Direction of motion discrimination after early lesions of striate cortex (V1) of the macaque monkey

Tirin Moore*, Hillary R. Rodman†, and Charles G. Gross

Department of Psychology, Green Hall, Princeton University, Princeton, NJ 08544-1010

Contributed by Charles G. Gross, October 30, 2000

Previous studies have established that humans and monkeys with damage to striate cortex are able to detect and localize bright targets within the resultant scotoma. Electrophysiological evidence in monkeys suggests that residual vision also might include sensitivity to direction of visual motion. We tested whether macaque monkeys with longstanding lesions of striate cortex (V1), sustained in infancy, could discriminate visual stimuli on the basis of direction of motion. Three monkeys with unilateral striate cortex lesions sustained in infancy were tested 2–5 years postlesion on a direction of motion discrimination task. Each monkey was trained to make saccadic eye movements to a field of moving dots or to withhold such eye movements, depending on the direction of motion in a coherent random dot display. With smaller motion displays, monkeys were unable to detect or discriminate motion within the scotoma, although they could discriminate moving from static stimuli. Yet, each monkey was able to discriminate direction of motion when the motion stimulus was larger, but still confined to the scotoma. The results demonstrate that the recovery after infant damage to striate cortex includes some sensitivity to direction of visual motion.

motion perception | recovery of function | blindsight | saccadic eye movements

Humans and monkeys with damage to V1 retain or recover the ability to detect and localize visual targets within the scotoma (1–6). In both species, the vision that survives V1 damage often depends on the mode of testing, typically requiring forced-choice or similar paradigms (6–8), and there is considerable variability in the extent and nature of the residual vision, particularly in humans. One factor that may contribute to the variability in the extent of residual vision observed across subjects with V1 damage is the age at which the lesion is sustained (9). We previously have shown that monkeys with lesions of V1 in infancy demonstrate greater residual vision than their adult-lesion counterparts, as measured by their ability to detect and localize visual stimuli within the scotoma (10). The residual vision after lesions in infancy is robust and does not depend on the type of testing paradigm as it does after adult lesions (11). Other studies in this laboratory have found that many neurons within extrastriate area MT retain their selectivity for the direction of a moving bar in the absence of V1 input (12), suggesting that destriate vision might include some residual motion sensitivity. In the present study, we trained monkeys with longstanding unilateral lesions of V1 on a direction of motion discrimination task and then tested them both inside and outside of the scotoma. Our results indicate that some ability to discriminate direction of motion survives lesions of V1 in monkeys, at least when the damage is sustained early in life.

Methods

Subjects. Three female *Macaca fascicularis* monkeys, weighing between 2 and 5 kg, were used as subjects. Each monkey had received large or total unilateral lesions of striate cortex 5–7 weeks after birth. Each of the three lesion subjects had previously been tested extensively in a perimetry paradigm, in which their ability to detect and localize small visual targets was measured (10). Each monkey was able to detect and localize visual targets presented within the hemifield contralateral to the lesion.

Striate Cortex Lesions. Lesions of striate cortex were made under strict aseptic conditions using procedures similar to those described (12). After pretreatment with atropine (0.08 mg/kg i.m.) and dexamethasone phosphate (0.8 mg/kg i.m.), the animals were restrained with a mixture of ketamine hydrochloride (10 mg/kg) and acepromazine (1 mg/kg). Anesthesia was maintained with this mixture, supplemented as needed (13). After removal of the overlying bone, the striate cortex was removed by subpial aspiration in two stages aided by a Zeiss operating microscope. First, the cortex on the dorsolateral and medial surfaces of the hemisphere was removed. Next, striate cortex on the dorsal and ventral "leaves" and banks of the calcarine fissure was removed by exposing the calcarine by removing the roof of the sulcus underlying the operculum. Although this approach makes it more difficult to remove striate cortex in its entirety than does a lobectomy procedure, it has the advantage of removing considerably less of the extrastriate tissue that may contribute to residual capacity. The dural flap made to expose the cortex was then sutured, and the soft tissues and skin were likewise sutured and the animal was allowed to recover. Animals I-1 and I-3 received left striate cortex ablations, whereas I-2 received a lesion of the right striate cortex.

Histology. At the conclusion of all experiments, each animal was anesthetized with a lethal dose of i.v. sodium pentobarbital and perfused transcardially with saline, followed by 4% paraformaldehyde in phosphate buffer. The brains were blocked in a stereotaxic apparatus and sectioned in either the parasagittal (monkeys I-1, I-2) or the coronal plane (I-3) with a freezing microtome at 50 μm. Separate series of sections were stained with cresyl violet or Gallyas myelin. Histological analysis of the unoperated hemisphere was unavailable in monkey I-3 because this monkey developed a severe infection just before the histological phase of the study. The infection affected only the unoperated hemisphere and began more than a year after the behavioral experiments.

The extent of the visual field representation destroyed in the striate cortex was estimated in two ways. First, the extent of striate cortex damage was evaluated from the cortex itself using published accounts of striate cortex topography and the intact hemisphere where possible as a guide to approximating the convolutions of the tissue removed in the lesioned hemisphere (14, 15). Second, zones of degeneration in the dorsal lateral

Abbreviation: dLGN, dorsal lateral geniculate nucleus.

*To whom reprint requests should be addressed. E-mail: tirin@princeton.edu.

†Present address: Department of Psychology and Yerkes Regional Primate Research Center, Emory University, Atlanta, GA 30322.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Article published online before print: *Proc. Natl. Acad. Sci. USA*, 10.1073/pnas.021519698. Article and publication date are at www.pnas.org/cgi/doi/10.1073/pnas.021519698

geniculate nucleus (dLGN) were charted and compared with standardized maps of visual topography in the dLGN based on the work of Malpeli and colleagues (16, 17). For both methods, the estimated error was about 1° within the central 7° of the representation and increased to about 5–10° by about 40° eccentricity. To obtain a conservative measure of the lesions (i.e., to err on the side overestimating, rather than underestimating, the amount of spared tissue), the field defects were plotted from the envelope of the two measures of sparing at each point of the representation.

Behavioral Testing. During testing, the monkey sat in a primate chair 57 cm away from the visual display. After each monkey was trained to fixate a central spot of light (0.5° diameter) for a duration of 1 s, it was trained on an oculomotor go/no-go discrimination task. In this task, the monkey was required to make saccadic eye movements to one stimulus (S+ or positive stimulus) and to withhold such eye movements to another (S− or negative stimulus). The stimuli were presented on separate trials at the same visual field location. Saccadic eye movements to the S+ were rewarded with a drop of juice when they landed within a large error window (20° in diameter) centered on the stimulus. A reward followed the S− stimulus when the monkey maintained fixation on the fixation spot within a 4° × 4° error window for the duration of the 1-s stimulus presentation. The two trial conditions were pseudorandomly interleaved and could occur in differing proportions of the total trials. Typically, there were three times as many S− trials as there were S+ trials. This was done because the monkeys generally had more difficulty withholding eye movements than eliciting them. The addition of "correction" trials after incorrect responses prevented the monkey from simply saccading on every trial or never saccading. A correction trial was a repetition of the previous trial condition that followed each incorrect response; these trials continued until the monkey responded appropriately. Each monkey quickly learned to reverse its response after each incorrect trial and thus seldom required more than a single correction trial. Responses during correction trials were not included in the data analysis. All behavioral testing was performed monocularly; each monkey was fitted with an eye patch covering the eye contralateral to the striate lesion. During testing, eye position was monitored via a scleral search coil.

Each monkey was first trained on the discrimination task in the hemifield ipsilateral to the lesion such that when the S+ and S− were very different stimuli (e.g., moving dots vs. static dots), they were rewarded on more than 85% of the trials. When the task was moved into the hemifield contralateral to the striate lesion, the monkey was first tested to a criterion level of performance on a discrimination of moving vs. static dots to ensure that they could actually perform the go/no-go task within the scotoma. The criterion was a >70% performance on both the S+ and S− trials. Once the direction discrimination began, each monkey was tested until it reached the criterion performance, after which it was tested on a series of 4–8 (usually eight) postcriterion blocks, at 48 trials/block, not including correction trials. If the monkey did not reach criterion, it was tested for a minimum of 1,008 trials, or 21 blocks. In the event that the monkey performed at chance (50%) for >5 blocks, the discrimination was reverted back to one the animal could perform until criterion was reached again on the easier discrimination. The monkey then resumed testing on the harder discrimination.

Motion Stimuli. Motion stimuli consisted of circular fields (5° or 15° diameter) of moving dots generated from the presentation of 50–55 successive frames of 20–25 dots (0.1–0.3° diameter) on a flat-screen video monitor (Zenith, 26.0 × 19.5 cm, 50 Hz). The luminance of each dot in the display was 87.5 cd/m^2 on a scotopic background of 0.13 cd/m^2 (Minolta photometer); thus, the contrast of each dot was 2.8 log units. The location of each dot in the first frame was random. In each subsequent frame, all dots had an assigned probability (0–100%) of being plotted in a specified direction (θ) at a constant displacement (Δr). That is, each dot, when replotted, could provide a displacement signal coherent with the previous displacement. During any two successive frames, if a dot was not replotted coherently, it was replotted at a random location within the stimulus aperture (18). The density of dots was approximately 7.1 dots per degree per s. Motion stimuli used in the present experiments contained either 98% (fully coherent motion) or 0% (motion "noise"). We chose 98% as the fully coherent motion stimulus over 100%, as it was our observation that the former contained considerably less rigid "pattern" motion but still contained a near maximal amount of motion signal.

Analysis of Operant Data. Discrimination performance was based on the degree to which the monkey could choose the correct trial on which to initiate a saccadic eye movement to the motion display. The monkey's performance was therefore the average of the percent correct on the S+ and S− trials, regardless of whether the trial types occurred at different frequencies. To determine whether or not the monkey could discriminate between the positive and negative stimuli above that expected by chance, the number of total saccadic eye movements made to the two stimuli was compared in a 2 × 2 contingency table analysis.

Results

Histological Findings. Figs. 1–3 show the histological reconstruction of the striate cortex lesions. Fig. 4*A* shows a reconstruction of the field defect of each animal and the position of the motion aperture within it. In all cases, the borders of the cortical lesions were clearly marked by gliosis along the margin; residual striate tissue, when present, was easily recognized by its preserved characteristic pattern of lamination. Similarly, zones of retrograde cell degeneration in the dLGN were clearly demarcated, although there was considerable variability in the degree to which the nucleus showed a distortion in overall shape relative to its intact counterpart. In each case, occasional large neurons were found scattered through the otherwise degenerated zones, presumably reflecting a surviving small projection to extrastriate areas on the prelunate gyrus (19, 20).

Case I-1 (Fig. 1). In this case, all of striate cortex on the dorsolateral, medial, and ventral surfaces and calcarine fissure was removed with the exception of some tissue on both banks of the anterior half of the calcarine. The most medial and lateral portions of anterior calcarine were removed as intended; in addition, there was limited invasion of the lesion into the posterior banks of the inferior occipital and lunate sulci. The summary field defect (Fig. 4*A*) shows a largely complete lesion, with sparing restricted to the representation of two swaths of the visual field beyond 25° eccentricity.

Case I-2 (Fig. 2). No residual striate cortex was visible in any portion of the lesioned hemisphere in this case. The dLGN showed no zones of sparing. Some invasion of the posterior banks of the inferior occipital and lunate sulci was present. The summary field defect (Fig. 4*A*) shows a complete lesion.

Case I-3 (Fig. 3). In this case, striate cortex removal was complete except for two restricted zones of sparing. The first was comparable to the sparing observed in I-1, involving portions of both banks of the anterior half of the calcarine fissure, corresponding to the representation of a swath of visual field beyond approximately 35° eccentricity. The second was a small region of the ventromedial margin of the occipital pole, corresponding to a small crescent within the upper central 10°. Both zones of sparing are shown in the summary field defect in Fig. 4*A*. Minimal involvement of the inferior occipital and lunate sulci

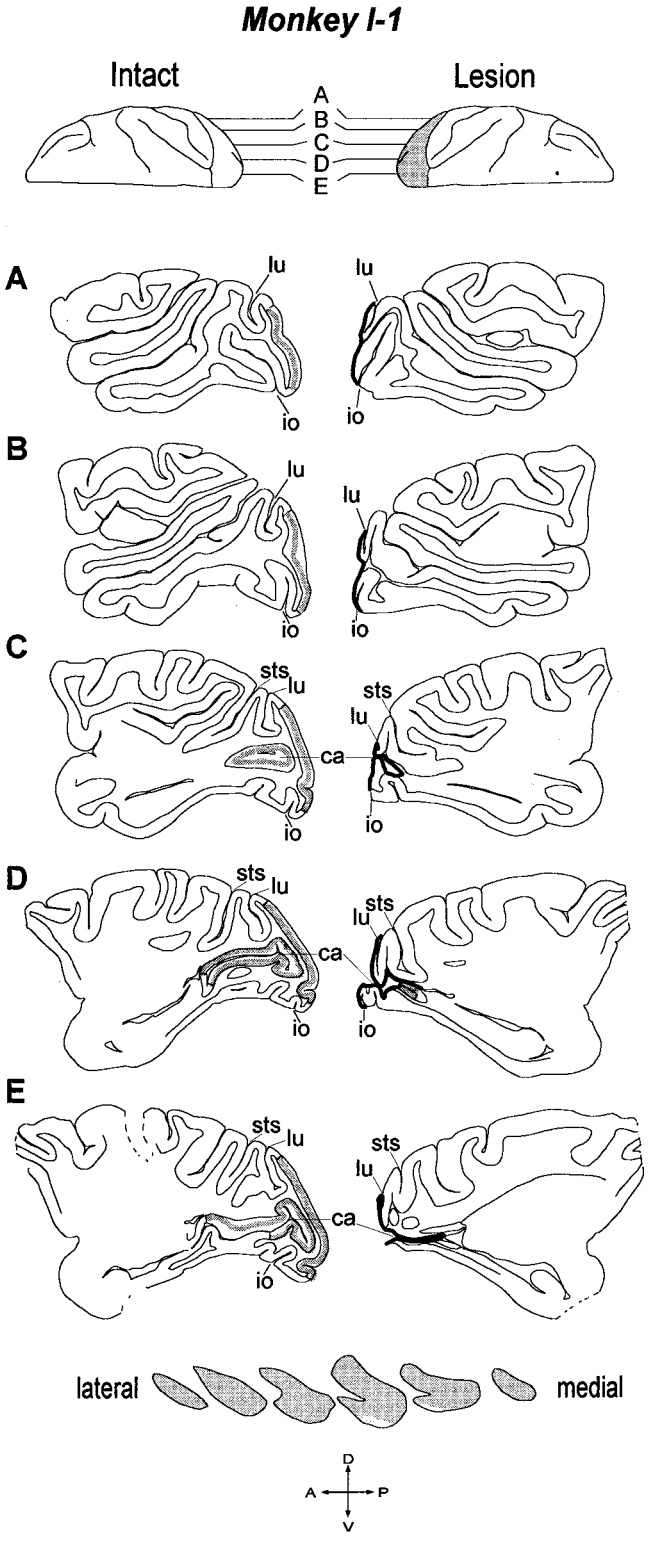

Fig. 1. Serial reconstruction of the striate cortex lesion in monkey I-1. Sagittal sections through the cortex of this animal show the intact (*Left*) and operated (*Right*) hemisphere. Striate cortex is indicated (shading) in the intact hemisphere (*Left*) localized by its characteristic lamination. The bold lines in the operated hemisphere (*Right*) show the borders of the lesion. The diagram of the dorsal views of the two hemispheres at the top indicates the level of section shown in the sagittal series, from lateral to medial (*A–E*). Representative sagittal sections through the ipsilesional dLGN show the zones of degeneration (gray). Abbreviations in this and other figures are: a, anterior; p, posterior; d, dorsal; v, ventral; ca, calcarine; lu, lunate; io, inferior occipital; ip, intraparietal; ot, occipitotemporal; sts, superior temporal.

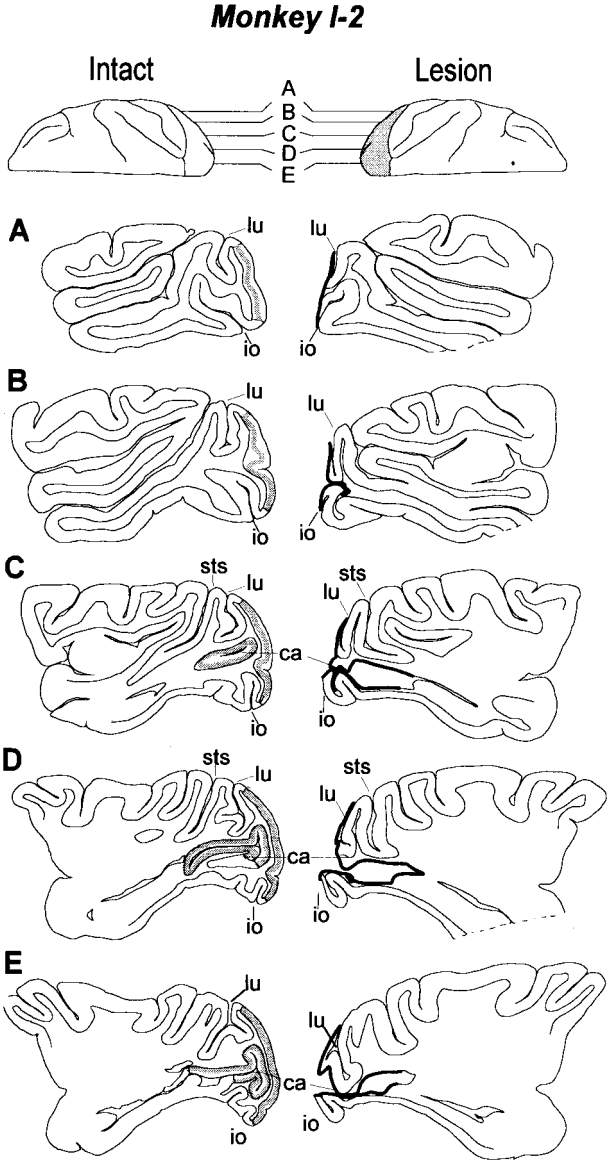

Fig. 2. Serial reconstruction of the striate cortex lesion in monkey I-2. Sagittal sections through the cortex of this animal show the intact (*Left*) and operated (*Right*) hemisphere. Degeneration of the ipsilesional dLGN was complete in this animal.

was also present. Degeneration in the dLGN was clearly demarcated and consistent with the cortical findings.

Motion Discrimination. We initially tested each monkey using small (5° diameter) motion apertures. Each monkey could discriminate moving from static dots in the intact visual field as well as within the scotoma. However, when the monkeys were required to discriminate coherently moving dots from motion "noise" (0% correlated motion), the performance of each monkey was at or near chance within the scotoma. When the task was changed further to a discrimination of upward from downward motion, each monkey performed at chance within the scotoma. In the intact field, performance on both types of discrimination was similar (Fig. 4*B*).

With the small motion display, monkeys with V1 lesions were clearly unable to discriminate direction of motion. However, evidence from previous studies suggests that residual motion

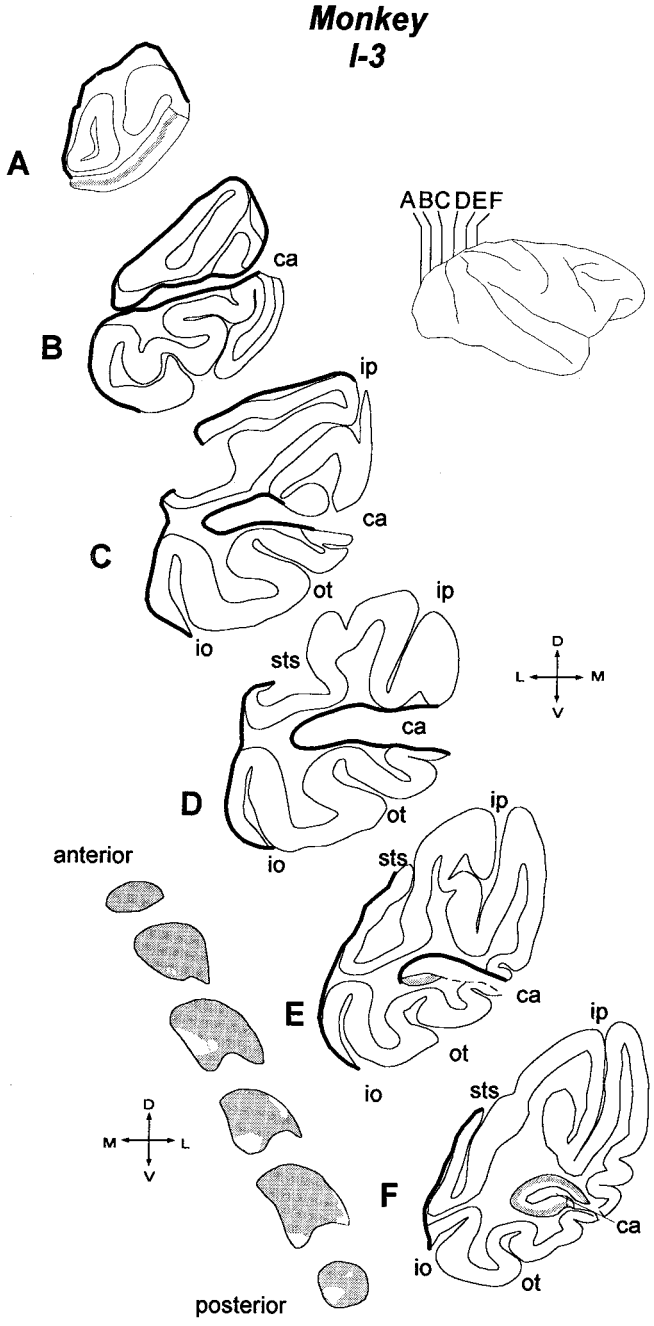

Fig. 3. Serial reconstruction of the striate cortex lesion in monkey I-3. Coronal sections through the cortex of this animal show operated hemisphere only (see *Materials and Methods*). The diagram of the lateral view of the cortex at the top indicates the level of section shown in the coronal series, from caudal to rostral (*A*–*E*). Representative coronal sections through the ipsilesional dLGN show the zones of degeneration (gray).

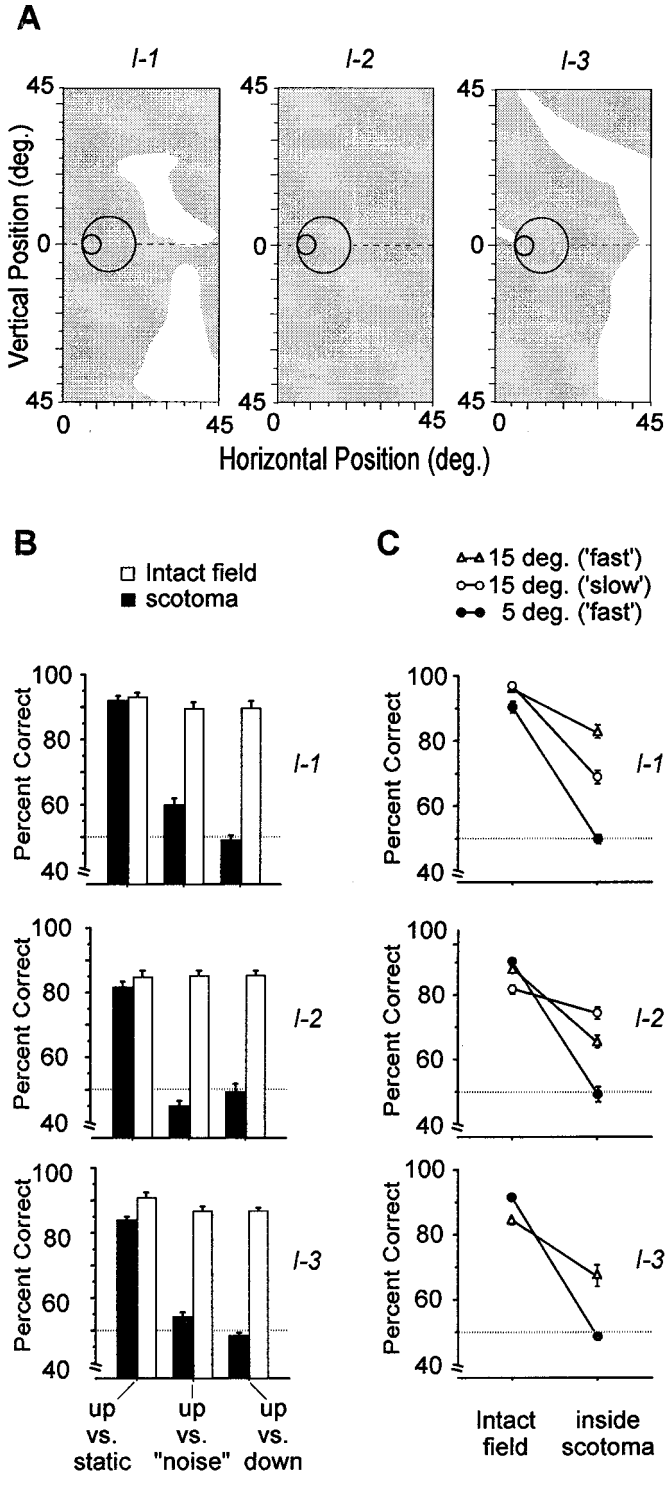

Fig. 4. (*A*) Position of the stimulus aperture within the visual defects of each monkey with a unilateral striate cortex lesion (I-1, I-2, I-3, and A-3). Unshaded areas represent zones of the visual field with corresponding intact striate cortex. Circles show the positions of the 5° and 15° motion apertures within the reconstructed field defect. (*B*) Performance of monkeys with infant V1 lesions on the discrimination of moving from static, coherently moving from motion ''noise'' and upward from downward motion. Motion stimuli were presented in a 5° aperture. Speed of motion was 20°/s. The gray line indicates performance expected by chance. (*C*) Performance of the same monkeys on the discrimination of upward from downward motion when the motion stimuli were presented in 5° or 15° apertures. Speeds of motion were 4°/s (''slow'') or 20°/s (''fast'').

sensitivity requires the summation of motion signal over large distances within the scotoma (21). Therefore, we next tested whether each monkey could discriminate direction of motion of dots when the dot display was much larger (15° diameter) but still within the scotoma. To do this, we enlarged the original motion display to a 15° aperture by rear-projecting it onto a tangent screen. The projected display was centered on the horizontal meridian at 13.5° from the fixation point and spanned from 6° and 21°, thus still within the scotoma of each monkey (see Fig. 3). Like the size of the aperture, the size of each dot in the motion display increased from 0.1° to 0.3°.

With the larger display, each monkey was able to discriminate direction of motion within the scotoma (Fig. 4*C*). The performance of each monkey within the scotoma was poorer than in the intact field (Scheffé, $P < 0.001$). However, in both the intact field and within the scotoma, saccades were disproportionately elicited to upward stimuli, indicating that the monkeys could distinguish between the two directions of motion (ipsilateral: $\chi^2 = 331$, $P < 0.0001$, I-1; $\chi^2 = 195$, $P < 0.0001$, I-2; $\chi^2 = 152$, $P < 0.0001$, I-3; contralateral: $\chi^2 = 141$, $P < 0.0001$, I-1; $\chi^2 = 98$, $P < 0.0001$, I-2; $\chi^2 = 64$, $P < 0.0001$, I-3).

Two of the lesion subjects (I-1 and I-2) were tested further with a slower speed of motion (4°/s). With a slower speed, both of the monkeys with early lesions could discriminate the direction of motion within the scotoma. As with the faster speed, saccades were disproportionately elicited to upward stimuli in both hemifields, indicating that the monkeys could distinguish between the two directions of motion (ipsilateral: $\chi^2 = 289$, $P < 0.0001$, I-1; $\chi^2 = 146$, $P < 0.0001$, I-2; contralateral: $\chi^2 = 122$, $P < 0.0001$, I-1; $\chi^2 = 129$, $P < 0.0001$, I-2).

Spatial Heterogeneity of Motion Sensitivity Within the Scotoma. We next examined the degree to which the size and position of the motion stimulus within the estimated scotoma affected the motion sensitivity of the infant-lesion animals. This was done by masking either the inner or the outer half of the 15° motion aperture and retesting two of the infant-lesion animals, I-1 and I-2. The motion aperture therefore consisted of a semicircular field of moving dots presented between 6° and 13.5° or 13.5° and 21° along the horizontal meridian (Fig. 5).

The performance was variable within the scotoma in both monkeys tested in the masking experiment, yet both animals could distinguish between upward and downward motion in either the inner or outer half of the motion aperture. When only the inner half of the motion aperture was visible, monkey I-1 performed the discrimination poorly, averaging only 60.5% correct, although it could clearly distinguish between the two opposing directions of motion ($\chi^2 = 23$, df = 1, $P < 0.0001$). In contrast, this monkey's performance was much better when only the outer half was visible (82.5% correct). For monkey I-2, the results were reversed. Although this monkey could discriminate between the opposing directions of motion in both the inner and outer halves of the aperture (inner, $\chi^2 = 112$, df = 1, $P < 0.0001$; outer, $\chi^2 = 35$, df = 1, $P < 0.0001$), its performance was much better in the inner half (Scheffé, $P < 0.005$).

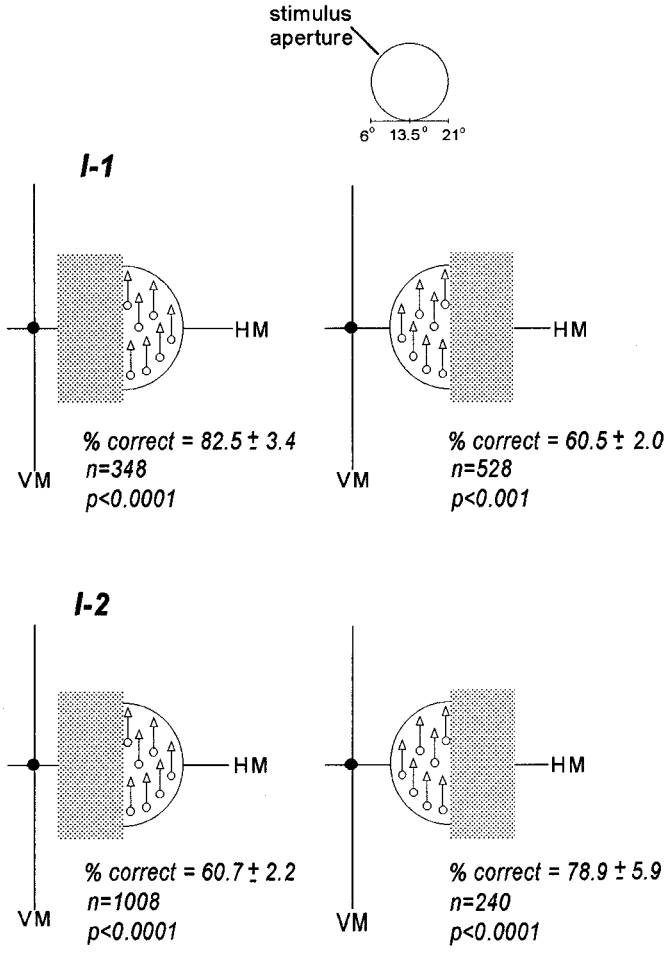

Fig. 5. Discrimination of upward from downward moving dots when either the inner (*Left*) or outer (*Right*) half of the stimulus aperture was occluded. (*Top*, *Inset*) Full horizontal extent of the stimulus aperture when neither half is occluded. Each of the two diagrams for monkeys I-1 and I-2 shows which of the two halves of the stimulus was occluded and the resultant performance. The occluder, shown in this figure, was not visible to the monkey. VM, vertical meridian, HM, horizontal meridian.

Discussion

"Pure" Motion Discrimination. Random dot motion displays have proven to be useful tools in assessing motion perception mechanisms, both in physiological (18) and psychophysical (22) experiments, primarily because they greatly minimize the "familiar position cues" from which motion, and its direction, can be inferred (23). Unlike a single moving stimulus, a random dot field does not vary its position over time. Dots within the display disappear at one edge and appear at another, and the entire field of dots maintains a constant position. This does not, however, rule out the use of strictly local signals in judging direction from random dot displays. In a 98% correlated motion display, virtually all of the dots continue in coherent trajectory throughout the display period. Although it may have been difficult for each monkey to follow only a single dot in the field of moving dots, particularly within the field defect, we cannot rule out the possibility that their discrimination of direction of motion was made solely on the basis of a single dot. Instead, we can only emphasize that the use of either global or local cues requires an amount of residual visual function not previously expected in the absence of V1.

Mechanisms of Recovery. The lesion reconstructions revealed that the contralateral hemifield stimuli were located at positions that did not overlap with spared visual representations in striate cortex. However, we cannot exclude a role for those spared portions in residual vision. We cannot rule out, for instance, the possibility that spared "islands" of striate cortex undergo plastic changes as a result of the ablation and that the remnant visuotopy shifts to include the removed visual field. Indeed, there is evidence of map reorganization after cortical lesions (24–26). This reorganization presumably results from both cortical and subcortical plastic mechanisms and, in the visual system, might include changes at the level of the dLGN as well as within striate cortex. Such a possibility awaits experimental testing, but our results with monkey I-2, an animal with a complete striate lesion, show that spared representations within striate cortex, however plastic, are not necessary for the residual visual capacities observed.

The fact that monkeys with early V1 lesions could discriminate direction of motion in the scotoma is remarkable given the functional importance of the geniculostriate pathway in primate vision and the extensive anatomical degeneration that takes place after damage to primary visual cortex. Damage to V1 in both infant and adult monkeys results in rapid retrograde

degeneration of approximately 98% of neurons in the dLGN and takes place within months of the damage (27). After a lesion of V1 in infancy, the transneuronal cell loss among retinal ganglion cells proceeds faster than after a lesion in adulthood, reaching approximately 80% loss after 1–2 years in the infant case and up to 8 years in the adult case (28, 29). The fact that monkeys used in this study were tested 2–5 years after the lesion suggests that their visual performance within the scotoma was based on a remaining 20% of retinal ganglion cells.

Previous electrophysiological and lesion experiments have provided evidence that pathways involving the superior colliculus are those responsible for most of the residual vision in the absence of V1. The visual activity in areas MT and STP that survives removal of V1 input is eliminated by subsequent damage to the superior colliculus (30, 31). Likewise, the recovery of detection and localization behavior after lesions of V1 is eliminated by subsequent lesions of the superior colliculus (4), at least when the lesions are sustained in adulthood. It is therefore likely that the residual visual behavior observed in the present study is largely because of the remnant tectofugal pathways and particularly those that project to extrastriate visual areas, namely the tecto-pulvinar-extrastriate and tecto-geniculate-extrastriate pathways. Moreover, the wealth of evidence from studies of the cat visual system suggests that after visual cortical lesions in infancy, these pathways are stabilized after damage to the geniculostriate pathway and are thus more extensive in maturity (32, 33). Our previous demonstration of greater residual vision after early damage (10), together with the present results, suggests that the visual system that remains after an early lesion differs extensively from that remaining after a lesion in adulthood. Not only does early damage in monkeys spare some ability to discriminate direction of motion, but the demonstration of this ability, and the ability to localize visual targets, does not require testing paradigms akin to forced-choice, as is the case after adult damage (6, 7).

This work was supported by National Science Foundation Grant BNS-9109743 and National Institutes of Health Grants MH-19420, MH-12336, and EY-11374.

1. Cowey, A. & Weiskrantz, L. (1963) *Q. J. Exp. Psychol.* **19,** 232–245.
2. Poppel, E., Held, R. & Frost, D. (1973) *Nature (London)* **243,** 295–296.
3. Humphrey, N. K. & Weiskrantz, L. (1967) *Nature (London)* **215,** 595–597.
4. Mohler, C. W. & Wurtz, R. H. (1977) *J. Neurophysiol.* **40,** 74–94.
5. Segraves, M. A., Goldberg, M. E., Deng, S. Y., Bruce, C. J., Ungerleider, L. G. & Mishkin, M. (1987) *J. Neurosci.* **7,** 3040–3058.
6. Moore, T., Rodman, H. R., Repp, A. B. & Gross, C. G. (1995) *Proc. Natl. Acad. Sci. USA* **92,** 8215–8218.
7. Cowey, A. & Stoerig, P. (1995) *Nature (London)* **373,** 247–249.
8. Weiskrantz, L. (1996) *Blindsight: A Case Study and Implications* (Oxford Univ. Press, New York).
9. Payne, B. R., Lomber, S. G., Macneil, M. A. & Cornwell, P. (1996) *Neuropsychologia* **34,** 741–774.
10. Moore, T., Rodman, H. R., Repp, A. B., Gross, C. G. & Mezrich, R. S. (1996) *J. Neurophysiol.* **76,** 3928–3933.
11. Moore, T., Rodman, H. R. & Gross, C. G. (1998) *Neuroscientist* **4,** 227–230.
12. Rodman, H. R., Gross, C. G. & Albright, T. D. (1989) *J. Neurosci.* **9,** 2033–2050.
13. Rodman, H. R. (1991) *J. Neurosci. Methods* **38,** 209–222.
14. Gattass, R., Sousa, A. P. & Gross, C. G. (1988) *J. Neurosci.* **8,** 1831–1845.
15. Van Essen, D. C., Maunsell, J. H. & Bixby, J. L. (1981) *J. Comp. Neurol.* **199,** 293–326.
16. Malpeli, J. G. & Baker, F. H. (1975) *J. Comp. Neurol.* **161,** 569–594.
17. Malpeli, J. G., Lee, D. & Baker, F. H. (1996) *J. Comp. Neurol.* **375,** 363–377.
18. Newsome, W. T. & Pare, E. B. (1988) *J. Neurosci.* **8,** 2201–2211.
19. Cowey, A. & Stoerig, P. (1989) *Exp. Brain Res.* **75,** 631–638.
20. Rodman, H. R., Sorenson, K. M., Shim, A. & Hexter, D. P. (2001) *J. Comp. Neurol.*, in press.
21. Weiskrantz, L., Barbur, J. L. & Sahraie, A. (1995) *Proc. Natl. Acad. Sci. USA* **92,** 6122–6126.
22. Baker, C. L., Jr., Hess, R. F. & Zihl, J. (1991) *J. Neurosci.* **11,** 454–461.
23. Nakayama, K. & Tyler, C. W. (1981) *Vision Res.* **21,** 427–433.
24. Glees, P. & Cole, J. (1950) *J. Neurophysiol.* **13,** 137–148.
25. Jenkins, W. M. & Merzenich, M. M. (1987) *Prog. Brain Res.* **71,** 249–266.
26. Yamasaki, D. S. & Wurtz, R. H. (1991) *J. Neurophysiol.* **66,** 651–673.
27. Cowey, A. & Stoerig, P. (1991) *Trends Neurosci.* **14,** 140–145.
28. Dineen, J. T. & Hendrickson, A. E. (1981) *Invest. Ophthalmol. Visual Sci.* **21,** 749–752.
29. Weller, R. E. & Kaas, J. H. (1989) *Visual Neurosci.* **3,** 327–349.
30. Bruce, C. J., Desimone, R. & Gross, C. G. (1987) *J. Neurophysiol.* **55,** 1057–1075.
31. Rodman, H. R., Gross, C. G. & Albright, T. D. (1990) *J. Neurosci.* **10,** 1154–1164.
32. Payne, B. R. & Lomber, S. G. (1998) *Exp. Brain Res.* **121,** 334–349.
33. Payne, B. R. & Cornwell, P. (1994) *Trends Neurosci.* **17,** 126–130.