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Adaptation of Saccades and Fixation to Bilateral Foveal Lesions in Adult Monkey

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Bilateral foveal lesions were made by laser photocoagulation in adult monkeys. One day post-lesion, animals fixated with a new retinal locus inferior to the fovea (in the visual field) that they used permanently. Fixation stability improved modestly over two days. Initially, saccades maladaptively brought the lesioned foveae to visual targets. Over at least several weeks, saccade trajectories gradually changed bringing targets to intact retina, although some animals never totally adapted. The slow time course of saccadic adaptation to foveal loss suggests a mechanism different from that documented in other studies of saccadic adaptation and from that used by the fixation system.

Macaca nemestrina Saccades Fixation Adaptation Foveal lesions

It has been demonstrated that the oculomotor system is one which has the ability to adapt to changes in the environment, as well as to injury of its components. Studies have demonstrated that saccade trajectories can be quickly altered when verbal instructions to deviate from a visible target are given (Hallet, 1978), or when visual feedback concerning saccadic accuracy is altered by displacing the target during the course of the saccade (Henson, 1978; Miller, Anstis & Templeton, 1981). In both cases, the saccadic system makes a rapid adjustment to compensate for these manipulations, sometimes in a matter of a few trials. Adaptation is also seen when injury or disease degrade the motor system and distort the accuracy of saccadic trajectories. For example humans with VIth nerve palsy make saccades which undershoot their goal (hypometric) (Kommerell, Olivier & Theopold, 1976). However, these saccades regain accuracy over a period of only three days. Hypometrias have been induced in monkeys by weakening the horizontal rectus muscles, but are corrected after a similar period of time (Optican & Robinson, 1980).

A qualitatively different type of adaptation is required when the oculomotor system loses central retinal input as in age-related maculopathy or Stargardt–Behr disease. Since visually guided eye movements can no longer be initiated from or terminated at the fovea, the system must incorporate an eye position offset when calculating these movements. Studies in human patients have shown that the fixation system can adopt a systematic offset (a preferred retinal locus or PRL) as a result of foveal loss (Timberlake, Peli, Essock & Augliere, 1987), but that the saccadic system does not adapt easily to retinal degeneration (Whittaker & Cummings, 1988; White & Bedell, 1990). However, human studies are confounded by variability in the size, inhomogeneity, and lack of bilateral correspondence of the area of diseased retina, as well as by what is often a continuous degenerative process (see White & Bedell, 1990 for a description of variability in a patient sample).

We looked at the nature and extent of adaptation of the fixation and saccadic systems to central retinal loss in monkeys using an induced lesion. This preparation enabled precise control of both the temporal and spatial aspects of the lesion. We found that both fixation and saccades compensate for foveal loss, but that adaptation in the fixation system occurs virtually overnight, while saccadic adjustment is much slower and appears incomplete. The different time courses of adaptation in these two systems as a result of retinal lesions suggests that the mechanisms underlying their plasticity are very different from each other, and from other mechanisms postulated for saccadic adaptation.

METHODS

Three adult monkeys (*Macaca nemestrina*) were trained to fixate a stationary 12 min arc target and make saccades to follow 10 deg arc steps of this same target. Fixation training was initiated using a five-light discrimination paradigm (Skavenski, Robinson, Steinman & Timberlake, 1975) which is used to insure foveal fixation, since it forces the animal to constantly watch for changes in a high acuity display.

After monkeys became proficient at this task, surgery was performed to implant an eye coil for recording eye position using the magnetic field search-coil technique (Fuchs & Robinson, 1966), and an aluminum post to

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stabilize the head so that eye position with respect to head position could be monitored. After recovery from surgery, the search-coil apparatus was calibrated by moving the five-light display through known angles with respect to the animal. Fixation training then proceeded using accuracy and duration of fixation eye movements as the behavioral criteria for reward. Animals made the transition to these new contingencies quickly, and were gradually trained to fixate for 10 sec intervals on a 12 min arc white spot of light. An electronic window was used to monitor eye position. This window was gradually reduced over sessions, thereby forcing the animals to suppress their large saccades and to use slow, smooth eye movements and small saccades to maintain these 10 sec fixations.

The animals were also trained to make saccades to 10 deg arc target steps up, down, left and right, with direction selected in random order. To accomplish this, the fixation stimulus was turned on approximately centered in the visual field. When the monkey fixated this stimulus, it was extinguished and then re-illuminated at a 10 deg arc eccentric position. The eccentric target was displayed for 1 sec, and if it was acquired in this time, the monkey received a squirt of water and a correct trial was logged.

Data acquisition

For each animal, baseline data on fixation characteristics and saccade accuracy were taken over a number of sessions prior to the retinal lesions. After the lesions were performed, data were collected using the same protocol beginning the next day and for several months thereafter.

Animals were run through a calibration routine prior to all recording sessions, in which they were required to fixate for 3 sec at the center and each of four 10 deg arc eccentric positions. This allowed us to use the angle of the coil in the fields to obtain an accurate comparison between pre- and post-lesion fixation loci. In addition, care was taken to precisely position the monkey's heads in the same place with respect to the fixation target across sessions. Analog voltages related to horizontal and vertical eye position were filtered at 100 Hz (antialiasing) and sampled and digitized at 200 Hz. In addition, the mean horizontal and vertical voltages obtained in each of the five calibration positions were processed with a trigonometric algorithm that computed the angle of the eye-coil with respect to the magnetic field when the monkey looked straight ahead (coil-bias), as well as coil sensitivity to horizontal and vertical rotations (for a description of the algorithm see Appendix A). During subsequent data acquisition these parameters were used to correct nonlinearities introduced by coil goniometrics in the fields and to convert the arbitrary voltages to horizontal and vertical eye position in minutes of arc. In system checks with a dummy coil overall system noise had a SD of <2 min arc on both meridia and departures from linearity were $<1\%$ at the 10 deg arc eccentricities used here.

A fixation session consisted of fifty 10 sec trials. Fixation was monitored by an electronic window which was set to limit maximum eye excursion to 0.9 deg arc from the target. It should be noted that this criterion was relaxed by opening the window to 10 deg arc centered on the "new" fixation locus immediately following the lesion, so that the animals' choice of this locus would not be influenced by our behavioral contingencies. By the second post-lesion day, it was apparent that the new locus was established and the window was reduced to limit its excursion to within 1.5 deg arc of the target over subsequent post-lesion sessions. Monkeys received a squirt of water for maintaining fixation for the required interval; however, if the eye left the window, the trial was aborted, no water was given, and a time out ensued.

Saccade data were taken immediately following fixation sessions. Once again, fifty correct trials comprised a session. Data were taken by the same procedure that was used in training. The animal was reinforced for acquiring the eccentric target within the 1 sec period providing that the final saccade brought the animals eyes to within 1 deg arc of the goal. This criterion was usually met easily, since monkeys generally take only one or two saccades to land almost directly on the target. Immediately after the lesions, this criterion was first relaxed and then tightened as with fixation so that the natural course of recovery of the eye movements could be followed. Trials were also discarded in the event that the animal guessed and made a saccade to an inappropriate target position, but still managed to acquire the correct target within the allotted time.

After baseline data were collected, bilateral laser photoagulation was administered to the foveae. In one animal (M46), 600 μm dia lesions corresponding to 3 deg arc of visual field (de Monasterio, McCrane, Newlander & Schein, 1985) were made with a red krypton laser, which heats the retinal pigment epithelium and thereby destroys overlying neural elements. In the other two monkeys (M62 and M63), 400 μm dia lesions were made removing 2 deg arc of the visual field with a green argon laser, which destroys neural tissue directly. The animals were anesthetized for the surgery, and were kept in this state until they were returned to their home cages. They awoke in their cages to normal room illumination, and spent the remaining approx. 7 hr of the day of the surgery in these normal conditions. Room lights were turned off as usual that night. Since lesions of this type produce no physical discomfort, animals were tested the next day, (after an additional approx. 3 hr of lighted home cage environment) at which time data on fixation and saccades were taken. These eye movements were recorded on subsequent days until saccades showed no further improvement, a period exceeding 2.5 months. The effectiveness and spatial extent of the lesions were verified by fundus photography. Finally, the animals were sacrificed and histology was performed on the retinae. Retinal histology, performed after recording was complete, revealed a total absence of functional photoreceptors and other cells in the area of the lesion. Further evidence as to the

effectiveness of the lesions was obtained in single unit recording in striate cortex which revealed totally non-responsive neurons in an area of cortex roughly corresponding to the cortical projection of the lesioned retinae immediately after the lesions were made (Heinen & Skavenski, 1992).

RESULTS

Fixation

Pre-lesion. Pre-lesion data were gathered on two animals (M63 and M62). The third monkey (M46) was also trained to fixate before the lesion, and although no data was taken on him at this point, his fixation looked similar to that of the other two animals. Before the lesion, these animals exhibited fairly tight fixation, using mostly smooth eye movements (slow control) to hold the eye on the target. Ten of the best 10 sec fixations from each of the pre-lesion sessions were analyzed to assess the monkey's capability in the task. Standard deviations of eye position for separate horizontal and vertical components as well as bivariate contour ellipse area (BCEA), a measure of two-dimensional standard deviation which is not restricted by the arbitrary choice of eye rotation axes, are shown for each monkey's ten best 10 sec fixations in Table 1 (see Steinman, 1965 for a description of the assumptions underlying and method of calculating BCEA).

Both animals showed variability (standard deviations and BCEAs) of eye position which fall in the range of good monkey and human fixation (Skavenski *et al.*, 1975). Both monkeys also showed a strong upward drift component in their slow control. The mean speed and angle of these drifts were calculated and are also shown in Table 1.

Post-lesion. During the recording session on the day following the retinal lesions, all animals looked predominately above the target, putting it below the scotoma in the visual field. It should be noted that animals are actually using superior *retina* to fixate in this situation due to the optical inversion of the lens. They would occasionally jump above and below the target, i.e. use retinal loci above and below the lesion to fixate, but this was sporadic and rare. The shift in fixation location was determined from an initial upward shift in the vertical angle of the eye coil in the magnetic fields (coil bias) of 3.8 deg arc in M63 and 1.6 deg arc in M62 when compared to pre-lesion calibrations. A much smaller change was observed in the horizontal dimension in the two animals respectively. The coil offsets recorded from several sessions immediately preceding and subsequent to the lesion are shown in Fig. 1.

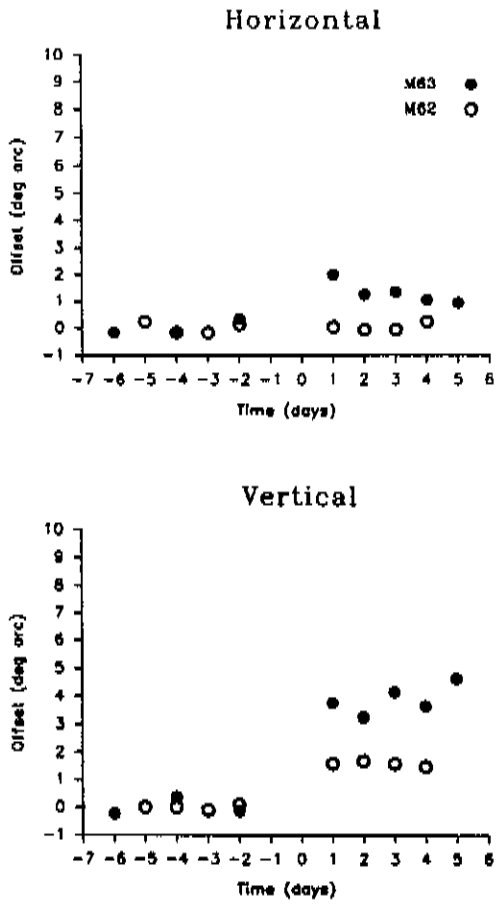

FIGURE 1. Angular offset of the coil in the fields for two monkeys before and after foveal lesions. Both horizontal and vertical offsets are shown. Lesion is time zero. Values are shifted so that the mean pre-lesion offset is zero.

Calibrations run on M46 after the lesion reflected an upward vertical angular offset of 1.4 deg arc and a horizontal offset of 0.1 deg arc which remained constant until the end of data acquisition. Consistent with the other monkeys, M46 had also developed a PRL below the lesion in the visual field which was reliably used across trials.

BCEAs in M63 were markedly increased in size on this first day, averaging 41,176.2 min arc^2 over the session. Mean drift rate was 33.0 min arc/sec which was very similar to pre-lesion values. His predominant drift angle was 67.5 deg arc, and although slightly different than pre-lesion data, was still in an upward direction. By the second post-lesion day the mean BCEA had shrunk to 7493.0 min arc^2, which was about the size at which it remained for the several months in which data was still taken. M62 did not show such a large mean BCEA in his first post-lesion session. However, the variability was substantially larger than his pre-lesion overall mean of 9331.1 min arc^2, a value where it remained throughout post-lesion testing. Figure 2 shows the change in BCEA of both monkeys over sessions before and after lesions. Figure 3 is a schematic of the change in fixation over

TABLE 1. Mean standard deviations (SD) for horizontal and vertical, bivariate contour ellipse area (BCEA), and drift rates and angles for two monkeys before the lesion during ten *best* 10 sec fixations. A drift angle of 90 deg is straight up

Subjects	SD_H (min arc)	SD_V (min arc)	BCEA (min arc^2)	Drift rate (min/sec)	Drift angle (deg)
M63	5.1	10.0	230.7	37.9	118.9
M62	6.1	18.8	720.7	33.5	93.6

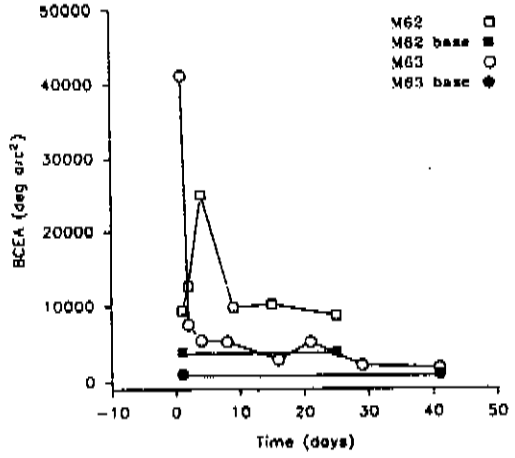

FIGURE 2. Bivariate contour ellipse area (BCEA) over time following the lesion in two monkeys. Open symbols are post-lesion values. Solid symbols are mean pre-lesion baseline values.

time in M63 showing BCEA size, predominant drift angle, and offset of eye position in the visual field.

To characterize the *best* performance that the animal was capable of, BCEAs, SDs, angle of drift and drift rate for the ten best 10 sec fixations obtained after the lesion are summarized and shown averaged across post-lesion sessions in Table 2.

Although fixation data were not taken on M46 immediately after the lesion, measures of his eye position variance at several months post-lesion were like that of the other animals. All three monkeys showed drift rates and angles of drift that were similar to the pre-lesion measures, although M63's drift angle was somewhat variable for several weeks after the lesion. In summary, as a result of the retinal lesions, all animals developed a PRL in the lower visual field which they used consistently in fixation. Monkeys M62 and M63 had BCEAs

TABLE 2. Mean standard deviations (SD) for horizontal and vertical, bivariate contour ellipse area (BCEA), and drift rates and angles for all three monkeys after the lesion during the ten *best* 10 sec fixations after bilateral foveal ablations

Subjects	SD_H (min arc)	SD_V (min arc)	BCEA (min arc²)	Drift rate (min/sec)	Drift angle (deg)
M63	12.42	14.6	1033.69	35.0	95.1
M62	9.9	27.1	1318.4	41.5	80.1
M46	7.1	17.2	766.8	51.5	44.8

which stabilized rapidly after the lesions, but remained elevated relative to pre-lesion values. Both animals had a drift rate and angle comparable to before the lesion, but M63's drift angle took several weeks to stabilize. M46's BCEAs, drift, and angle were comparable to the other animals when he was tested at 9 months post-lesion.

Saccades

Saccade data were analyzed for both accuracy and latency. Accuracy was assessed in terms of error remaining between the PRL and the target following the primary saccade. These measures were taken in M63 and M62 before, and in all animals after the lesion. Both M63 and M62 had similar saccade trajectories, and those from one monkey, M63, are displayed in Fig. 4. Shown here are the mean trajectories of 10 saccades made to each of the four 10 deg arc targets. The data are shown in their actual spatial layout in Fig. 4(a), and then replotted in Fig. 4(b) with the mean trajectories transposed by the magnitude of the target step and in the opposite direction so that the four mean saccade offset positions coincide at the center of the figure. This display allows comparison of the errors regardless of direction of movement and it can clearly be seen that pre-lesion mean primary saccade errors for M63 was very small;

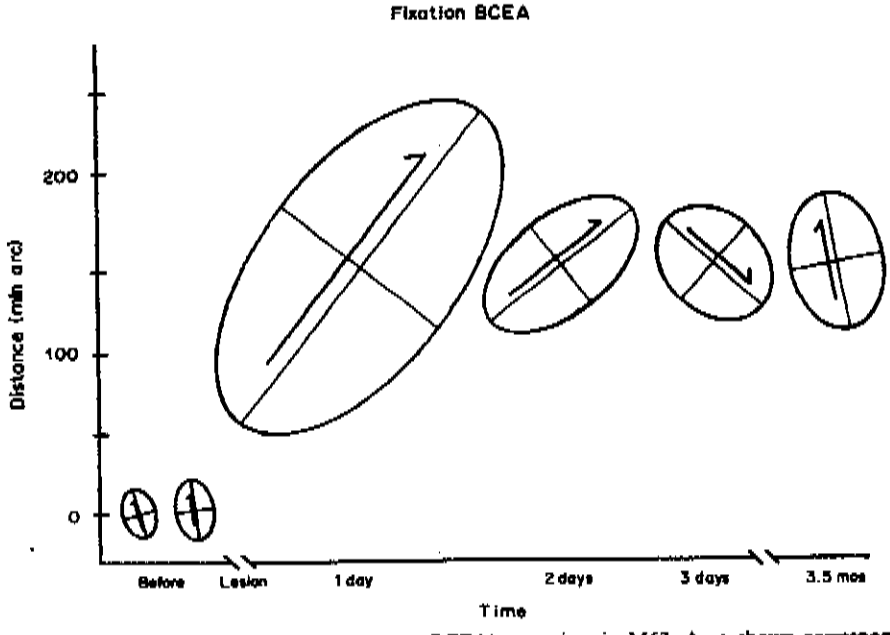

FIGURE 3. Schematic of bivariate contour ellipse area (BCEA) over time in M63. Area shown corresponds to where the animal's fovea was 68.2% of time. BCEAs shown are from one typical 10 sec fixation. The offset of the fovea after the lesion is also depicted, as well as the predominant drift angle (arrow).

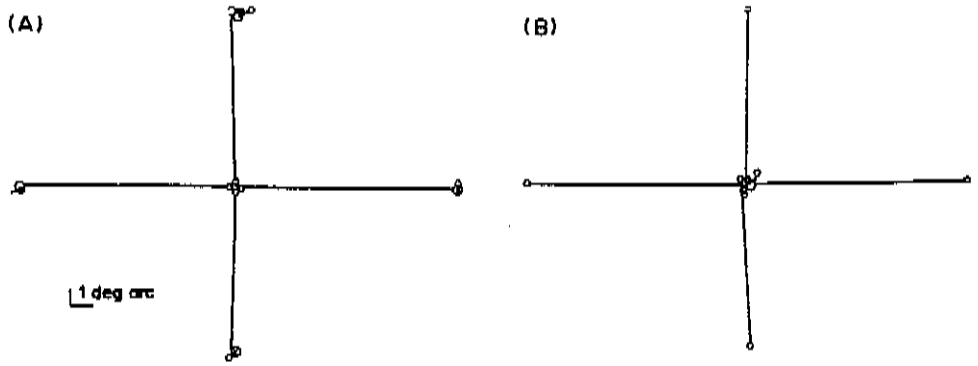

FIGURE 4. Mean saccade trajectories for 10 target presentations in each of four directions in monkey M63. Small circles are start of primary and secondary saccades, large circles are targets. Note that the vertical scale of the display is slightly compressed. (A) Actual spatial layout. (B) Each trajectory is transposed so that targets now coincide in the center, allowing comparison of endpoints of all saccades, e.g. a leftward trajectory now begins at the left and ends at the center.

−0.08 deg arc on the vertical and −0.1 deg arc on the horizontal meridia (where negative errors are down or left). M62 had small errors in his primary saccades also, averaging −0.26 and −0.01 deg arc on the vertical and horizontal meridia, respectively.

Recordings taken on M63 immediately following the lesion showed a vertical error which averaged 0.82 deg arc below the target. This means that the saccade offset was such that the new fixation locus was taken below the target by an amount that placed the image on the foveal scotoma. Over successive days, this error decreased. Mean errors for both animals were fit with an exponential function and are plotted against time in Fig. 5. The time constants for the best fit exponentials were 5 and 20 days respectively. Figure 6(A–E) shows a set of M63's mean trajectories for days 1, 2 and 4, after the lesion, 1 week later, and 2 months later. It is important to note that these plots demonstrate saccades made relative to the new fixation locus. Therefore, the starting point of each trajectory is the new PRL, and the ending point is where the PRL lands. This sequence of five plots shows that the monkey has an initial tendency to bring the lesioned foveae to the target, and then use a second saccade to fixate with the PRL. Over sessions, his saccade trajectory slowly moved upwards so that the PRL and not the blind fovea fell on the target after the primary saccade. It can be seen in this figure in that the "V" formed by leftwards and rightwards saccades becomes shallower over time.

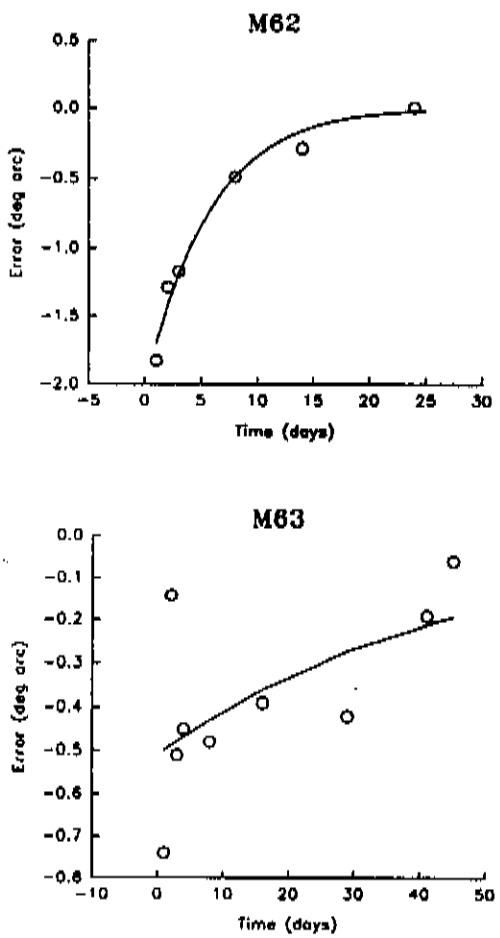

FIGURE 5. Mean post-lesion primary saccade error over each session for two monkeys. Errors are the mean distance between the location of the PRL following the saccade and the target for 50 saccades. Negative numbers indicate saccades below the target. Solid line is the best-fitting exponential through the data.

Data were first gathered on the third animal's (M46) saccades at 9 months after the lesion when his adaptation had stabilized. Surprisingly, he exhibited vertical errors which took the PRL an average of 1.5 deg arc *above* the target, which were reliable and consistent over several subsequent weeks of testing. A sample of his mean saccadic trajectories can be seen in Fig. 6(F). This monkey developed a remarkably different motor pattern to compensate for the lesion. His primary saccades did not place the lesion over the target, but very far above it, and he used a secondary corrective saccade to finally bring the PRL to that target.

The latencies to saccade initiation were normal in M63 and M62 following the lesion, having overall mean values of 159.9 and 130.5 msec respectively, however, M46's latencies were quite long, at 295.3 msec. Saccade dynamics were also normal following the lesion in M63 and M62, in that there were no differences in the amplitude–duration relation for 100 primary saccades made in the saccade tracking task before and after foveal lesions. The mean duration of a 10 deg arc saccade was about 37 msec which coincided with the duration of

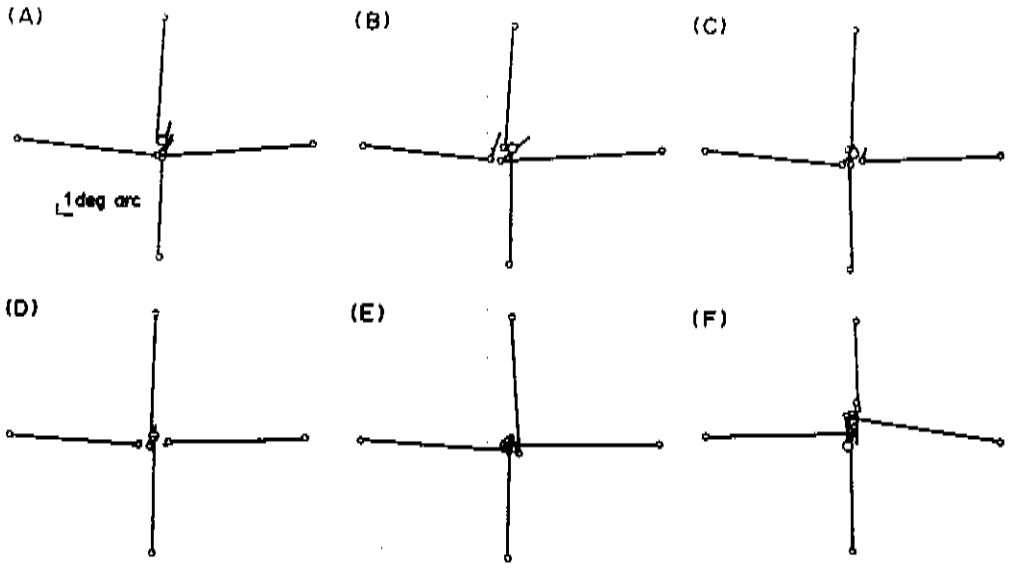

FIGURE 6. Mean saccade trajectories for 10 target presentations in each of four directions. Trajectory is the path that the PRL followed. Note that the vertical scale of the display is slightly compressed. (A–E) Post-lesion trajectories for M63 post-lesion at days 1, 2 and 4, after 1 week, and after 2 months. Initially the saccade brought the PRL below the target, hence obscuring the target with the lesioned fovea. Note that the "V" formed by left and right primary saccade trajectories becomes shallower over time, indicating that saccades are gradually bringing the PRL to the target. (F) Trajectories for M46 at 9 months post-lesion. Note that this monkey took his PRL high above the target, keeping it visible at all times, and used a second saccade to bring his PRL down onto the target.

M46's 10 deg arc saccades measured long after lesions.

To summarize, immediately following the lesion, both M63 and M62 consistently took the PRL well below the target. The saccades of both animals became more accurate over time, with M62 reaching pre-lesion levels within a month. However, M63 did not show complete recovery even after 2 months of testing. M46, on the other hand, overcompensated for his lesions by placing both the lesioned fovea and PRL well above the target, and used a secondary saccade to correct this error.

DISCUSSION

These experiments were conducted to assess the adaptive capabilities of the fixation and saccadic oculomotor systems following bilateral central retinal lesions. We found that adaptation did occur, but was remarkably different for these two systems in terms of both time-course and completeness of recovery. The fixation system adjusted to the lesion virtually overnight, in that animals quickly formed a new fixation locus below the lesion in the visual field where it remained throughout testing. The size of the PRL (as assessed by BCEA) decreased to a stable value within 2 days. The accuracy of the saccadic system did not stabilize for at least several weeks, and in two of the three animals saccades never regained pre-lesion performance in that they did not bring the target directly to the new fixation locus. Another interesting aspect of the saccadic recovery was that the final pattern of eye movements was idiosyncratic across animals. Although two of the monkeys eventually generated saccades which brought the PRL close to or on the target, the third animal made primary saccades which took the PRL 1.5 deg arc above the target and necessitated a large corrective saccade.

Studies in humans with central retinal lesions caused by disease have yielded similar results, in that patients generally form somewhat stable PRLs which are also often located in the inferior visual field (Timberlake *et al.*, 1987). It has also been reported that saccades on the other hand adapt with great difficulty or continue to maladaptively take the lesioned retina to visual targets in such patients (Whittaker & Cummings, 1988). White and Bedell (1990) analyzed video recordings and noted that about two-thirds of their macular disease patients also show incomplete adaptation after prolonged (years) experience with the disease. Qualitatively, our monkeys appear to have adapted more completely, however differences in recording methods and tightness of behavioral control over the subjects makes direct comparison difficult.

The tight behavioral contingencies and high motivation of our animals may also account for the fact that two of our three monkeys performed the saccade tracking task with normal latencies. Long latencies such as observed in one of our monkeys and in patients with age related macular disease has been interpreted to mean that more time is necessary to program saccades that place targets on parts of the retina other than the fovea (White & Bedell, 1990). A similar suggestion was made by Whittaker and Cummings (1990) whose literature review and experiments find that saccades which bring the fovea to a visible target were faster than saccades to remembered positions. Our monkeys performed the saccades they were trained to make in unpredictable directions with normal amplitude duration relations. Consequently our results support the suggestion of Ron,

Robinson & Skavenski (1972) that the presence of the target for the saccade at the time it is made causes it to be fast. Clearly our monkeys and people with central scotomas (Whittaker & Cummings, 1988) can make saccades with normal latencies and amplitude–duration relations, therefore motivational factors must be contributing when they do not.

Several interesting issues are raised by these findings. One of these concerns the consistent use of a lower visual field locus for fixation. Superior and inferior retinal photoreceptors and ganglion cells are distributed in a symmetrical fashion (Curcio, Sloan, Packer, Hendrickson & Kalina, 1987; Perry & Cowey, 1985). However, some evidence exists for a lower visual field advantage in extrastriate cortex in that there is a preponderance of cells which respond to lower visual field stimuli in V3 (Felleman & Van Essen, 1987). It is possible that later visual processing utilizes this field more extensively than it does the upper visual field. This speculation is also consistent with the anecdotal observation that most of what is important in the environment takes place in the lower visual field of primates.

Another issue which is raised concerns the time–course of recovery which both of these eye movement systems follow. Adaptation of the fixation system occurred in a very short period of time. No previous work has been done on adaptation of fixation, and no current models of the fixation system exist, therefore it is difficult to speculate about the underlying mechanism for this recovery. It has been shown however that normal people can fixate quite well on a fixation stimulus placed at a peripheral locus, which suggests that the fixation system is driven largely by visual feedback from anywhere on the retina (Murphy, Haddad & Steinman, 1974). The rapid adaptation of fixation with the PRL may reflect a simple switch to using this built-in capacity.

Although variability in fixation in humans with macular disease and in our monkeys is greater than that found in humans fixating at an eccentric locus (Murphy *et al.*, 1974), it should not be interpreted that these two types of fixation utilize different mechanisms. It may also be that for whatever reason, these more extensive movements are optimal for vision at an eccentric retinal locus. Also, it was uncertain that our animals were fixating at the limit of their capacity after the lesion, since they were not forced to maintain more accurate fixation at this time.

Saccadic adaptation to the retinal lesions occurred over a much longer period of time relative to that of the fixation system, and relative to the time–course of adaptation found in other saccadic plasticity studies. Experiments that either altered visual feedback about saccadic accuracy (Henson, 1978; Miller *et al.*, 1981) or which used verbal commands to change saccade trajectories (Hallet, 1978) have documented a very rapid reprogramming of saccades which can occur after as few as five or six trials. More time is required to recalibrate saccadic trajectories which are perturbed due to damage to the oculomotor system. Kommerell *et al.* (1976), showed that in patients with VIth nerve palsy, approx. 3 days were needed to reverse overcompensation to the imbalance in innervation. Similar studies have been done with monkeys in which the lateral rectus muscle of one eye was surgically weakened, initially creating hypometric saccades and glissades (eye position drift) with that eye (Optican & Robinson, 1980). Patching the good eye enabled the weak eye to make good saccades within 3 days, but created hypermetric (overshoot) saccades in the patched eye. When the patch was reversed, the hypermetria decreased, and recalibration occurred with a time constant of approx. 1.5 days.

The type of saccadic adaptation in the present experiment seems to be of an entirely different nature than that seen in these other paradigms. For example the time constants for error reduction in the two animals followed serially were 5 and 20 days; intervals that were far longer than observed in other adaptation paradigms. These long time constants suggest that the adaptation to foveal loss is not like adaptation to motor disorders. To illustrate further, the type of adaptation used for compensating motor anomalies (weakened or changed muscle length) could be used but apparently is not. All three monkeys adapted PRLs about 1.5 deg arc directly above the lesioned fovea on the retina. In an intact eye this shift could be seen as comparable to downward surgical rotation of the eye on the superior/inferior rectus muscle pair: a circumstance to which the motor system might be expected to rapidly adapt if Optican and Robinson's (1980) results are generalizable to this muscle pair. Again, the long time–course and incompleteness for saccade adaptation in our lesioned animals rule out this sort of rapid motor adaptation. In sum, the eye movement control system never treats the shift from fovea to PRL as being equivalent to a shift of the real fovea.

Although some adaptation of the motor components of the saccade may need to be recalibrated, the major reorganization probably occurs in the coordinate systems in which saccades are first prepared in the brain. Current models of saccade generation all rely upon an initial retinotopic coordinate system for the calculation of the vector for the saccade, i.e. the desired movement is based on the vector formed by the retinal coordinates of the fovea and the target of the saccade (Robinson, 1975; Van Ginsbergen & Van Opstal, 1989). However, work showing that subjects can effectively make saccades which bring goal targets onto peripheral retina (e.g. Hallet, 1978) suggests that retinal error signals may be determined between arbitrary non-foveal loci. Our result argues against the idea that retinotopic computation of the saccade trajectory is made by calculating the vector between the retinal coordinates of the object being fixated and the goal of the saccade. If that were the case then saccades would take the eye correctly to the PRL immediately after foveal lesions. None of our animals did that. Instead, they took their lesioned foveae to the goal target suggesting that the retinal vector is calculated between the fovea and the retinal coordinate of the goal target. This means that the fovea is the origin in computing retinal error signals in the normal animal. In addition, the long time course and incompleteness of

the adaptation seen for saccades suggests that use of the fovea as the origin of the visual signal is particularly resistant to change.

REFERENCES

Curcio, C. A., Sloan, K. R., Jr, Packer, O., Hendrickson, A. E. & Kalina, R. E. (1987). Distribution of cones in human and monkey retina: Individual variability and radial asymmetry. *Science, 236,* 579–582.

Felleman, D. J. & Van Essen, D. C. (1987). Receptive field properties of neurons in area V3 of Macaque monkey extrastriate cortex. *Journal of Neurophysiology, 57,* 889–920.

Fuchs, A. F. & Robinson, D. A. (1966). A method for measuring horizontal and vertical eye movement chronically in the monkey. *Journal of Applied Physiology, 21,* 1068–1070.

Fuchs, A. F., Kaneko, C. R. & Scudder, C. A. (1985). Brainstem control of saccadic eye movements. *Annual Review of Neuroscience, 8,* 307–337.

Hallet, P. E. (1978). Primary and secondary saccades to goals defined by instructions. *Vision Research, 18,* 1279–1296.

Heinen, S. J. & Skavenski, A. A. (1992). Recovery of visual responses in foveal V1 neurons following bilateral foveal lesions in adult monkey. *Experimental Brain Research.* In press.

Henson, D. B. (1978). Corrective saccades: Effects of altering visual feedback. *Vision Research, 18,* 63–67.

Kommerell, G., Olivier, D. & Theopold, H. (1976). Adaptive programming of phasic and tonic components in saccadic eye movements. Investigations in patients with abducens palsy. *Investigative Ophthalmology, 15,* 657–660.

Miller, J. M., Anstis, T. & Templeton, W. B. (1981). Saccadic plasticity: Parametric adaptive control by retinal feedback. *Journal of Experimental Psychology: Human Perception and Performance, 7,* 356–366.

de Monasterio, F. M., McCrane, M. K., Newlander, J. K. & Schein, S. J. (1985). Density profile of blue-sensitive cones along the horizontal meridian of macaque retina. *Investigative Ophthalmology and Visual Science, 26,* 289–302.

Murphy, B. J., Haddad, G. M. & Steinman, R. M. (1974). Simple forms and fluctuations from the line of sight. *Perception and Psychophysics, 16,* 557–563.

Optican, L. M. & Robinson, D. A. (1980). Cerebellar-dependent adaptive control of primate saccadic system. *Journal of Neurophysiology, 44,* 1058–1076.

Perry, V. H. & Cowey, A. (1985). The ganglion cell and cone distributions in the monkey's retina: Implications for central magnification factors. *Vision Research, 25,* 1795–1810.

Robinson, D. A. (1963). A method of measuring eye movement using a scleral search coil in a magnetic field. *IEEE Transactions on Bio-Medical Engineering, BME-10,* 137–145.

Robinson, D. A. (1975) Oculomotor control signals. In Lennerstrand, G. & Bach-y-Rita, P. (Eds), *Basic mechanisms of ocular motility and their clinical implications.* (pp. 337–374). Oxford: Pergamon Press.

Ron, S., Robinson, D. A. & Skavenski, A. A. (1972). Saccades and the quick phase of nystagmus. *Vision Research, 12,* 2015–2022.

Skavenski, A. A., Robinson, D. A., Steinman, R. M. & Timberlake, G. T. (1975). Miniature eye movements of fixation in rhesus monkey. *Vision Research, 15,* 1269–1273.

Sparks, D. L. & Mays, L. E. (1983). Spatial localization of saccade targets. I. Compensation for stimulation-induced perturbations in eye position. *Journal of Neurophysiology, 49,* 45–63.

Steinman, R. M. (1965). Effect of target size, luminance, and color on monocular fixation. *Journal of the Optical Society of America, 55,* 1158–1165.

Timberlake, G. T., Peli, E., Essock, E. A. & Augliere, R. A. (1987). Reading with a macular scotoma I: Retinal location of scotoma and fixation area. *Investigative Ophthalmology and Visual Science, 27,* 1137–1147.

Van Ginsbergen, J. A. M. & Van Opstal, A. J. (1989). Models. In Wurtz, R. H. & Goldberg, M. E. (Eds), *Reviews of oculomotor research* (pp. 69–101). Amsterdam: Elsevier.

White, J. M. & Bedell, H. E. (1990). The oculomotor reference in humans with bilateral macular disease. *Investigative Ophthalmology and Visual Science, 31,* 1149–1161.

Whittaker, S. G. & Cummings, R. W. (1988). Saccade control without foveal function. *Investigative Ophthalmology and Visual Science (Suppl.), 29,* 135.

Whittaker, S. G. & Cummings, R. W. (1990). Foveating saccades. *Vision Research, 30,* 1363–1366.

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APPENDIX A

The method we used to transform the arbitrary coil voltages (e_θ and e_ϕ) into horizontal and vertical eye positions respectively was based on the equations derived by Robinson (1963):

$$e_\phi = K_1 \sin\phi \text{ and } e_\theta = K_2 \sin\theta\cos\phi \tag{A1}$$

where ϕ is the vertical angle of the coil and θ is its horizontal angle relative to the rotating magnetic field. K_1 and K_2 are constants which depend on the number of turns of the coil, its area, strength of the magnetic field and all gains of amplifiers in the paths before e_θ and e_ϕ are measured. When K_1 and K_2 are determined from the maximum voltages obtained when the coil is rotated perpendicular to the field on the vertical and horizontal meridia respectively then, arbitrary voltages e_θ and e_ϕ may be converted to angles θ and ϕ by:

$$\phi = \arcsin\left(\frac{e_\phi}{K_1}\right) \text{ and } \theta = \arcsin\left[\frac{e_\theta}{K_2\cos\phi}\right]. \tag{A2}$$

In principle, the appropriate values of K_1 and K_2 may be obtained by displacing a fixation target to cause the eye to rotate through angle CAL, measuring e_ϕ and e_θ and solving:

$$K_1 = \frac{e_\phi}{\sin(CAL)} \text{ and } K_2 = \frac{e_\theta}{\sin(CAL)\cos(\phi)}. \tag{A3}$$

In practice, the eye coil implant procedure always results in the coil being tilted by a nontrivial angle from being orthogonal to the visual axis and so violates the assumptions necessary for solving equation (A3); namely, e_θ and e_ϕ are not zero when the animal is fixating with the eye straight-ahead (eye in primary position). This coil bias can be corrected by rotating the magnetic field generating system until the field is parallel to the coil ($e_\theta = e_\phi = 0$) when the animal is fixating straight ahead but small errors could result. To be precise, these small coil biases should be measured. In addition our experiments required that we measure coil bias before and after foveal lesions when the fixation target was in the same position relative to the head to determine what part of the intact retina was being used for fixation (the new PRL). Assuming the unknown horizontal and vertical coil-biases to be $H°$ and $V°$ respectively, then moving the eye through a vertical angle CAL produced a voltage:

$$\begin{aligned} e_\phi &= K_1 \sin(CAL + V) \\ &= K_1[\sin(CAL)\cos(V) + \sin(V)\cos(CAL)]. \end{aligned} \tag{A4}$$

Substituting

$$K_1 = \frac{e_V}{\sin(V)}$$

and rearranging terms we obtained the offset $V°$:

$$V° = \arctan\left[\frac{e_V \sin(CAL)}{e_\phi - e_V\cos(CAL)}\right]. \tag{A5}$$

Where coil sensitivity:

$$K_1 = \frac{e_V}{\sin(V)}$$

was calculated from the measured potential e_v when the animal was fixating the target straight-ahead when the coil-bias angle was substantial. If coil-bias is small, then K_1 should be calculated using:

$$K_1 = \frac{e_{(CAL+V)}}{\sin(CAL + V)}.$$

The horizontal coil-bias (H^3) and sensitivity (K_2) was obtained using analogous equations for that meridian. The mean coil-bias angles for both meridia plotted in Fig. 1 indicate good stability across sessions before the lesions for both monkeys and after the lesion in M62 which indicates that the positioning of the monkeys head relative to the target was quite reliable from session to session.