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Basic visual capacities and shape discrimination after lesions of extrastriate area V4 in macaques

WILLIAM H. MERIGAN

Department of Ophthalmology and Center for Visual Science, University of Rochester, Rochester

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Abstract

Ibotenic acid lesions were made in four macaque monkeys in a region of cortical area V4 that corresponds to the lower quadrant of one hemifield. For visual testing, fixation locus was monitored with scleral search coils and controlled behaviorally to place test stimuli either in the lesioned quadrant or in a control location in the opposite hemifield. Some basic visual capacities were slightly altered by the lesions; there was a two-fold reduction of luminance contrast sensitivity as well as red-green chromatic contrast sensitivity, both tested with stationary gratings. On the other hand, little or no loss was found when contrast sensitivity for detection or direction discrimination was tested with 10-Hz drifting gratings nor was there a reliable change in visual acuity. Hue and luminance matching were tested with a spatially more complex matching-to-sample task, but monkeys could not learn this task in the visual field locus of a V4 lesion. If previously trained at this locus, performance was not affected by the lesion. In contrast to the small effects on basic visual capabilities, performance on two form discrimination tasks was devastated by V4 lesions. The first involved discriminating the orientation of colinear groups of dots on a background of randomly placed dots. The second involved discriminating the orientation of a group of three line segments surrounded by differently oriented line segments. Some selectivity of the deficits for form discrimination was shown by the lack of an effect of the lesions on a global motion discrimination. These results show that while V4 lesions cause only slight disruptions of basic visual capacities, they profoundly disrupt form discriminations.

Keywords: Contrast sensitivity, Hue discrimination, Texture discrimination, Area V4, Macaque

Introduction

Extrastriate area V4 has been implicated in visual form discrimination by several types of evidence. It is located next to, and provides much of the neuronal input to, cortical areas TEO and TE (Baizer et al., 1991), which make up the inferotemporal cortex. These areas, in turn, have been tied to form discrimination by the devastating effects lesioning them has on form discriminations (Gross, 1973), and by the discovery of highly specific trigger features (e.g. faces) for their neurons (Gross et al., 1972). Single neurons in both inferotemporal cortex and area V4 show complex modulation of their responses by behavioral context (Moran & Desimone, 1985; Haenny et al., 1988), further suggesting an important role in attentional aspects of form discriminations. Finally, much of the neuronal input to area V4 comes from cortical area V2 (DeYoe & Van Essen, 1985; Shipp & Zeki, 1985), which has also been shown by lesion studies (Merigan et al., 1993) to play an important role in form discrimination.

There have been numerous attempts, prior to the present study, to clarify the role of area V4 by studying the visual consequences of its removal. Many studies (Dean, 1979; Heywood & Cowey, 1987; Heywood et al., 1992; Walsh et al., 1992) aimed for complete bilateral removal of area V4 in monkeys and tested visual capacities without monitoring eye position. (At the time of Dean's study the full extent of V4 was not known.) These methods are problematical for two major reasons, possible reliance by the monkey on an intact portion of V4, given the impossibility of ensuring that all of V4 is removed, and inadvertent damage to the optic radiations. Despite these limitations, the earlier studies suggest that V4 lesions cause little or no deficit in color discrimination, but rather substantial effects on form discriminations.

More recent work (Desimone et al., 1990; Schiller, 1993; Schiller & Lee, 1991) has avoided one difficulty of earlier studies by making more localized lesions of the lower field representation of area V4, and testing vision while monitoring fixation. Unfortunately, the lesion technique in these studies remained aspiration of cortical tissue, a technique that can damage fibers of passage, making precise reconstruction of lesion extent impossible.

The present study used ibotenic acid injections to make localized, axon sparing, V4 lesions of the representation of a lower field quadrant, and then determined both basic and complex visual capacities with controlled fixation testing. A major goal was to compare the effects of V4 lesions with the previously

Reprint requests to: William H. Merigan, Department of Ophthalmology, Box 314, University of Rochester Medical Center, Rochester, NY 14642, USA.

measured effects of lesions of areas V1 and V2 (Merigan et al., 1993).

Methods

Subjects

The subjects were four adult, female monkeys (*Macaca nemestrina*) of approximately 5 kg body weight. They had free access to monkey chow, supplemented regularly with fresh fruit, and their water was withheld for approximately 20 h before threshold testing 5 days each week. All testing was done binocularly using behavioral control of fixation locus, and no monkey had more than 0.5 D of refractive error in either eye.

Placement of lesions

In an aseptic procedure, the portion of area V4 from the dorsal tip of the inferior occipital sulcus (IOS) to about 10 mm dorsal to this locus and between the lunate and superior temporal sulci (LS and STS) was exposed with a craniotomy and durotomy. A dense grid of ibotenic acid injections (2 μl, 10 μg/μl) was made throughout this region with 2 mm center-to-center spacing, at a depth of about 0.7 mm below the cortical surface. An additional row of injections was placed below those along the borders of the LS and the STS at a depth of about 2.7 mm. The total number of injections and the injected hemispheres for the four monkeys were as follows: monkey 1-93 in left hemisphere, monkey 2-67 in right hemisphere, monkey 3-77 in right hemisphere, and monkey 4-55 in right hemisphere.

Implantation of scleral search coil and headmount

After placement of the lesion in monkeys 1 and 2 and before placement of the lesion in monkeys 3 and 4, a scleral search coil was implanted in the right eye of each monkey under isoflurane anesthesia, so that eye position could be monitored, and a stainless-steel sleeve attached to the skull so that the monkey's head could be immobilized (Judge et al., 1980). The fixation window was ±0.5 deg and stimuli were presented after the monkey fixated within the window for 0.5 s.

Apparatus

All testing was conducted using one of three displays. The first was a high-resolution Tektronix 606 oscilloscope (P-31 phosphor) that was used at a distance of 57 cm to test visual acuity. Display luminance was 15 cd/m^2. The second was a 19-inch color monitor (Conrac 7211), used at a distance of 211 cm to test contrast sensitivity, hue discrimination and colinearity, and texture perception. The mean luminance of this display was kept at 65 cd/m^2. The third was a 14-inch Macintosh color monitor with a mean luminance of 42 cd/m^2, used at a distance of 114 cm to test global motion perception.

Procedure

Procedures common to all measures

A red fixation spot was projected onto the face of the display, and when the monkey fixated within ±0.3 deg of the spot, the test stimulus was presented at a single location in each session that was chosen to test a region of the visual field. The interval between trials was 4 s, correct choices (see below) were rewarded with fruit juice, incorrect choices were followed by a 6-s beeping tone, and fixation breaks or premature responses were followed by a 3-s beeping tone.

Not all measures were obtained for all monkeys. For example, mapping of the visual field with contrast sensitivity measures was done for only monkeys 1 and 2, whereas contrast sensitivity in monkeys 3 and 4 was measured at only two locations; 3 deg below the horizontal meridian and 4 deg right or left of the vertical meridian. All of the results obtained for all monkeys are shown in Figs. 4-12. Daily sessions consisted of 200 trials. In those conditions in which thresholds were measured, stimulus difficulty became one step easier after each error, and one step more difficult, with probability 0.33, after each correct choice. Thresholds were taken at 75% correct responding either by linear interpolation, or by probit fits to the daily psychometric functions (Finney, 1971).

1. *Contrast sensitivity* was measured by having the monkey discriminate the orientation of small patches of vertical or horizontal grating displayed on the Conrac monitor. The grating targets were Gabor functions (cosinusoidal gratings multiplied by horizontal and vertical Gaussian weighting functions) generated on an Adage 3006 raster display unit and presented at a frame rate of 60 Hz (noninterlaced). The horizontal and vertical Gaussian weighting functions had space constants of $s = 1.14$ deg. Thus, the grating was above 37% of peak contrast (full width at the $1/e$ point) over a region of 2.28 deg. The time course of appearance of the stimuli was made slow to minimize temporal modulation other than that inherent in the temporally modulated or drifting gratings. Thus, contrast onset followed one-half cycle of a raised cosine of 0.5 Hz. (1) *Isochromatic luminance contrast sensitivity* and (2) *isoluminant red-green chromatic contrast sensitivity* were tested with stationary 1 cycle/deg patches of grating. Isoluminance was determined for each monkey by finding the luminance balance that gave the highest contrast sensitivity at a spatial frequency of 0.3 cycle/deg (Merigan, 1989). Thresholds were first tested with the orientation discrimination that was also used for acuity (below) and then replicated with a procedure that required the monkey to report on each trial whether or not the grating was present (Yes-No procedure). In all procedures, the monkey responded by pressing on one of two panel-mounted buttons (left for horizontal grating, stimulus absent, or direction to the left; and right for vertical grating, stimulus present, or direction of movement to the right). Drifting luminance gratings of 1 cycle/deg and 10-Hz drift rate (the horizontal Gaussian envelope remained fixed and the cosinusoidal component moved to the right) were then used to test (3) *contrast sensitivity for detection of drifting gratings* (monkey reported if the drifting grating was present) and (4) *contrast sensitivity for discriminating their direction of motion* (monkey reported if grating was moving to the right or left).

2. *Acuity* was tested by having the monkey discriminate between vertical and horizontal sinusoidal gratings. High spatial-frequency gratings were bordered with a 1.5-deg circular surround. These stimuli were presented 3 deg below and 4 deg to the right of the fixation spot. The spatial frequency of the grating increased with probability 0.33 after each cor-

rect trial, and decreased after each error. Other procedures were as described for contrast sensitivity.

3. *Hue matching* was tested in a matching to sample procedure using the stimulus configuration shown in the upper right of Fig. 9. High-contrast (50% of maximal contrast possible with this monitor), isoluminant, Gaussian patches of color were used with two colors presented on each trial, one of the colors appearing as the sample, and later as one of the match stimuli, and the other color as the other match stimulus. The mean color tested in each session was one of the four principal directions (0 deg, 90 deg, 180 deg, and 270 deg) in the McLeod Boynton color space (Krauskopf et al., 1982). The two colors presented were ± angular deviations from the mean color. Early in each session the deviations were large (up to ±45 deg for training), and they decreased during testing according to a staircase procedure (as above) of ±1 deg steps. Both simultaneous (sample remained on when matches appeared) and successive (sample turned off at match onset) matching to sample procedures were used. While fixation was within ±1/4 deg of the fixation spot, the sample patch was presented and followed after 1.5 s by the two flanking match stimuli. The monkey responded by pressing the side of the appropriate match, all the while maintaining fixation.

4. *Colinearity discrimination* was tested with procedures previously described (Merigan et al., 1993) using targets containing two lines of either vertical or horizontal colinear dots, whose orientation was masked by a background of irregularly placed dots. Sample targets are shown in Fig. 11.

5. *Texture segmentation* was tested with textures made up of short oriented line segments (Merigan et al., 1993). Three adjacent, misoriented line segments formed either a vertical or horizontal group, as illustrated in Fig. 12, and the monkey had to identify the orientation of the group.

6. *Global motion perception* as described by Pasternak (Pasternak & Merigan, 1994) was tested in monkey 4 for comparison with the colinearity and texture discriminations described above. The dynamic dot pattern consisted of a 2 × 2 deg patch of 100 bright dots on a dark background, presented, like the other stimuli described above, 3 deg below and 4 deg to the right or left of fixation. Dots were moved 0.3 deg every 60 ms for a global velocity of 5 deg/s. The range of directions for individual dots was ±90 deg of the mean direction. The monkey reported on one of two buttons whether the mean direction of global motion was to the right or left. Threshold for the fraction of dots necessary for direction discrimination was determined with the staircase procedure described above.

Reconstruction of lesions

At the conclusion of behavioral testing, the lesion placed in each of the monkeys was reconstructed with anatomical techniques. The monkey was euthanized and perfused with a saline rinse, followed by 4% paraformaldehyde in phosphate-buffered saline. The brain was removed, blocked, and 40-μm sections were cut on a freezing microtome. One section in four was reacted for cytochrome oxidase activity (Wong-Riley, 1979), a second with cresyl violet, and a third for myelin (Gallyas, 1979). The lesions were then reconstructed from these anatomical sections.

Results

Fig. 1 shows the approximate location of the V4 lesion in the macaque brain and the visual field representation served by this portion of V4, the contralateral lower quadrant of the visual field. The exact visual field locations affected by the lesions varied with lesion dimensions.

Fig. 2 shows horizontal histological sections through the lesion of monkey 4, which approximately correspond to the planes indicated by dotted lines in Fig. 1. The lesion extends from the large arrows in the lunate sulcus (LS) to the small arrows on the anterior bank of the superior temporal sulcus (STS).

The location and extent of the V4 lesion in all four monkeys is illustrated in Fig. 3 on a flattened cortical representation. The heavy solid lines show the boundaries of the STS, LS, and inferior occipital sulcus (IOS), while the dashed lines show the primary fundi of these sulci. A short dotted line within the STS shows the secondary fundus of this sulcus. The black areas show regions of complete destruction of cortex. The inset shows the approximate retinotopic organization of this portion of area V4 from Gatass and colleagues (Gatass et al., 1988).

Fig. 4 shows a representation, in visual field coordinates, of the measurement of contrast sensitivity across the V4 lesioned area (hatching) and control regions for two monkeys. The center of each symbol represents the location tested, and the diameter of each is proportional to linear contrast sensitivity at that location. The open circles show the same measures for a location 3 deg below and 4 deg left and right of fixation, a location where several indices of acuity and contrast sensitivity shown in the next figures were measured. These results are displayed with linear scaling of contrast sensitivity, which exaggerates the effect of the V4 lesion on contrast sensitivity, in order to provide an estimate of the visual field loci affected by the lesion.

Fig. 5 shows the luminance contrast sensitivity measures for locations marked by the open symbols in Fig. 4 in the more traditional log contrast sensitivity plot. It can be seen that, although the effects of the lesion on sensitivity are rather small, they are substantial relative to the SEM. We repeated the testing of luminance contrast sensitivity in monkey 4 using low (0 Hz), middle (3 Hz), and higher (10 Hz) velocity, 2 cycle/deg drifting gratings, with otherwise identical procedures, and found approximately a two-fold loss at the lowest velocity, less loss at 3 Hz and no threshold change at 10 Hz.

Chromatic contrast sensitivity, tested with stationary 2 cycles/deg isoluminant red-green gratings, is shown for the four monkeys in Fig. 6. Again sensitivity was decreased about a factor of two for all monkeys.

Fig. 7 shows two types of thresholds measured with drifting, 10-Hz luminance gratings in two monkeys. Data marked "det" show detection thresholds measured with a Yes–No procedure, while those marked "dir" show contrast thresholds for discriminating the direction of motion of the gratings. In no case were the lesion effects statistically significant (Mann-Whitney *U* test) nor did any show a substantial effect of the V4 lesions.

Visual acuity at the visual field locations marked by the open symbols in Fig. 3 is shown in Fig. 8 for all four monkeys. Acuity was little affected by the lesion for all monkeys except monkey 3, for whom it was decreased by almost a factor of three.

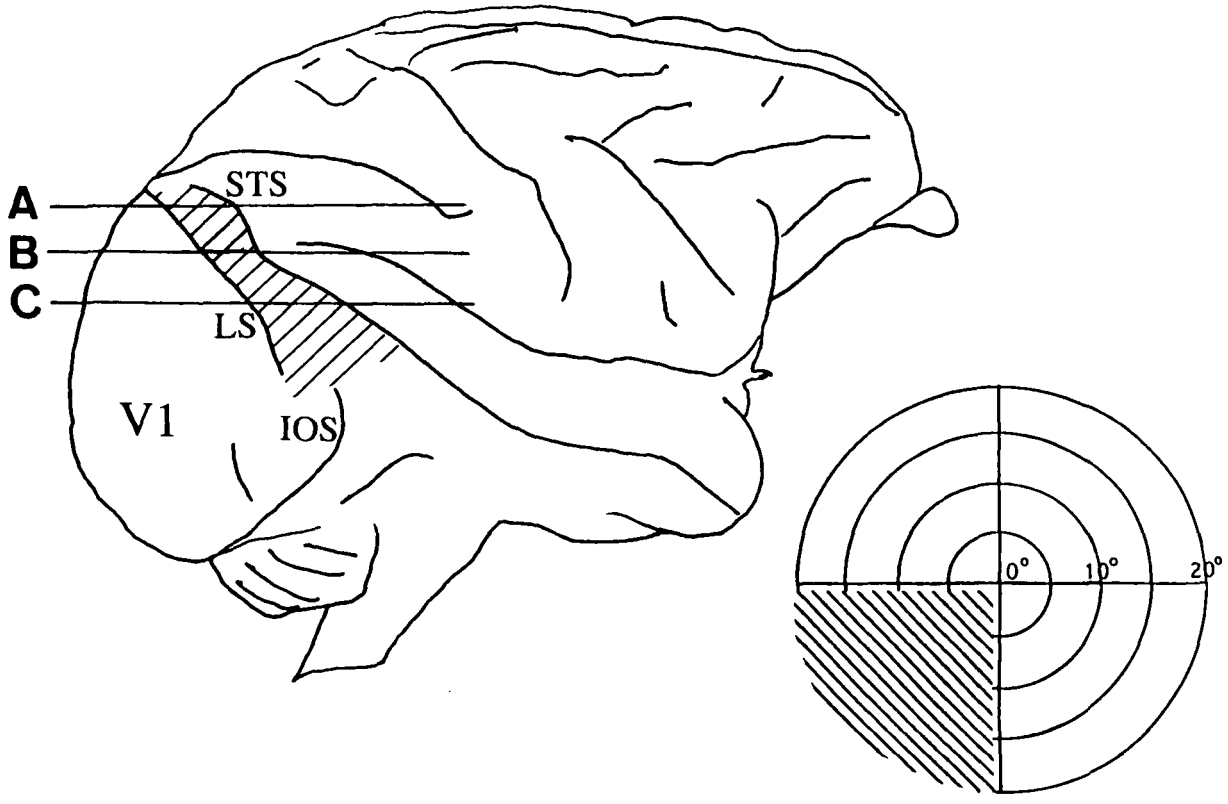

Fig. 1. Line drawing of a lateral view of the macaque brain showing the location of the V4 lesions of this study. Lines A, B, and C show the approximate locations of histological sections through the lesion in monkey 4 (Fig. 2). The inset shows that the intended lesion affects the lower contralateral quadrant of the visual field.

Acuity was subsequently tested in monkey 3 with a detection rather than an orientation measure, and for this test was found to be decreased only by about 15%. Acuity was transiently decreased after the lesions in the other monkeys, but recovered within a few weeks to almost normal levels.

Hue discrimination was tested in four directions of the color space of Krauskopf et al. (1982) using Gaussian-shaped patches of color, and typical results (here for the reddish direction) are presented in Fig. 9 for monkeys 1 and 3. The monkeys were unable to perform this task in the location of the V4 lesion. Every effort was made to establish this performance in these monkeys at the lesion location, including testing at a variety of color directions, testing with luminance stimuli, and gradually, from trial to trial, moving the test stimuli into the lesion locus across the vertical meridian.

Subsequently, in the final monkey tested (monkey 4), the sequence of conditions was modified to include training on the matching to sample task in each of the visual field quadrants before the lesion was placed. Fig. 10 shows that the hue matching performance of this monkey around the reddish direction in color space was not affected by the lesion. This monkey was also able, without explicit pretraining, to do luminance matching, matching on untrained color dimensions, as well as the matching of simple two-dimensional shapes, including triangles and squares.

All thresholds described to this point involved basic visual capacities, and such measures were either little affected (visual acuity, sensitivity for 10-Hz drifting gratings, hue discrimination) or reliably reduced by about a factor of two (luminance and chromatic contrast sensitivity). The next measures were of more complex discriminations, and the effects were more dramatic.

Detection of lines of colinear dots among background dots is shown in Fig. 11 for lesion and control locations. Example stimuli, shown above the data, display the lines of dots on a background of seven dots. Monkey 2 showed only a modest reduction in the number of background dots tolerated, while monkeys 3 and 4 were more severely affected.

Fig. 12 shows discrimination of the orientation of groups of three misoriented lines in a texture of line segments. All three monkeys tested could perform this task above 80% correct in the control region of the visual field, but performance was reduced to chance in the region corresponding to the V4 lesion.

Monkey 4 was also tested on a discrimination of global motion direction (Pasternak & Merigan, 1994) after a V4 lesion. Testing was at the lesion location and in the opposite field, and no pre-lesion training was given on this task. However, the V4 lesion caused no effect on sensitivity to global motion direction.

Discussion

This is the first study of the visual effects of V4 lesions in which it is clear from lesion reconstructions that the lesions were complete over the visual field locations tested, and did not extensively invade other cortical areas. The behavioral mapping of contrast sensitivity in two monkeys confirmed that the visual field extent of the lesion included all loci in which stimuli were later presented. These features of the experiment make it cer-

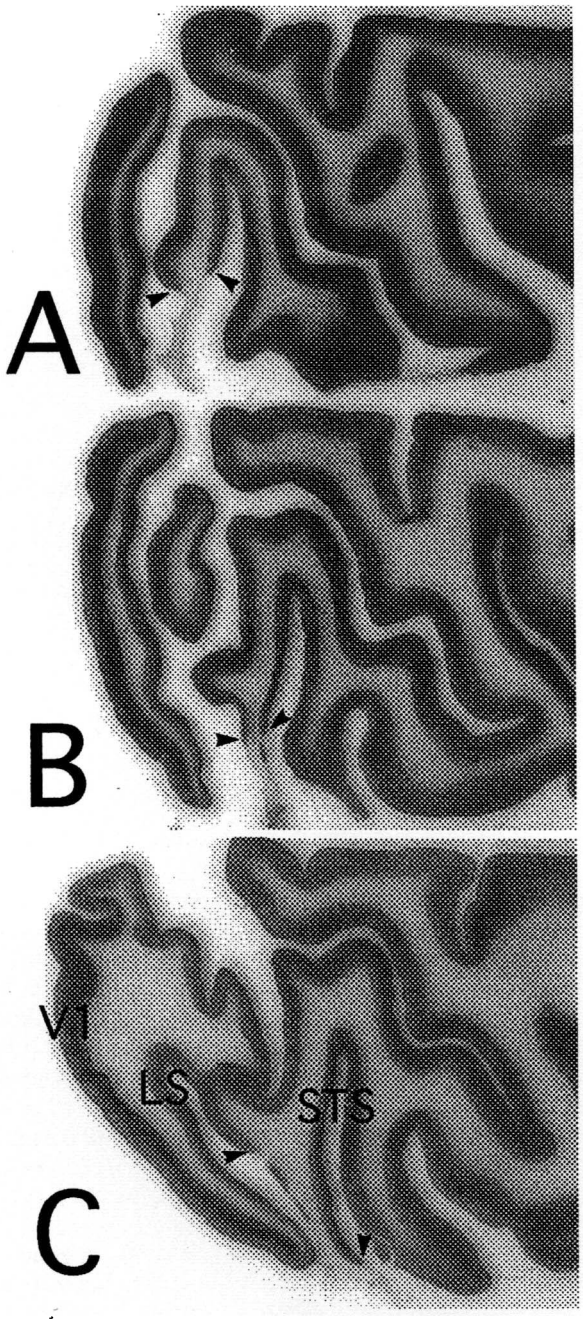

Fig. 2. Histological sections in a horizontal plane through the lesion of monkey 4 that correspond roughly to the planes shown in Fig. 2 as A, B, and C. Sections are 40 μm in thickness and are stained for cytochrome oxidase. The lesion can be seen between the arrows as a region of reduced or absent grey matter above the unstained white matter.

tain that the two-fold decrease in low temporal-frequency contrast sensitivity can be attributed entirely to lesions of area V4. Furthermore, the sparing of contrast sensitivity at higher temporal frequencies cannot be ascribed to sparing of some portion of V4. This study also demonstrates that the sparing of color discrimination reported in many previous studies does not require reliance on some spared part of the V4 visual field. Two dramatic effects of the V4 lesions were an apparent inability to learn a visual task that involved a comparison of stimuli in different parts of the visual field (matching to sample), and a complete disruption of two types of visual shape discrimination. Because of the precise reconstructions, we can say with reasonable certainty that these large effects were also due to the loss of area V4 alone.

Contrast sensitivity and acuity

All four monkeys showed an approximately two-fold loss of contrast sensitivity for low temporal-frequency luminance and chromatic gratings. In addition, we subsequently obtained a similar result by measuring contrast sensitivity with a detection, Yes-No procedure in two of the monkeys, thus insuring that the sensitivity loss was not due to disruption of the monkey's ability to discriminate different orientations. This loss, although reliable, was probably not sufficiently large to cause any difficulty in the suprathreshold discriminations measured later. Nonetheless, it was a bit surprising, both because some earlier studies of V4 lesions had found little effect on grey level discriminations (Heywood & Cowey, 1987), but nonetheless could not rule out an effect as small as the present two-fold loss, and because lesions of other cortical areas beyond V1 (Pasternak et al., 1989; Merigan et al., 1993) cause little change in contrast sensitivity. Two possible explanations come to mind. The first is that the large receptive fields of area V4 neurons may mediate greater psychophysical contrast sensitivity than lower level cortical areas, perhaps by virtue of their greater areal summation (Pelli, 1985). It is not known if the contrast sensitivity of single neurons in area V4 is actually greater than that of those in V1, although it would not be surprising, given that the sensitivity of MT neurons is higher than that of V1 neurons (Sclar et al., 1990). A second possibility is that one effect of the lesion was to increase equivalent internal noise (Watson, 1990) in visual cortex, thus reducing signal/noise ratios in regions mediating contrast sensitivity.

Examination of Figs. 5-7 suggests that these contrast sensitivity losses were confined to lower temporal frequencies, not being found with 10-Hz drifting gratings. It is noteworthy that contrast sensitivity for lower velocities is mediated largely by the subcortical P pathway (Merigan & Katz, 1990; Merigan et al., 1991*b*), which projects predominantly to area V4, while the M pathway, which dominates detection of higher velocity stimuli (Merigan et al., 1991*a*), sends a robust projection to area MT, bypassing area V4. These results suggest that damage to higher level cortical areas may selectively affect sensitivity mediated by lower level visual pathways which terminate strongly in the damaged areas.

One interesting feature of the contrast sensitivity loss found here is that it represents a highly localized retinotopic loss, although it resulted from the lesion of a high-level visual area. The additional losses for color matching and shape discrimination found subsequently were also tightly confined to particular portions of the visual field. This result is consistent with the rather precise physiological retinotopy found in area V4 (Gatass et al., 1988) for classical receptive fields. We found no effects extending across the vertical meridian which would have suggested activity of the large suppressive surrounds of V4 cells (Desimone et al., 1993). It will be interesting to determine if lesions of less precisely retinotopic areas such as TEO (Boussaoud et al., 1991) and MST (Duffy & Wurtz, 1991) produce visual loss specific to particular portions of the visual field.

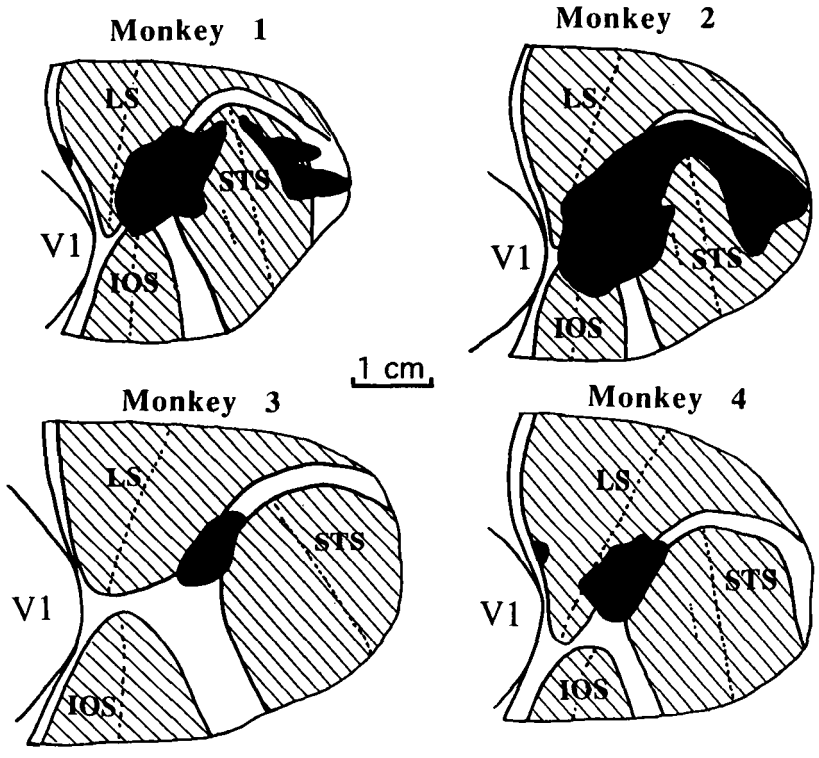

Fig. 3. Reconstruction of lesions for the four monkeys of this study on a flattened representation of a small region of visual cortex (Van Essen & Maunsell, 1980). This approach allows three-dimensional information about cortex obtained from histological sections to be displayed on a two-dimensional map. The heavy solid lines show the borders of the superior temporal sulcus (STS), the lunate sulcus (LS), and the inferior occipital sulcus (IOS), and the dashed lines the primary and secondary (STS only) fundi of the sulci. Striate cortex (V1) is located to the left of the reconstructed areas. Blackened areas represent regions of complete damage to cortex.

The present results for visual acuity showed little effect for three of the monkeys, but a three-fold loss in monkey 3. That this loss was present for orientation discrimination acuity, but not grating detection acuity, shows that the monkey could detect something on the right or left display, but could not tell the orientation of the detected grating. This is the type of effect we would expect if a cortical lesion disrupted veridical perception (which is needed for any sort of shape recognition), but allowed some degraded information such as an aliased signal (Williams, 1985). This large effect on orientation acuity in monkey 3 was not correlated with either a larger lesion or more severe effects on other thresholds. Furthermore, an earlier study (Merigan et al., 1993) had shown that V2 lesions, which would be expected to remove a large part of the neuronal input to area V4, had little effect on visual acuity. When we examined the time course of the V4 lesion effect, we found that all monkeys showed an initial drop in acuity for about two test sessions after the lesions, and then three of the monkeys abruptly returned to near normal acuity. It is possible that the V4 lesion may have resulted in a permanent alteration in the appearance of the test stimuli, but that monkeys 1, 2, and 4 quickly relearned the orientation discrimination despite the altered appearance.

One earlier study (Schiller, 1993) also examined the effects of V4 lesions on luminance contrast sensitivity in one monkey,

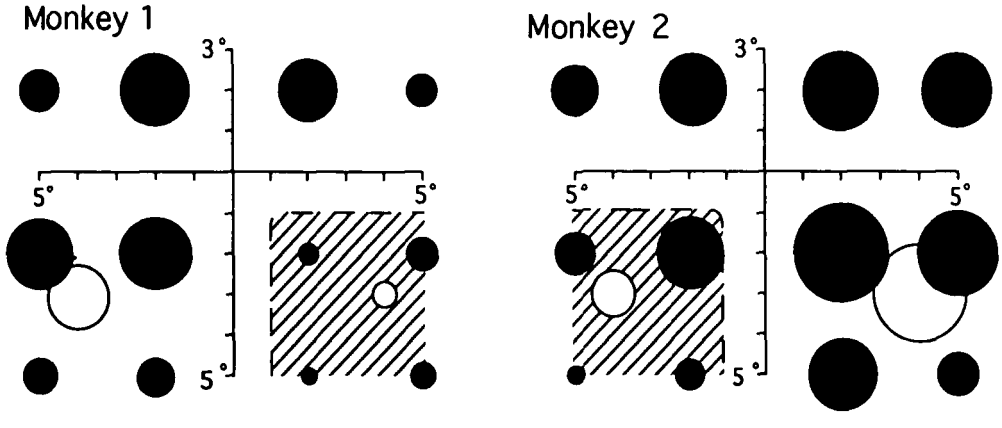

Fig. 4. Luminance contrast sensitivity measured in different visual field locations for monkeys 1 and 2. Sensitivity was measured with stationary patches of 1 cycle/deg grating (Gabor stimuli). Open circles show measures made 3 deg below the horizontal meridian and 4 deg left and right of the vertical meridian, a location where the measures shown in subsequent figures were obtained. Filled circles show measures obtained at other locations. The center of each symbol shows the location tested and its diameter is proportional to the linear contrast sensitivity measured at that location. The hatched region shows the visual field quadrant targeted by the lesions.

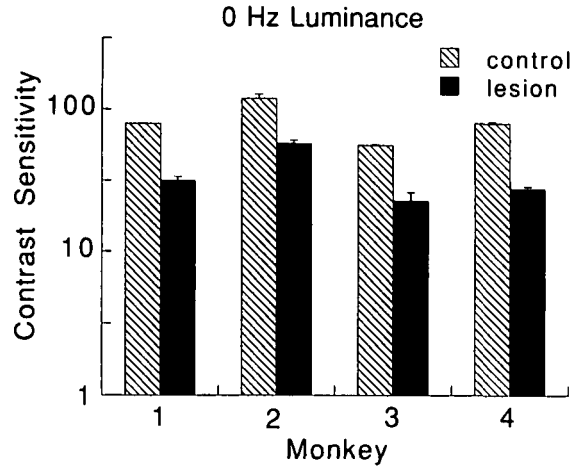

Fig. 5. Luminance contrast sensitivity shown for four monkeys in lesion and control locations. Data for monkeys 1 and 2 were previously shown as the open symbols in Fig. 2. Error bars are ±SEM.

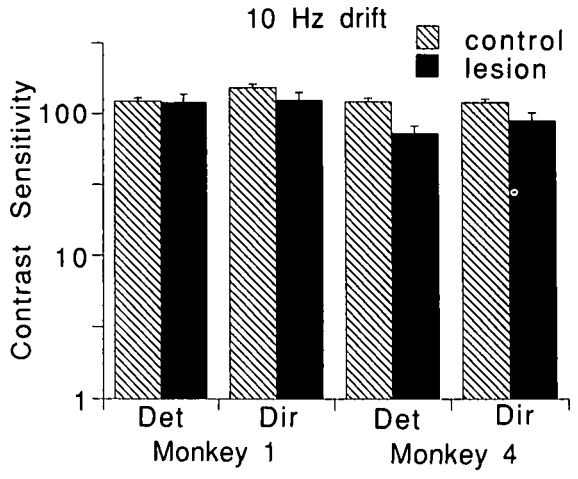

Fig. 7. Luminance contrast sensitivity of two monkeys for detection and direction discrimination tested with patches of 10-Hz drifting grating. Error bars are ±SEM.

although the lack of detailed description makes it somewhat difficult to compare to the present findings. Small checkerboard stimuli with dominant spatial frequencies of 2.3 and 4.15 cycles/deg were presented at an eccentricity of either 2.5 or 5 deg in a detection paradigm (only a single stimulus on the display). The monkey showed a contrast sensitivity loss after the V4 lesion of about 20–25%, a much smaller effect than found in the present study. Compounding the difficulty of comparisons between the studies are the very different contrast thresholds at the non-lesioned location, which were from 1–2% in this study and approximately 7 and 25% in the earlier study, possibly a reflection of the very different stimuli used in the two studies.

Hue matching

The severe disruption of hue matching-to-sample performance seen in monkeys 1 and 3 seemed not to indicate a deficit in color perception. Both monkeys showed no difficulty performing a test of chromatic contrast sensitivity, measured with an orientation discrimination, although all four monkeys did show a two-fold loss of sensitivity on this test. Thus, they had good residual sensitivity to color differences, although it has been pointed out before (Victor et al., 1989) that such sensitivity does not necessarily demonstrate any capability on color identification. The subsequent finding that both monkeys also showed a complete inability to match stimulus luminance indicated that the deficit was probably related to the test procedure and not to the color dimension tested. This was a truly profound deficit, given that the matching to sample procedure was quickly learned in all other parts of the visual field of monkey 1 and in the only other location tested in monkey 3, but we were unable to train performance in the lesion locus, despite such efforts as slowly moving the discriminanda across the vertical meridian.

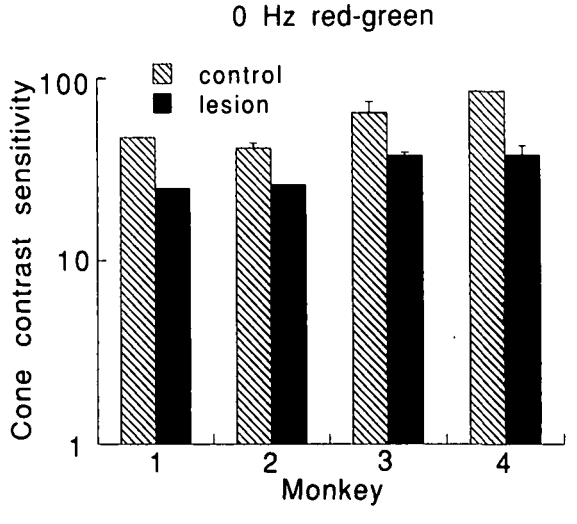

Fig. 6. Chromatic contrast sensitivity measured with a red-green (constant blue) stationary, isoluminant grating patch of 1 cycle/deg for all four monkeys. Error bars show ±SEM.

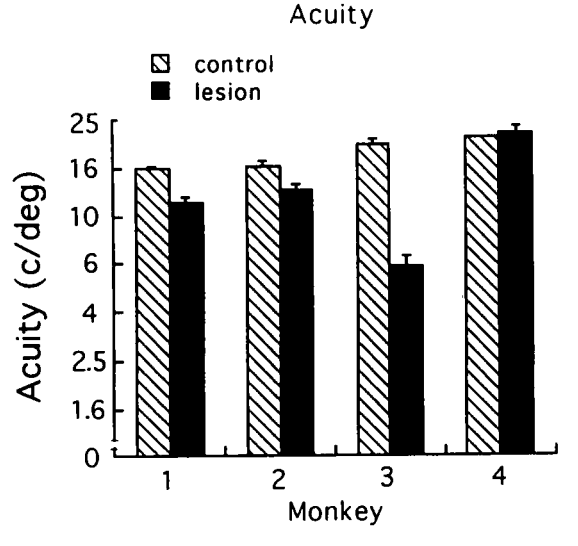

Fig. 8. Visual acuity of the four monkeys tested in the V4 lesion and control locations in the visual field. Error bars show ±SEM.

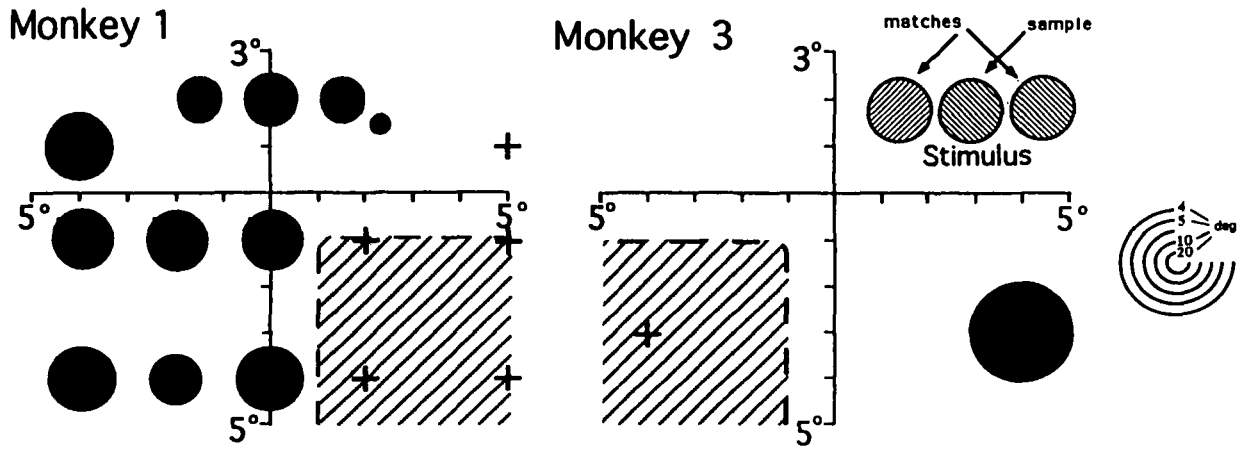

Fig. 9. Results of hue matching for monkeys 1 and 3 at several locations in the visual field. At each location the size of the symbol represents sensitivity to hue (inverse of hue threshold in degrees of difference in the MacLeod-Boynton color space) and its center shows the location tested. The scale to the right of the figure relates symbol size to hue discrimination in degrees. Hatching shows the quadrants targeted by V4 lesions and the + symbols show locations at which hue matching could not be done.

Effects of V4 lesions on chromatic contrast sensitivity has been reported in an earlier study (Schiller, 1993) with chromatic contrast sensitivity for a red-green stimulus reduced slightly less than a factor of two in one monkey and an indeterminate amount in a second. The same study found no effect of V4 lesions on simple color discriminations, including yellow *vs.* red, blue, or green, but a very large effect (Fig. 9) on a hue discrimination almost identical to that used in the present study. This finding appears inconsistent with the present study as well as with the studies of Heywood and colleagues (Heywood & Cowey, 1987; Heywood et al., 1992). It is not clear if the two monkeys from the Schiller study that showed this effect had been trained on the task before the V4 lesion was made, but this was presumably true at least for monkey Zeno, since it had received an MT lesion before the V4 lesion.

When monkey 4 of the present study was pretrained on the matching-to-sample procedure in all four quadrants before the lesion was made, there were no measurable effects of the V4 lesion. Naturally, replication of this finding in future studies will increase our confidence that pretraining was indeed the important variable. However, it seems unlikely that a qualitatively different lesion could have been the basis of the different effects in monkeys 1 and 3 and in monkey 4. No basis for

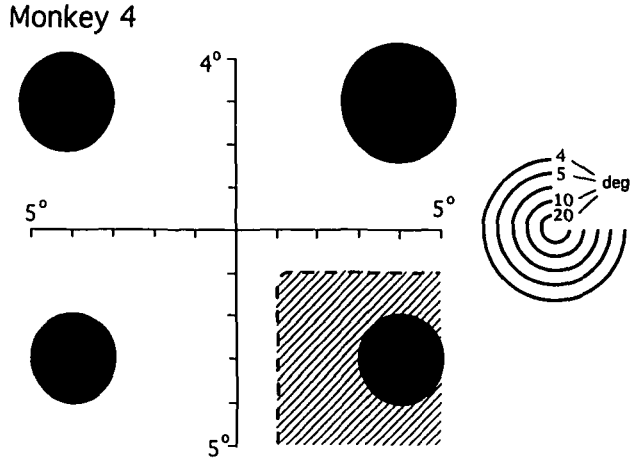

Fig. 10. Results of testing a third monkey on hue matching after pretraining in each quadrant before the V4 lesion. All conventions are the same as in Fig. 11.

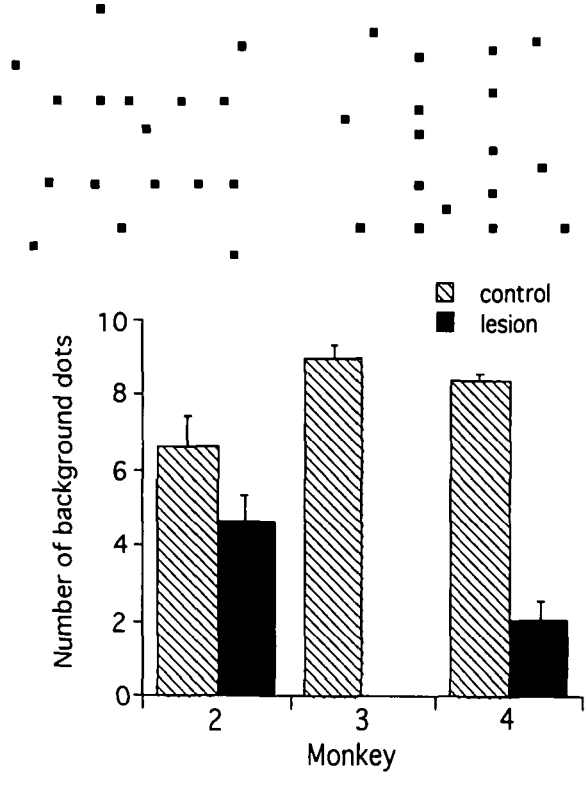

Fig. 11. The number of background dots that brought the discrimination illustrated above the data to threshold performance. Results are shown for three monkeys in control and V4 lesion locations. On each trial only a single stimulus was presented. The stimulus shown to the left above the data has two horizontal lines of dots masked by seven background dots and for this stimulus a left response was correct. The stimulus to the right has two vertical lines of dots masked by seven background dots and for this stimulus a right response was correct. Error bars are ±SEM.

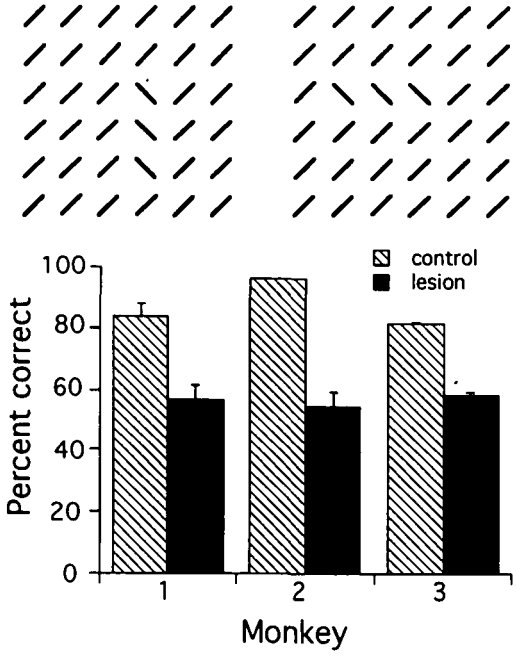

Fig. 12. Percent correct performance for the two monkeys in control and V4 lesion locations for the discrimination task illustrated above the data. The stimulus to the left has a vertical row of right-oblique lines, and that to the right has a horizontal row. The monkey was required to identify the orientation of the row of differently oriented segments. Error bars are ±SEM.

such a difference is evident in a comparison of the extent and locus of the lesions (Fig. 2) in monkey 3 (disrupted matching to sample) and monkey 4 (no disruption), nor did the lesions in different monkeys have very different effects on luminance and chromatic contrast sensitivity (Figs. 5 and 6).

Role of previous learning

The possibility that pre-lesion training would prove to be an important determinant of the effects of V4 lesions was not anticipated in the planning of this study. For this reason, the study was not designed to examine training effects, although the present findings suggest that this may be a fruitful direction for future work. It appears from this study and previous work that there may be two types of V4 lesion effects. The first consists of those visual performances that are disrupted by V4 lesions regardless of pretraining or testing procedure. Alterations in contrast sensitivity appear to fall in this class, since monkey 4 showed reduced contrast sensitivity despite prior training at the lesion locus. However, these effects consisted of rather modest 1.5- to 2-fold reductions, and the basis of these sensitivity changes resulting from lesions of an extrastriate area are not clear. Disruptions of shape discrimination may also be part of this class since several previous studies have shown that complex shape discriminations, including discriminations between faces (Heywood & Cowey, 1987; Heywood et al., 1992; Walsh et al., 1992), are thoroughly disrupted by V4 lesions despite pretraining.

A second class of V4 lesion effects are those in which disruptions are evident only when the animal was not pretrained on the discrimination task at the visual field locus subsequently lesioned. This is the type of effect seen in the total loss of hue matching in monkeys 1 and 2 after V4 lesions, in contrast to the preservation of such matching in monkey 4 after prelesion training on the color matching-to-sample. Such an effect would not have been seen in most previous studies, because discriminations are typically trained at all tested locations before the lesions were made. Schiller (Schiller & Lee, 1991) reported what appears to be an analogous effect, in which the learning of visual discriminations was retarded after V4 lesions and each additional discrimination required thousands of trials for improvement. Unfortunately, this long-term improvement is not entirely unambiguous, since it is logically possible to perform well on the oddity task if only one of the stimuli falls into the lesion locus, and thus cannot be discriminated. In such a case, the task of the monkey might become simply to saccade to the lesion locus if none of the visible stimuli look different.

One issue of interest was the question of what needed to be learned prior to the lesion to prevent the deleterious effect of V4 lesions on post-lesion performance. Monkey 4 was pretrained only on color discrimination with the matching-to-sample task, and the results in Fig. 10 show no effect of the V4 lesion on this performance. However, despite this narrow pretraining, monkey 4 was also able to perform matching-to-sample for untrained stimulus features including luminance and simple two-dimensional shapes (e.g. triangle *vs.* circle). One issue for future studies will be the nature of the pretraining effect; is it necessary (as this result suggests) only to train the monkey on a procedure (matching-to-sample), but not on the specific stimuli to be discriminated?

Disruption of shape discrimination

V4 lesions had their most devastating effects on the two shape discriminations involving the orientation of lines of colinear dots and groups of oriented line segments. These abilities were completely eliminated in most cases, a result similar to that we obtained in a previous study after V2 lesions (Merigan et al., 1993). The fact that these functions were disrupted by both V2 and V4 lesions is consistent with the notion that the V2 to V4 projection is important to these abilities, although cortical areas to which V4 projects may also be involved. It will be important in future studies to show that lesions of other cortical areas (MT, FST, etc.) do not also disrupt these capabilities.

We do not know if these discriminations would have been less affected if the monkeys had been pretrained on them at the lesion location, as we later found for color discrimination. Only monkey 3 was pretrained on these tasks in the quadrant of the visual field that later received a lesion, and this monkey, as can be seen in Fig. 8, had an acuity loss that could by itself have accounted for part of the loss. Our confidence in the specificity of the deficit for grouping tasks was supported by our failure to find a defect in global motion perception in monkey 4, despite a lack of prelesion training on this discrimination. The global motion discrimination is affected by MT/MST lesions (Pasternak & Merigan, 1994).

Previous studies have also found effects of V4 lesions on shape discriminations. Walsh and colleagues (Walsh et al., 1992) tested monkeys on 17 pairwise shape discriminations, most of which were letters or two-letter groups which differed in orientation. They found that V4 lesions resulted in up to 300 additional errors before the monkeys reached criterion performance (nine out of ten correct on ten consecutive test trials) on the pre-

viously learned discriminations. Thus, they found only moderate disruptions in performance, quite different from what the present study found for the two grouping tasks. More recently, Schiller (1993) reported large effects on two of three discriminations of simple geometric shape discriminations in two monkeys (his Fig. 13). This result appears inconsistent with our observation in monkey 4 (above) that after a V4 lesion it could match simple geometric shapes (e.g. circle *vs.* triangle) with no difficulty. Further studies will be needed to determine if this difference in results was due to differences between the two-alternative choice procedure used here and the eight-alternative procedure used by Schiller.

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