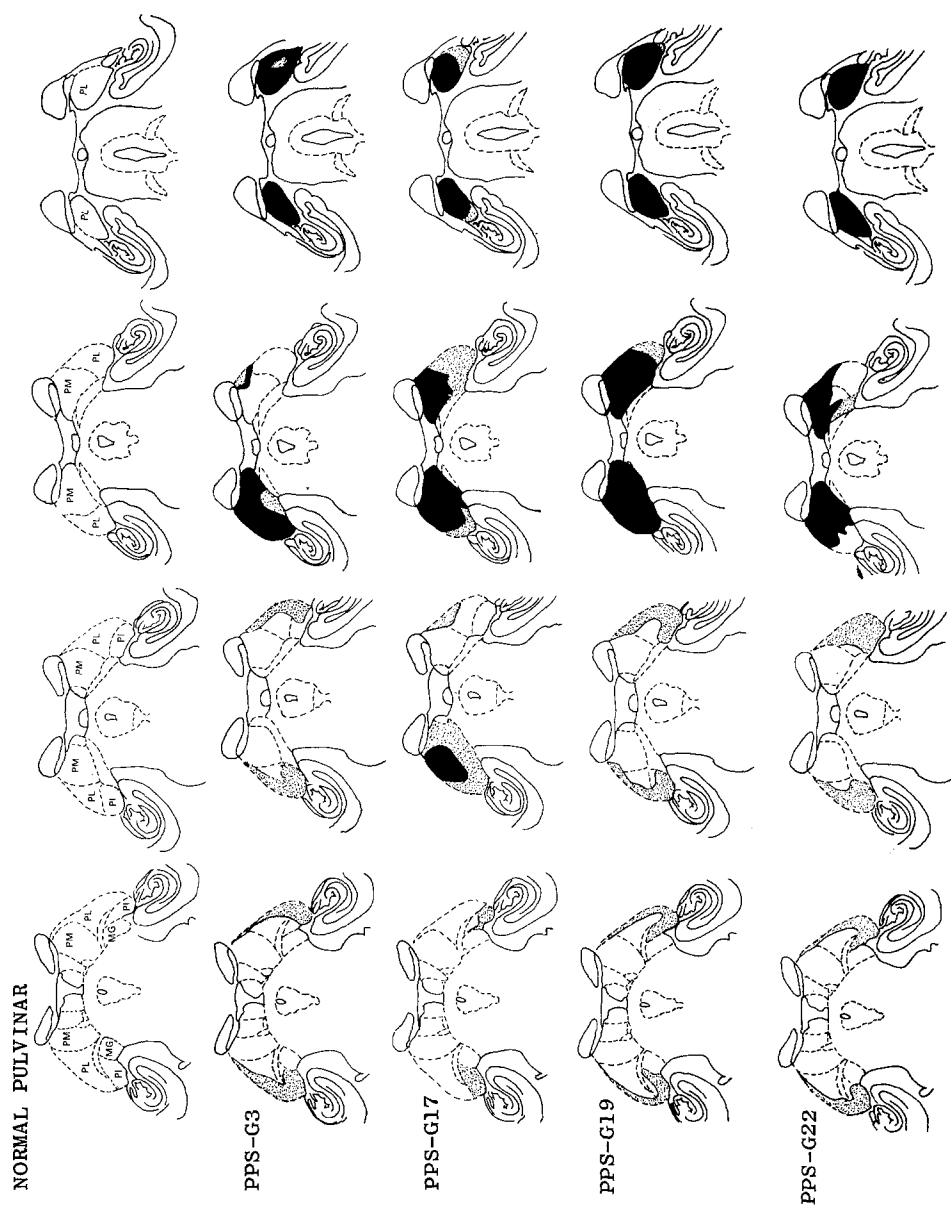
Fig. 3. Pulvinar lesions plotted onto standard coronal sections of a normal monkey brain. Blackened areas indicate the lesion; stippled areas indicate complete cell loss and dense gliosis. Labeling of thalamic nuclei on the normal brain: *PI* = pulvinar inferior, *PL* = pulvinar lateralis, *PM* = pulvinar medialis, *MG* = medial geniculate. Actual tracings of coronal sections through the thalamus of each monkey sustaining a pulvinar lesion are published elsewhere (Ungerleider and Pribram, in press)

The inferotemporal lesion was intended to correspond to area TE of von Bonin and Bailey (1947): 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 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.

Minimum and maximum extent of cortical damage for monkeys with inferotemporal and prestriate lesions are shown in Figure 2. Individual pulvinar lesions are plotted onto standard coronal sections of a normal monkey brain in Figure 3. Reconstructions of the individual brains, representative coronal sections through the lesions, and thalamic degeneration are published elsewhere (Ungerleider and Pribram, in press).

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.

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, was completely destroyed. All four monkeys showed extensive degeneration in the inferior nucleus of the pulvinar as well, most likely a retrograde effect from their subsequent prestriate lesions. 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.

Results

Preoperative Learning and Retention

All monkeys quickly acquired the size constancy problem; the total trials to criterion ranged from 60 (PPS-17) to 330 (PPS-G3). The majority of learning trials occurred in Stage I when the two stimuli were presented at equal distances. Once criterion was met on this initial size discrimination, all monkeys easily transferred to the unequal distance trials of Stage II, requiring only between 30 to 90 trials of additional training before criterion was again reached (Table 2). Mean savings scores from Stage I to Stage II of the problem

Trials to Learn Stage I − Trials to Learn Stage II
Trials to Learn Stage I + Trials to Learn Stage II

calculated independently for the two groups, yielded values of 0.51 for Group PPS and 0.34 for Group IT. Mann-Whitney U Tests (two-tailed) indicated no significant differences between Groups PPS and IT either on total trials to ac-

quire the size constancy problem ($U = 7$, $p = 0.886$) or on savings from Stage I to Stage II of preoperative learning ($U = 6, p = 0.686$).

On the preoperative retention test all but two monkeys (PPS-G3 and IT-338) performed at criterion; these two monkeys required only one additional day of training to bring their performance up to 90%.

Table 2. Trials to Criterion[a] on Preoperative Acquisition and Retention Tests and Postoperative Retention Tests of Equal Distance (Stage I) and Unequal Distance (Stage II) Phases of Size Constancy Discrimination Problem for Monkeys with Pulvinar and Prestriate Lesions (PPS) and Monkeys with Inferotemporal Lesions (IT)

| Monkey	Preoperative Acquisition		Preoperative Retention		Postoperative Pulvinar		Postoperative Cortex	
	Stage I	Stage II	Stage I	Stage II	Stage I	Stage II	Stage I	Stage II
PPS-G3	300	30	0	30	0	30	270	60
PPS-G22	240	30	0	0	30	0	0	60
PPS-G17	30	30	0	0	0	0	90	150
PPS-G19	150	60	0	0	0	0	30	90
PPS Mean	180	37.5	0	7.5	7.5	7.5	97.5	90
IT-G18	240	90	0	0	—	—	90	60
IT-342	120	90	0	0	—	—	60	240
IT-338	120	30	0	30	—	—	270	270
IT-343	120	90	0	0	—	—	60	390
IT Mean	150	75	0	7.5	—	—	120	240

[a]   Criterion trials are not included

Postoperative Retention

A comparison of the effects of pulvinar, prestriate, and inferotemporal lesions, analyzed separately for Stages I and II of the size constancy problem, is shown in Figure 4. Since Groups PPS and IT did not differ significantly from each other on either preoperative learning or preoperative retention tests, the mean preoperative savings scores were computed from the combined data of all eight monkeys.

The results indicate no effect due to the pulvinar lesion alone on size constancy discrimination performance. Preoperative and postoperative savings scores are almost identical on both the equal (Stage I) and unequal (Stage II) distance trials of the problem. In fact, Table 2 shows that all four monkeys performed either immediately at criterion following pulvinarectomy or required only 30 additional trials of training to do so. By contrast, size constancy discrimination deficits were observed in these monkeys following their subsequent prestriate lesions, and were found in the four monkeys given inferotemporal lesions as well.

In Stage I, when the two stimuli were at equal distances, both prestriate and inferotemporal lesions produced a moderate retention deficit relative to preoperative retention scores; mean positive savings scores were +0.30 and +0.18 following lesions of prestriate and inferotemporal cortex respectively.
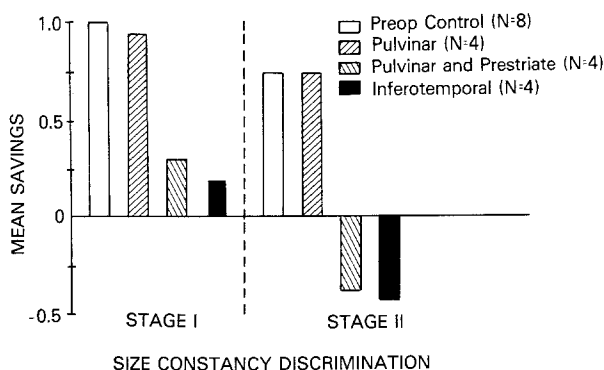
Fig. 4. Effects of pulvinar, prestriate, and inferotemporal lesions on retention of the size constancy problem. Mean savings scores on preoperative and postoperative retention tests, analyzed separately for the equal (Stage I) and unequal (Stage II) distance phases of the problem, were computed according to the formula: (Trials to Learn − Trials to Relearn) / (Trials to Learn + Trials to Relearn). The control bars shown in the figure are based on the preoperative data of all eight monkeys

Examination of Table 2 shows that only one monkey in Group PPS (PPS-G17) and one monkey in Group IT (IT-338) required more trials to relearn Stage I of the problem than to learn it originally.

In Stage II, when the unequal distance trials were introduced, both prestriate and inferotemporal lesions produced a large retention deficit; the mean negative savings scores, −0.38 and −0.42 following prestriate and inferotemporal lesions respectively, indicate that it took the monkeys longer to relearn the problem postoperatively than to learn it originally. As shown in Table 2, this was true for all monkeys in Group PPS and for all but one monkey (IT-G18) in Group IT.

Mann-Whitney U Tests (two-tailed) performed on the postoperative savings score data indicated no significant differences between the deficits produced by prestriate and inferotemporal lesions on retention of either Stage I ($U = 7, p = 0.886$) or Stage II ($U = 6, p = 0.686$) of the size constancy problem.

Finally, in contrast to preoperative learning, there was a notable lack of transfer from Stage I to Stage II of the problem on postoperative relearning. Only one monkey in Group PPS (PPS-G3) and one monkey in Group IT (IT-G18) showed evidence of positive savings from the equal to the unequal distance trials on the postoperative retention test (Table 2). All monkeys, however, did eventually reacquire the size constancy discrimination postoperatively.

Analysis of Size Constancy Errors Following Lesions of Prestriate and Inferotemporal Cortex

To determine the nature of the size constancy impairment produced by prestriate and inferotemporal lesions, instances in which monkeys incorrectly

chose the smaller objects were analyzed. Table 3 summarizes the per cent of total errors on each of the five different trial conditions in Stage II (L1/4dS, L1/2dS, L = dS, L2dS, and L4dS) of the postoperative retention test for individual monkeys. The results show that monkeys in both Groups PPS and IT made relatively few errors postoperatively on the equal distance trials (L = dS). On the unequal distance trials all monkeys made errors, but the pattern of errors made by the two groups differed markedly. A Friedman two-way analysis of variance by ranks (Siegel, 1956) indicated that errors following inferotemporal lesions were randomly distributed across all unequal distance trial conditions ($\chi^2 = 4.28$, $df = 3$, $p > 0.20$), but errors following prestriate lesions were not ($\chi^2 = 10.80$, $df = 3$, $p < 0.02$). Indeed, over 86% of the errors made by monkeys with prestriate lesions occurred on L4dS and L2dS trials; that is, cases in which the smaller object produced the larger retinal image size or cases in which the retinal image sizes of the two objects were equal.

Table 3. Per cent of Total Errors on Each Trial Condition in Stage II of the Postoperative Size Constancy Retention Test for Monkeys with Pulvinar and Prestriate Lesions (PPS) and Monkeys with Inferotemporal Lesions (IT)

|	Trial Condition				
Monkey	L1/4dS	L1/2dS	L = dS	L2dS	L4dS
PPS-G3	12.50	6.25	0	31.25	50.00
PPS-G22	0	0	0	33.33	66.67
PPS-G17	0	10.34	0	24.14	65.52
PPS-G19	6.25	6.25	12.50	31.25	43.75
PPS Mean	4.69	5.71	3.12	29.99	56.49
IT-G18	13.33	20.00	6.67	33.33	26.67
IT-342	27.08	25.00	4.17	25.00	18.75
IT-338	16.33	18.37	0	36.73	28.57
IT-343	16.67	28.20	3.85	37.18	14.10
IT Mean	18.35	22.89	3.67	33.06	22.02

Table 4. Per Cent of Errors Predicted by Retinal Image Size and Distance Response Strategies on each of the Trial Conditions of Stage II of the Size Constancy Problem

	Strategy Adopted by Monkey on Proportion(p) of the Trials			
Trial Category	Larger Retinal Image Size	Smaller Retinal Image Size	Farther Distance	Closer Distance
L1/4dS[a]	0%(p)	100%(p)	100%(p)	0%(p)
L1/2dS[a]	0%(p)	100%(p)	100%(p)	0%(p)
L = dS[a]	0%(p)	100%(p)	50%(p)	50%(p)
L2dS[b]	50%(p)	50%(p)	0%(p)	100%(p)
L4dS[c]	100%(p)	0%(p)	0%(p)	100%(p)

[a] Retinal image size of the larger stimulus is larger
[b] Retinal image sizes of the stimuli are equal
[c] Retinal image size of the smaller stimulus is larger
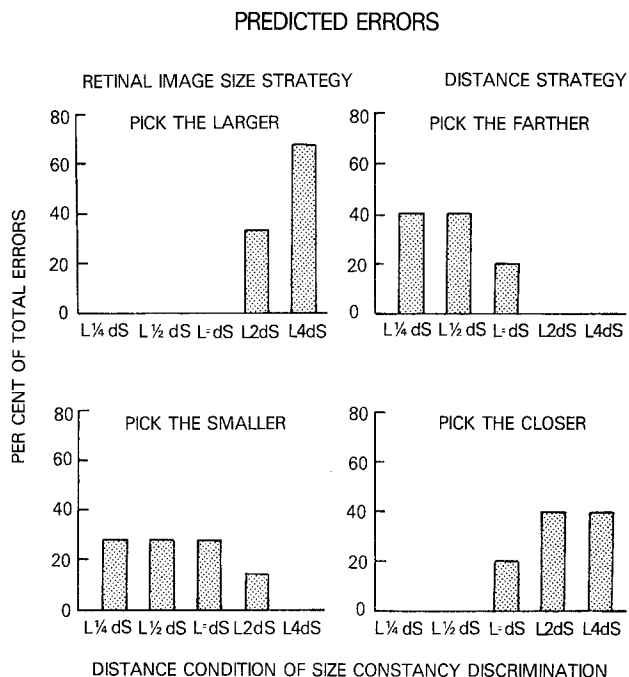
PREDICTED ERRORS

Fig. 5. Predicted distribution of size constancy errors on Stage II trials of the problem, given four possible incorrect response strategies adopted by the monkey. These strategies are: "pick the larger retinal image size", "pick the smaller retinal image size", "pick the farther object", "pick the closer object"

Since perceived size is a function of both retinal image size and distance, in a monkey with impaired size constancy the perception of object size might be dependent upon one or the other cue. Thus, given four possible incorrect response strategies based upon either retinal image size (pick the larger; pick the smaller) or distance (pick the farther; pick the closer) alone, predictions were made concerning the proportion of errors expected on each of the five different distance trial conditions of Stage II. These predictions, shown in Table 4, are based upon the stimulus parameters present in the different trial conditions (see Methods). Since the mechanism for size constancy did not appear to be totally absent, it was assumed that the incorrect response strategies shown in Table 4 were adopted on only a proportion of the trials.

The per cent of total errors for each of the distance trials conditions predicted by these strategies is plotted in Figure 5. The per cent of total errors for each of the distance trial conditions actually obtained following prestriate and inferotemporal lesions is shown in Figure 6. The distribution of observed errors for monkeys with prestriate lesions corresponds remarkably well to the distribution of errors predicted by a "pick the larger retinal image size" strategy. Thus, monkeys with lesions of prestriate cortex responded to the stimulus which produced the larger retinal image size whether or not that
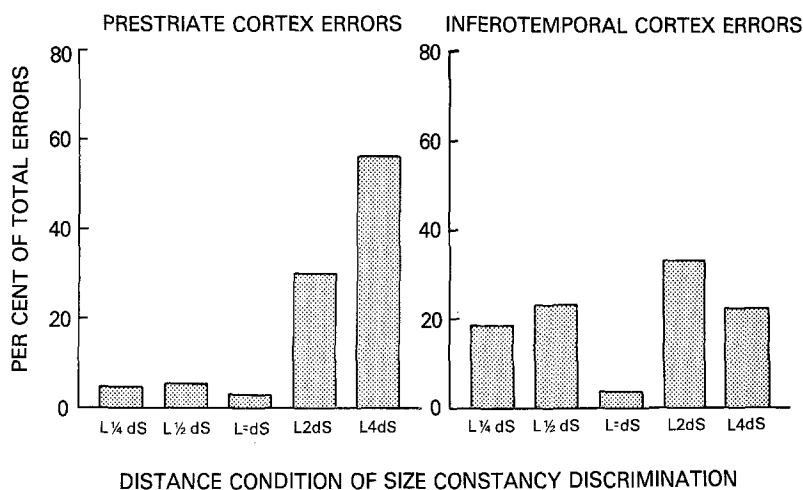
PRESTRIATE CORTEX ERRORS   INFEROTEMPORAL CORTEX ERRORS

DISTANCE CONDITION OF SIZE CONSTANCY DISCRIMINATION

Fig. 6. Observed distribution of size constancy errors on Stage II trials of the problem following lesions of prestriate and inferotemporal cortex

stimulus was objectively larger. For monkeys with inferotemporal lesions, the observed distribution of errors cannot be attributed to any of the four response strategies shown in Figure 5.

To test whether the pattern of errors for monkeys with inferotemporal lesions could be predicted by a combination of alternating retinal image size and distance strategies, the data were further analyzed in accordance with the model developed by Humphrey and Weiskrantz (1969). In brief, if a monkey adopts a retinal image size strategy a proportion p of the time, a distance strategy a proportion q of the time, and the correct strategy a proportion $1\text{-}p\text{-}q$ of the time, then one can predict his performance (per cent correct) on each of the trial conditions [L1/4dS: $100p + 0q + 100(1\text{-}p\text{-}q)$; L1/2dS: $100p + 0q + 100(1\text{-}p\text{-}q)$; L = dS: $100p + 50q + 100(1\text{-}p\text{-}q)$; L2dS: $50p + 100q + 100(1\text{-}p\text{-}q)$; L4dS: $0p + 100q + 100(1\text{-}p\text{-}q)$]. p and q values are chosen for individual monkeys which, substituted into the above equations, give expected scores as close as possible to the observed average scores over the postoperative period. A good fit of expected and observed scores is obtained only if the animal's behavior conforms to the model.

The results of this analysis, performed on the present data, are given in Figure 7. The diagonals shown within the figure represent lines of perfect fit; deviations from these lines indicate discrepancies between observed and expected scores. Three important findings emerge. First, there is an excellent fit of the prestriate data to the model; the mean absolute discrepancy between expected and observed scores is under 2.5%. Note, however, that the q values for all four monkeys in this group are close to zero, indicating again that size constancy errors made by monkeys with prestriate lesions reflect responding to retinal image size. Second, there is a poor fit of the inferotemporal data to the
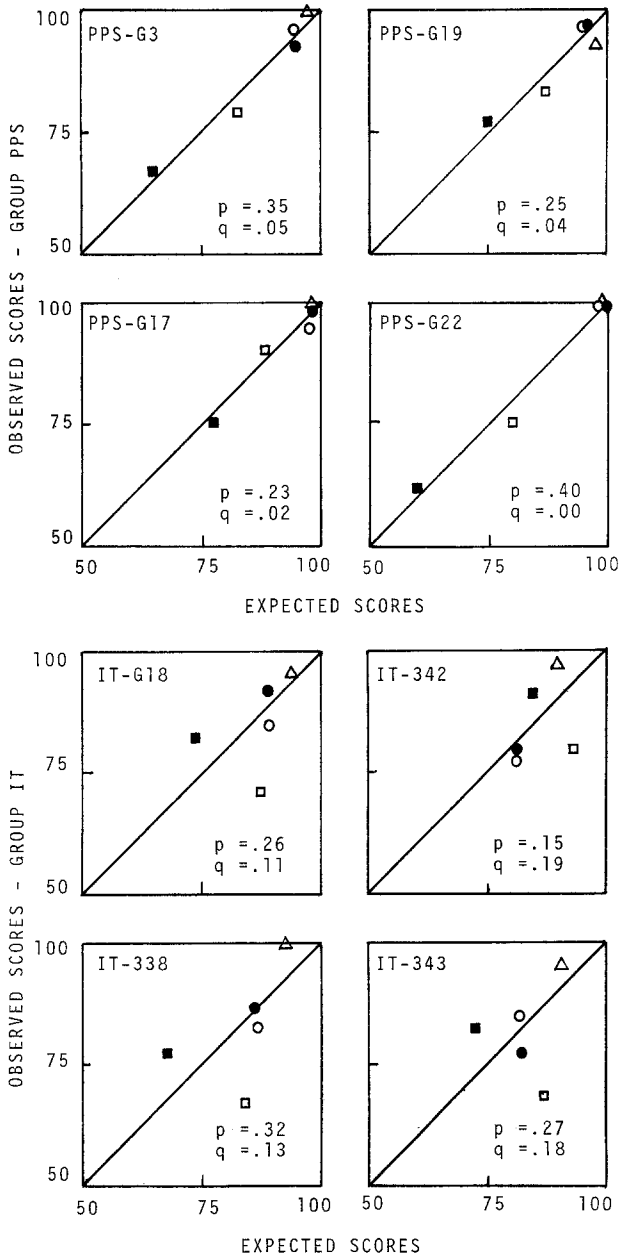
Fig. 7. Scores observed (mean per cent correct) over the postoperative Stage II period compared with scores expected from a model of size constancy deficits based upon alternating retinal image size and distance strategies. p is the proportion of time monkey adopts a retinal image size strategy. q is the proportion of time monkey adopts a distance strategy. Expected scores were calculated from the values of p and q shown for individual monkeys in Groups PPS and IT. Legend: ● L1/4dS; ○ L1/2dS; △ L = dS; □ L2dS; ■ L4dS

model, indicating that monkeys in Group IT did not consistently respond to retinal image size or to distance, nor did they alternate between the two. These results are in contrast with those from the Humphrey and Weiskrantz study; an analysis of variance for repeated measures indicates significantly greater discrepancies from the model for monkeys with inferotemporal lesions in the present study ($F = 31.34$, $df = 1/6$, $p < 0.005$). Third, the greater discrepancies in the present inferotemporal data are specifically on the L2dS and L4dS trials ($t = 7.59$, $df = 6$, $p < 0.001$; $t = 4.09$, $df = 6$, $p < 0.01$; respectively). This is easily explained. The model predicts twice as many L4dS as L2dS errors, an equal number of L1/4dS and L1/2dS errors, and fewer L = dS errors. Thus, if an animal performs well on L = dS trials (having just met criterion on Stage I of the problem) and behaves randomly on all unequal distance trials, he will not fit the model on L2dS or L4dS trials, but can approximate it on L1/4dS, L1/2dS, and L = dS trials. Monkeys in Group IT appear to have done this.

A final analysis of session by session errors was made in an attempt to understand the process of recovery shown by all monkeys. The data indicated only that the same pattern of errors made initially in postoperative retention was present throughout recovery, with a gradual reduction in the absolute number of errors.

Discussion

The present study provides strong evidence against Gross' (1973a, 1973b) theory of inferotemporal cortex as an integration center of distance (via superior colliculus-pulvinar afferents) and form (via striate-prestriate cortex afferents) information. Predictions generated by this theory were that on a size constancy problem where the integration of both afferent pathways would be required, 1) monkeys with pulvinar lesions, unable to code distance, should be impaired and adopt response strategies based on retinal image size; and 2) monkeys with prestriate lesions, unable to code retinal image size, should also be impaired, but adopt response strategies based on distance. Contrary to these predictions, monkeys with pulvinar lesions had no size constancy deficit; subsequent prestriate lesions did produce an impairment, but one directly dependent on a deficiency in distance perception. Because of this deficiency, almost all errors made by monkeys with prestriate lesions involved responding to retinal image size instead of object size. Monkeys with inferotemporal lesions also showed a size constancy impairment, but their errors could not be attributed to any consistent strategy.

Lesions of Prestriate Cortex

The finding that monkeys with prestriate lesions showed poor size constancy as a result of adopting a retinal image size strategy indicates that prestriate lesions produce a basic perceptual disorder: an impaired ability to take distance into account to assess the true size of an object. There are two possible explana-

tions for this deficiency. First, one source of depth information is the binocular cue of retinal disparity. Barlow et al. (1967) and Nikara et al. (1968) have described a group of cells in the cat visual cortex that appears to be specialized for coding depth; that is, they respond best when there is a disparity in the position of the receptive fields from the two eyes. In the monkey, these "binocular depth cells" have been found in area 18, both in the posterior bank of the lunate sulcus and in the annectant gyrus, which is buried in the lunate sulcus (Hubel and Wiesel, 1970). Lesions of prestriate cortex in the present study included this cortical tissue. Thus, one can assume that a large population of retinal disparity detectors was eliminated with ablation of prestriate cortex, producing an impaired ability to code distance. A second source of depth information resulting from the operation of the two eyes is differential sensory feedback from convergence on near and far objects (von Holst, 1955; Gogel, 1961; Richards and Miller, 1969). Since electrical stimulation of prestriate cortex has been found to produce eye movements in monkey (Walker and Weaver, 1940) and man (Lemmen et al., 1958) and specifically convergence and divergence (Jampel, 1960), it is possible that the mechanism which controls this distance cue has also been impaired by lesions of prestriate cortex. Indeed, inasmuch as the stimulus for vergence is the degree of inter-retinal disparity (Rashbass and Westheimer, 1961), a loss of retinal disparity detectors could be directly responsible for such an effect.

Holway and Boring (1941) showed, however, that under monocular viewing there is only a slight reduction in size constancy in man. Therefore, the disruption of both binocular sources of depth information, convergence and retinal disparity, may not fully explain the effect of reducing the monkey with a prestriate lesion to a "retinal image analyzer". The disruption of monocular mechanisms may be involved as well. The most powerful of all the monocular depth cues is motion parallax, the relative "motion" of stationary objects situated at varying distances which arises from head or body movement (Gibson, 1950). Although Bridgeman (1972, 1973) has found that single cells in the monkey striate cortex respond to relative motion, a further stage of neural processing in prestriate cortex may be required to evaluate this potential depth cue in the context of available retinal disparity and convergence information. Indeed, single cells in prestriate cortex, located in the posterior bank of the superior temporal sulcus, appear to be specialized to signal motion in the visual field by responding to changing retinal disparities (Zeki, 1974). Of interest in this connection, both monkeys preoperatively and monkeys with inferotemporal lesions would often begin to respond incorrectly, but would "notice" their error while pulling in the trolley and would attempt to correct themselves; this was never observed in monkeys with prestriate lesions. Thus, still another mechanism for depth perception apparently cannot be utilized by monkeys with prestriate lesions.

The fact that monkeys with prestriate lesions did eventually relearn the problem suggests that there are mutiple mechanisms located in different areas of the brain which enable the brain-damaged monkey to achieve size constancy. For example, many single cells in the monkey striate cortex exhibit receptive field enlargements with near fixations (Smith and Marg, 1975). The per-

ception of size constancy could be subserved by such a "zoom scaling" mechanism.

Lesions of Inferotemporal Cortex

While the nature of the impairment observed in monkeys with prestriate lesions seems clear, the data from monkeys with inferotemporal lesions are not easily explained. Size constancy errors made by monkeys with inferotemporal lesions could not be attributed to any consistent response strategy or even to any consistent combination of different strategies. Deficits produced by inferotemporal lesions seemed to reflect a higher order dysfunction, perhaps attentional (Pribram, 1958; Butter, 1969; Pribram, 1971; Dawson and Ganz, 1975) or mnemonic (Iwai and Mishkin, 1968, 1969; Cowey and Gross, 1970). Alternatively, the impairment may have been caused by continuous perceptual distortions analogous to macropsia and micropsia, visual disturbances in which objects suddenly grow large or small; these have been described in humans with temporal lobe seizures (Penfield and Jasper, 1954). In any event, the fact that inferotemporal and prestriate lesions produced equally severe size constancy deficits but qualitatively different patterns of errors provides further evidence for a dissociation in function between these two cortical areas (Iwai and Mishkin, 1968, 1969; Cowey and Gross, 1970; Gross et al., 1971; Manning, 1971; Manning et al., 1971).

Although monkeys with inferotemporal lesions were impaired on the size constancy problem, replicating Humphrey and Weiskrantz (1969), contrary to that report, monkeys in the present study did not alternate between strategies based on retinal image size and on distance. A partial explanation for this discrepancy may be found in methodological differences between the two studies. In the present study the stimuli were "real objects", in the sense that they stood apart from their textured background and therefore presented monocular parallax cues; in the Humphrey and Weiskrantz study the monkeys looked through windows at stimuli back-projected onto screens located within corridors, thus giving limited monocular depth cues. Indeed, even their normal monkeys had difficulty in learning the problem, and showed the same pattern of errors as the monkeys with inferotemporal lesions. Alternatively, there do exist histological differences between the two groups of operated monkeys: inferotemporal lesions in the Humphrey and Weiskrantz study were larger and invaded portions of prestriate cortex.

Lesions of the Pulvinar

The absence of a pulvinar effect in the present study supports earlier reports of similar negative findings. Monkeys with pulvinar lesions have been observed to perform as normals in the acquisition and retention of color, three-dimensional object, concurrent object, and pattern discrimination problems (Chow, 1954; Mishkin, 1972; Ungerleider and Pribram, in press). Moreover, the lack of a pulvinar deficit on a size constancy discrimination extends prior negative re-

sults to include a visual task in which spatial information must be perceived to solve the problem.

Christensen et al. (1975) have recently reported that monkeys with pulvinar lesions show abnormally prolonged duration fixations during acquisition of a visual discrimination problem, an effect which may be similar to eye movement impairments resulting from superior collicular lesions (Whitteridge, 1960; Denny-Brown, 1962; Wurtz and Goldberg, 1972). Involvement of the pulvinar in eye movement control would explain the finding that monkeys with pulvinar lesions are impaired in the acquisition of difficult pattern discriminations only when the stimuli are flashed very briefly (Chalupa et al., 1976). Future studies should pursue this possibility.

To summarize, prestriate cortex appears basically involved in the perception of size constancy. Prestriate lesions disrupt size constancy by producing an impaired ability to take distance into account in assessing object size. A compensation for this deficiency is possible, however, since size constancy recovers following prestriate lesions. On the other hand, inferotemporal lesions interfere with constancy only in that visual discriminations are involved, and pulvinar lesions have no effect at all.

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