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CHAPTER 19

Visually guided behavior after V1 lesions in young and adult monkeys and its relation to blindsight in humans

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Abstract: After lesions of striate cortex in primates, there is still the capacity to detect and localize visual stimuli. In this chapter we review three aspects of our study of this phenomenon in macaques. First, we found that macaques that received their striate lesions as infants had considerably greater ability to detect and localize stimuli than those that received similar lesions as adults. Second, we suggest that the visual functions that survive striate lesions in macaques made in adulthood resemble those in human ‘blindsight’. Third, we report that monkeys with striate lesions made in infancy are able to discriminate direction of visual motion.

Damage to primary visual cortex (striate cortex, V1) has a devastating effect on vision in humans and other primates. This chapter addresses three questions about the effects of striate cortex lesions in macaque monkeys. The first is whether striate lesions sustained in infancy have different effects from similar lesions made in adulthood. The second question is whether the vision that survives striate lesions in monkeys resembles the implicit or nonconsciously vision that survives striate lesions in humans, that is, can destriate monkeys show blindsight? The third and related question is whether destriate monkeys can discriminate direction of stimulus movement. All three questions are relevant for understanding the role of striate cortex in visually guided behavior and visual consciousness in human and nonhuman primates.

The effects of age at the time of lesion

The idea that brain lesions in infancy might have lesser effects than lesions in adults was first suggested in 1865 by Paul Broca (Berker et al., 1986). The first systematic experiments on the question were those of Margaret Kennard in the 1930s (Kennard, 1936, 1938; Kennard and Fulton, 1942). Since then considerable evidence has accrued indicating greater recovery after early brain damage in primates in several different regions of the cerebral cortex (Goldman, 1972; Carlson, 1984a, b; Carlson and Burton, 1988; Bachevalier et al., 1990; Bachevalier and Mishkin, 1994). However, until our studies, the behavioral effects of lesions of striate cortex in infancy and adulthood had never been compared in primates. Furthermore, it was possible that the effects could have been the opposite to the usual ones of lesser deficits after lesions in infancy that were found in other systems. This is because early striate lesions in monkeys produce more rapid and more extensive
secondary degeneration in the lateral geniculate (Milhailovic et al., 1971) and, transneuronally, in
the retina than do later ones (Cowey, 1974; Dineen
On the basis of this evidence, one might expect
clearly lesions of striate cortex to cause greater visual
deficits than later ones. To examine these alternatives
we studied the ability of monkeys to detect and
localize visual stimuli after striate lesions made in
infancy or adulthood (Moore et al., 1996, 1998).

Subjects

Six Macaca fascicularis monkeys received large
unilateral lesions of striate cortex either in adulthood
(the adult lesion group, A-1, A-2, and A-3) or at 5-6
weeks of age (the infant lesion group, I-1, I-2, and
I-3). Sagittal sections through the lesions of an adult
lesion animal are shown in Fig. 1 and of an infant
lesion one in Fig. 2. Both figures also show examples
of MR images of the lesion and intact hemispheres.

Fig. 1. Striate cortex lesion in Monkey A-1. Sagittal sections showing the intact (left) and operated (right) hemispheres. Striate cortex in the intact hemisphere is indicated by shading. In the operated hemisphere the bold lines show the borders of the lesion. The diagrams of a standard dorsal view of the brain (top) show the approximate level of the sagittal sections. The photographs show magnetic resonance (MR) images of sagittal sections through the intact and damaged hemisphere (details of the MR methods are in Moore et al.,
1995). The oblique lines show the drawing of the section closest to each MR image. ca, calcarine; lu, lunate; io, inferior occipital.
and illustrate how these images can provide an indication of the site and size of experimental lesions prior to histological analysis after sacrifice of the animal. The removal of striate cortex was complete or nearly so in two of the adult lesion animals A-1 and A-3 and two of the infant lesion ones I-1 and I-2. In the other two animals there was considerable sparing of the cortex representing portions of the visual field beyond eccentricities of 25°. Testing of the animals began 2-6 years after surgery.

**Behavioral procedures**

During training and testing sessions the animals were placed in a primate chair. They were trained to fixate and make saccades to visual targets. In the initial task, the targets were small spots that appeared at unpredictable locations and unpredictable times. For a reward the animal had to both detect the onset of the target and saccade to it. The targets were on for 1 s and failure to initiate an eye movement during that period was defined as a detection error. On a third of the trials no target was presented; on these ‘blank’ trials the animal was rewarded for maintaining fixation. This behavioral paradigm is summarized in Fig. 3.

All the animals were tested monocularly. For each monkey, detection was tested at 48 locations within the central 24° (Fig. 3). After training, each animal was tested for about 3400 trials divided into
12 repetitions. Each repetition covered all 48 points, 4 trials/point in the visual half field contralateral to the lesion and 8 trials/point in the half field side ipsilateral to the lesion.

As a control for light scatter, the blindspot in the ipsilateral field was plotted. The animals consistently failed to detect targets presented within their blindspot as shown in Fig. 4.

**Visual perimetry**

Figure 5 shows the overall results from one animal with an infant lesion (I-2) and one animal with an adult lesion (A-1). Both animals performed with near perfect accuracy in the field ipsilateral to the striate lesion. The infant lesion animal also performed well in the field contralateral to the lesion except for the most eccentric points. By contrast the adult lesion animal failed to detect contralateral stimuli about 70% of the time which is near chance performance.

Figure 6 shows the detection errors over the course of the 12 repetitions at 288 trials per repetition. All the infant lesion animals begin at a higher performance level than any adult lesion subject and their final level was also much higher. Only one of the adult lesion animals, A-3, showed considerable improvement over the course of testing.

The recovery of detection at each target site of one adult lesion animal (A-3) is shown in Fig. 7. The recovery occurred first at the most central points and then at the more peripheral ones. The infant animal that showed the poorest initial performance and therefore the most recovery (I-3) also showed this same center to periphery pattern of recovery (Fig. 7).

We then investigated the effect of target contrast: the animals with the best final performance in each age at lesion group were tested with the contrast reduced by 1 log unit (Fig. 8). The reduced contrast had no effect on the animal with the infant lesion (I-2) but it virtually reinstated the original deficit in the animal with the adult lesion (A-3). Thus, the recovery of the adult lesion subject seemed dependent on the target contrast whereas the animal with the infant lesion continued to detect and localize the less intense stimulus within the scotoma.

In summary, the animals that had received their lesions in infancy could detect and localize targets at most locations within the scotoma from the beginning of testing and, after some practice, performed virtually perfectly everywhere. By contrast, the animals that received their lesions as adults appeared blind to targets at most sites for some time. Only one of the adult lesion animals, and only after considerable time and practice, was able to localize targets fairly consistently within its scotoma. However, its performance was never as good as that of the worst infant lesion animal.

**Do monkeys with striate cortex lesions show blindsight?**

The virtually blind performance of the three adult lesion animals described in the previous section was unexpected. In previous studies, monkeys
and then circles on 1995.

pattern of

contrast: 25%

contrast

contrast

contrast

25%

contrast

25%

contrast

0-25% errors 25-50% errors 51-75% errors 76-100% errors

Fig. 4. As a control for possible light scatter, detection of target stimuli in and near the blindspot ipsilateral to the lesion was tested. Target stimuli were presented in 1° steps in the vicinity of the blind spot. (The center of the blindspot is usually 14°-17° from the fovea along the horizontal meridian.) Each circle represents 4-12 stimulus presentations. The stimulus contrast was 3 log units above the background except for monkey I-2 where a contrast of 2 log units above the background was used. Since the animals were unable to detect stimuli at two or more locations in the blind spot, the amount of effective light scatter must have been less than the radius of the optic disc (about 3°).

Infant Lesion (I-2)  Adult Lesion (A-1)

0-25% errors 25-50% errors 51-75% errors 76-100% errors

Fig. 5. Total detection errors (failure to initiate saccades) for monkeys I-2 and A-1. Each circle represents one test point; for each polar angle loci were tested at 8°, 12°, 16° and 24° eccentricity. The visual half field contralateral to the striate lesion is shown on the right for both plots (Moore et al., 1996).
Fig. 6. Detection performance across all repetitions for all the animals with striate lesions. Filled symbols, contralateral; open symbols, ipsilateral. (Moore et al., 1996.)

With striate lesions received in adulthood had been able to detect and localize stimuli (Cowey and Weiskrantz, 1963; Mohler and Wurtz, 1977; Newsome et al., 1985; Seagraves et al., 1987). Similarly, after striate lesions, some humans — those with 'blindsight' — were also able to do much better than our adult lesion monkeys (e.g. Weiskrantz, 1986). However, we noticed something that suggested that our animals actually had much more knowledge of target location than their high incidence of error had indicated.

When the animals did not detect targets in the scotoma (i.e. failed to saccade to them) their post-trial behavior indicated that they had knowledge of target location. On such trials, after the trial had ended and both the fixation point and target stimulus were turned off, the animals often made saccades to the site where that target had just been presented (Fig. 9). This post-trial saccade indicated that the animal had implicit knowledge of target location. We then changed the task to try to tap into this implicit knowledge and to make the procedure more similar to those in earlier studies that had demonstrated better performance after adult striate lesions (Mohler and Wurtz, 1977; Seagraves et al., 1987).

Fig. 7. Recovery of detection over successive blocks of 1152 trials for monkeys A-3 and I-3. Note that for both animals recovery was earlier at the more central sites.
High (3.0 log.) Contrast  Reduced (2.0 log.) Contrast

A-3

Ipsi Contra

I-2

Ipsi Contra

0-25% errors
26-50% errors
51-75% errors
76-100% errors

Fig. 8. The effect of reducing target contrast on detection of monkeys A-3 and I-2.

Fixation Point
Stimulus
Eye

I-1

30%

30%

30%

A-1

30%

30%

30%

Fig. 9. Frequency of location of final position of saccades that occurred after the offset of an undetected target stimulus presented in the contralateral field in monkeys I-1 and A-1. The targets were presented at an eccentricity of 24° at the polar angle shown by the arrows. The polar angles of the saccades were collapsed into 30° bins. These data indicate that the animals had information about the location of the trials even on trials that they failed to saccade to the target.
**Use of a procedure resembling 'forced choice'**

In our original behavioral procedure (Fig. 10, *left*) the fixation point stayed on after the target appeared. We then changed the procedure so that the fixation point went off when the target appeared (Fig. 10, *right*). When the fixation point turned off, it was as if the animal was 'forced' to saccade to a new location. To our surprise, with this change the two adult lesion monkeys, A1 and A2 who had previously acted virtually blind in the half field contralateral to their striate lesion, were now able to detect and accurately localize many of the targets in the contralateral half field.

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**Fig. 10.** Effect of time of stimulus onset on detection. (Left) Poor performance of A1 and A2 in the original paradigm in which the fixation point remains on after onset of the target stimulus (bottom). See also legends to Figs. 3 and 5. (Right) Localization of previously undetected targets during the new procedure in which the fixation point was extinguished simultaneous with the onset of the target. The locations of the targets are shown by the small arrows in the figure on the left and the black circles in the figure on the right. Mean endpoints of the saccades are shown by the open symbols. Note that with the original procedure the targets in the contralateral field were not detected, whereas with the revised paradigm the endpoints of the saccades were on or near the targets in both half fields (Moore et al., 1995).
Figure 10 (right) shows the endpoints of saccades to targets at specified eccentricities with the new procedure. The end points of the saccades in both half fields were close to the targets. The accuracy of saccades to all the targets is shown in Fig. 11, where the direction of the target is plotted against the direction of the saccade for both half fields. The accuracy of the saccades on the contralateral side is virtually as good as on the ipsilateral side for one animal and only slightly impaired for the other.

Monkeys appear to show blindsight

Whereas in the first saccade paradigm (Fig. 10, left) the adult lesion monkeys continued to fixate on the fixation spot and ignored the targets flashed into the scotoma, in the new paradigm they now initiated accurate saccades to the targets. Note that the new procedure essentially forces the animals to initiate a saccade and, in humans with blindsight, it is a forced choice paradigm that is usually required to reveal accurate detection and localization (Stoerig and Cowey, 1997). In a comparable situation as first shown by Zihl and Werth (1984) humans that do show blindsight also require a cue as to target onset in order to localize and detect stimuli.

In summary, the adult lesion animals showed good detection and localization but only when the fixation point was turned off thereby ‘forcing’ the animal to saccade to a new location. This provides an answer to our second question: monkeys with striate lesions do show visually guided behavior similar to that of human blindsight patients. Cowey and Stoerig came to a similar conclusion in a study in which monkeys were able to categorize visual targets in the scotoma as nontargets in spite of their ability to localize them accurately (Cowey and Stoerig, 1995; Stoerig and Cowey, 1997; Stoerig et al., 2002).

Can destriate monkeys discriminate direction of movement?

Motion discrimination

We trained the three infant lesion animals and the best performing adult lesion animal (A-3) from the experiments described above on various movement discrimination tasks using a go/no-go procedure (Fig. 12). On each trial the animal fixated on the fixation point and then a moving stimulus was presented in either the field contralateral or ipsilateral to the lesion. If the stimulus moved in one direction (S+), the animal was rewarded for saccading to it. However, if the stimulus moved in the opposite direction (S−) the animal was rewarded for continuing to fixate. Discrimination performance was based on the degree to which the monkey could choose the correct trial on which to initiate
Direction of Motion Discrimination (Go/No-Go Task)

Fig. 12. Direction of movement discrimination paradigm. The monkey first fixated a fixation point, shortly after which a stimulus was presented. The task required the animal to saccade (arrow) to the stimulus ('go' trial, left) or to withhold such movements ('no-go' trial, right) depending on the stimulus.

Fig. 13. Position of the stimulus apertures (5° and 15° diameter circles) within the scotomata of the animals with striate lesions used in the movement discrimination experiments. Unshaded areas represent zones of the visual field with corresponding intact striate cortex. (In part after Moore et al., 2001a,b). The estimate of the field defects are based on maps of the visual topography of striate cortex (Gattass et al., 1988) and the lateral geniculate nucleus (Mandelli et al., 1996).

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. We tested the three infant lesion animals and the best performing adult animal (A-3). The stimuli were presented within the scotoma (Fig. 13) and as a control in the field ipsilateral to the lesion.

When the discriminandum was a horizontal bar that fell within the scotoma, each monkey could easily discriminate upward from downward motion inside the scotoma. This is consistent with the finding that in the absence of striate cortex many single neurons in Area MT and Area V3A are still sensitive to direction of motion of a bar (Rodman et al., 1989; Girard et al., 1991, 1992). However, discrimination of
the direction of movement of a bar is not the same as true discrimination of direction of movement (Nakayama and Tyler, 1981). Since the moving bar begins its traverse at very different positions when the movement direction is upward as opposed to downward, the monkeys could solve the discrimination by localizing the bar within the scotoma at the start of the trial. In order to determine whether the animals had true discrimination of movement, we removed the possibility for the animal to use stimulus displacement cues by using as discriminanda, coherently moving dot fields random dot kinetograms (Britten et al., 1992). The dots were presented in a 15° aperture that fell within the scotomata of all the animals.

All the infant lesion animals and adult monkey A-3 were able to discriminate moving from static dot fields inside their scotomata as accurately as in their good half field (Fig. 14). On the discrimination of direction of movement of the random dot patterns the infant lesion animals were somewhat impaired in the scotoma but performed significantly above chance (for both the speeds tested, 4° and 20°/s) (Fig. 14). By contrast, the best performing adult lesion animal (A-3) was totally unable to tell direction of motion in her scotoma even though she had recovered some detection and localization capacity as described earlier (Fig. 14).

This ability of the infant lesion animals to detect direction of motion was crucially dependent on the size of the aperture used in the testing. When the aperture was reduced to 5° diameter all three infant lesion animals performed at chance at the up versus down discrimination (Fig. 15). Two of them (I-2 and I-3) could not even distinguish upward movement from random motion of the dots. However in the smaller aperture they could still discriminate moving from static dot patterns (Fig. 15). This dependence of the residual motion sensitivity of the infant lesion animals on the size of the motion

![Graphs](image-url)

Fig. 14. Direction of motion discrimination in a 15° aperture. Percent correct trials on discrimination of upward moving from static dot patterns and of dot patterns moving upward or downward at 4°/s or 20°/s within the scotoma or in the intact field. Note that all the animals could discriminate moving from static stimuli but only the infant lesion animals could discriminate direction of movement above a chance level. (After Moore et al., 2001a,b).

![Graphs](image-url)

Fig. 15. Direction of motion discrimination in a 5° aperture. Percent correct trials on discrimination of upward moving and static dot patterns, of upward and randomly moving ('noise') dot patterns and of upward and downward moving dot patterns in the scotoma and intact fields. Note that none of the infant animals could discriminate direction of movement in the 5° aperture, whereas they were able to do so in the 15° one. (After Moore et al., 2001a,b).
stimulus fits with the finding of Weiskrantz et al. (1995) that human blindsight patients require large scale displacements to detect direction of movement of a spot. The total inability of the adult lesion animal to discriminate direction of movement of a random dot field is strikingly similar to the situation with those human patients who show blindsight after striate lesions. They too are unable to discriminate direction of movement of a random dot pattern although they can often detect the direction of movement of a bar, grating or a single large dot (Blythe et al., 1986; Magnussen and Mathiesen, 1989; Barton and Sharpe, 1997; Azzopardi and Cowey, 2001; Cowey and Azzopardi, 2001). The behavioral abilities of human blindsight patients and the adult lesion monkey are thus parallel to the properties of single neurons in Area MT after striate lesions: sensitivity to the direction of motion of a bar but not to that of random dot fields, i.e. no true direction of movement discrimination (Rodman et al., 1989; Azzopardi et al., 1998).

**Implicit knowledge about direction of movement**

When we examined the fine grain of the monkeys' saccades to the moving dot fields we realized that the metrics of the saccades differed on correct trials from those on incorrect ones. As shown in Fig. 16 for one of the infant lesion animals, when the animal made a correct saccade to upward moving dots the vertical component of the saccade was higher than when the animals made, incorrectly, a saccade to the downward moving dots. Thus it had extracted information about the direction of movement that was reflected in the metrics of the oculomotor response. This finding is consistent with the ability of the infant lesion animals to perform the direction of movement discrimination far above chance level.

What was surprising, however, was the behavior of the adult lesion animal that had completely failed the go/no-go discrimination of the direction of stimulus movement (Fig. 17). This animal's eye movements indicated that it too was able to extract information about direction of movement despite its overt behavioral performance. Unlike the infant lesion animals, the adult animal appeared to have covert or implicit 'knowledge' of direction of movement while still failing the discrimination task. As in the case of the role of the temporal cues in our perimetry study, and in human blindsight, the ability to use visual information after striate lesions depends on how that ability is assessed.

![Graph showing eye position and time from saccade onset](image)

Fig. 16. Evidence for implicit direction of motion sensitivity in animal 1-1. Mean horizontal (top) and vertical (bottom) components of saccadic eye movements made to fields of moving dots during direction of motion discrimination (with the 15° aperture). The animal was trained to saccade to a field of upward moving dots and withhold movement to downward moving ones. Although the animal performed above chance, as noted above, it made enough errors on the no-go or downward movement trials to compare the saccades made to upward and downward movement. The black trace shows the amplitude of correct or go saccades to the upward moving stimulus and the grey trace shows the amplitude of incorrect saccades to the downward moving stimuli. Note that the vertical amplitudes to the upward moving dots are greater than that to the downward moving ones. (After Moore et al., 2001a.)
Concluding discussion

Comparison of monkeys and humans

The visual behavior of the monkeys that received striate lesions as adults closely resembled that of humans with blindsight in two major ways. First, they could detect and localize visual stimuli in their scotomata only if there was a temporal cue to respond as in a forced choice paradigm. Second, they were unable to discriminate the direction of a moving dot field although again, like human blindsight patients, they could detect whether a dot display was moving and the direction of movement of a bar (Barton and Sharpe, 1997; Azzopardi and Cowey, 2001). The analysis of the incorrect saccades of the monkeys suggest that they may have information about the direction of moving dots but are unable to use this information to control their behavior in an operant task. It would be interesting to see if there were any parallels of this implicit knowledge of direction of movement in human blindsight patients.

The visual guided behavior of the infant lesion animals was much better than that of the adult lesion animals and was different from that of the usual blindsight patients both in not needing a forced-choice situation and in being able to discriminate the direction of movement. Patient GY is a much studied blindsight patient who, since he received his striate lesion in childhood at the age of 8, makes an interesting comparison with our infant lesion animals (Kenridge et al., 1999). He was similar to them in that his ability to localize a visual stimulus remained above chance even in the absence of temporal cues as to target onset. Yet, he was different from them in his apparent inability to discriminate the direction of movement of a random dot field (Azzopardi and Cowey, 2001).

The neural basis of the vision that survives striate lesions

What are the neural mechanisms that underlie the visual functions that survive striate lesions in adulthood? At least in the monkey, these residual or recovered functions are abolished by lesioning the superior colliculus (Mohler and Wurtz, 1977; Rodman et al., 1990). Tecto-fugal fibers project to the pulvinar (Benevento and Standage, 1983) which in turn projects widely to extra-striate visual cortical areas and might provide them with visual information sufficient to sustain residual visual functions. There are three lines of evidence that the activity of extra-striate areas might be crucial for the full scope of the visual capacities after striate lesions.

The first line of evidence is that several extra-striate areas in the dorsal visual cortical system...
continue to respond to visual stimuli after striate lesions, namely area MT, area V3A and the Superior Temporal Polysensory area (STP) (Bruce et al., 1986; Rodman et al., 1989, 1990; Girard et al., 1991, 1992; Gross, 1991). In fact, after striate lesions, cells in these areas maintain their normal visuotopic organization (except for STP, which is not visuotopically organized in normal animals) and continue to be sensitive to orientation and direction of motion of a moving bar. Thus, the activity of these areas could be the basis of the visually guided but unconscious behavior that survives striate lesions.

The second line of evidence for a role of extrastriate cortex in blindsight is that in three separate sets of studies, patients with hemidecortication showed little or no signs of explicit blindsight (i.e. involving forced-choice testing) in the blind half field (King et al., 1996; Stoerig et al., 1996; Faubert et al., 1999). Similarly, hemidecortication in monkeys prevents accurate saccades to a target (Tusa et al., 1986).

The third line of evidence is that there is considerable activity in extrastriate cortex after striate lesions in humans as revealed by functional imaging (Barbur et al., 1990; Stoerig et al., 1998; Baseler et al., 1999; Bittar et al., 1999).

**Striate lesions in infancy**

What might be the underlying mechanism for the much greater recovery in the infant lesion animals? Following damage to striate cortex in both infant and adult monkeys, the retino-geniculate pathway degenerates dramatically. The remaining excitatory input to the geniculate appears to be from the superior colliculus (Kisvarday et al., 1991). A possible insight into greater recovery following early damage comes from studies of transneuronal degeneration of retinal ganglion cells after striate lesions. These studies show much faster and possibly greater degeneration in younger monkeys (Cowey, 1974; Dineen et al., 1982; Weller and Kaas, 1989). Thus, it is conceivable that the faster degeneration of the retino-geniculate pathway in infant-damaged monkeys actually facilitates recovery by disinhibiting the residual retino-collicular pathway (Moore et al., 2001). In other words, the retino-tecto-geniculate pathway may be unmasked more rapidly after early striate cortex damage and thus more able to contribute to the residual function after such damage.

Conversely, surviving lateral geniculate neurons, the geniculo-extrastriate pathway, and direct retinal inputs to the pulvinar may play a greater role after early lesions, as suggested by studies of early visual cortex lesions in cats (reviewed in Payne et al., 1996). After early lesions of V1 in monkeys, surviving geniculate neurons show dramatically expanded dendritic branching (Hendrickson and Dineen, 1982), and are overwhelmingly drawn from the calbindin-immunopositive subpopulation of the koniocellular channel, suggesting the survival of a unique population which projects to specific components of extra-striate cortex (Rodman et al., 2001).

**Acknowledgments**

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