# PATTERNS OF EYE MOVEMENT ADAPTATION TO FOVEAL LESIONS IN ADULT PRIMATES

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## Introduction

This paper is concerned with the way in which monkeys adapt their eye movement patterns to the loss of the central fovea. Its origin was not in a model which makes it seem a bit strange as a selection to be presented in honor of a man who's career in science has been with experiments born of tight quantitative models with little uncertainty about the outcome. One motive for doing this work was to try to uncover the oculomotor deficits that accompany human macular disease. We have succeeded in some measure and, for that reason, felt it was a useful contribution despite not having been born of a model.

In monkeys, as in humans, the foveal region of the retina is highly specialized for seeing color and detail in the environment. The primary purpose of eye movements is to move the fovea onto the portion of the scene that we want to examine in detail and then hold it there for as long as we need to extract the details. Among the disorders that fall on us as we age is one known as age related macular degeneration. The outcome of this disorder is a loss of the fovea and near peripheral retina over substantial distances. It results in the reverse of tunnel vision in which you can't see what you are looking at but can see the surround. Such patients are considered to be handicapped but not legally blind since they see something. This is understatement. The older patients with this disorder often stop reading or watching TV and stop going to the store because they can't see the price tags very well. Exactly why they have such difficulty with reading is somewhat of a mystery.

At first, an eye movement problem was suspected to underlie the reading disorder. Whittaker, Budd and Cummings (1988) and Timberlake, Mainster, Peli, Auglier, Essock and Arend (1986) recorded the eye movements of patients with macular degeneration. They found surprisingly little wrong with the patients' fixation that would account for the reading problems. We began recording the eye movements of monkeys with macular lesions to try to uncover the potential problem in the humans with macular disease. A part of our motivation was that we already had the monkeys in a study of the neurophysiological changes that could take place in the adult visual cortex. We simply undertook the eye movement study to maximize the amount of information we could get from each animal. Also, the foveal lesions were surgically produced so that we had precise information on the extent and time of the lesion and we could study the time course of the recovery process.

We trained the monkeys to fixate a small (0.1 deg diameter) target and to track it when it moved. When they were good at that task, Dr. Trempe of the Retina Foundation photocoagulated a 600 $\mu$m region containing the fovea in both eyes. The laser light was absorbed by the retinal cells and it completely destroys the neural tissue. Histology of the retinae done after the animals were sacrificed revealed only a few scattered cells within the lesion. The lesion was 3 degrees arc in diameter in the visual field. Heinen and Skavenski (1992) used the magnetic-field search-coil technique to record the fixation eye movement in lesioned animals over a prolonged



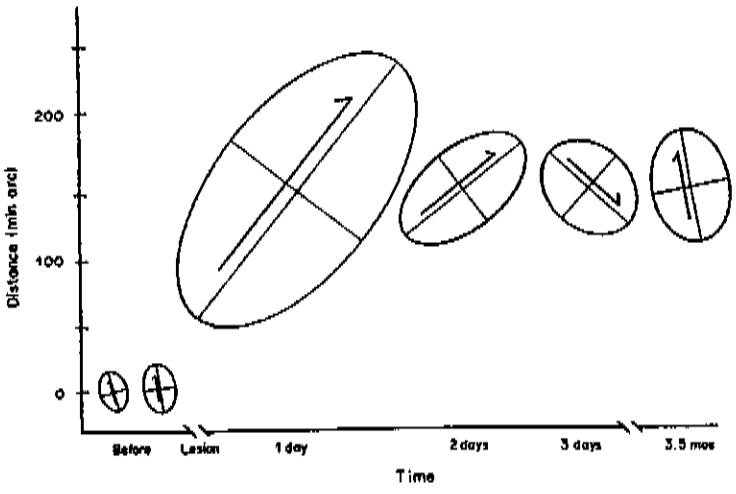


Figure 1. Bivariate contour ellipses showing fixation stability in one monkey over time. Each ellipse contains 68% of the eye position samples obtained in a representative 10 sec trial collected in several single recording sessions made at the times shown on X- axis labels before and after bilateral foveal lesions. Both the horizontal and vertical axes show distance in min arc. Our methods reproducibly place the animal's head in the same location and orientation across recording sessions to allow longitudinal comparison of absolute eye position in space.

time. The bivariate contour ellipses in Fig. 1 show that the adaptation of fixation to foveal loss was immediate.

Before the lesion, the ellipses are small, and are representative of good primate fixation. The centroids of those ellipses were used to define zero eye position on the horizontal and vertical meridia. The day after lesion there was an upward shift in the fixation position and a marked increase in the variability of fixation. Over the next few days, fixation stability tightened to a much smaller value around the same mean position. This mean position was always used by the animal for fixation and was called a preferred retinal locus or PRL for that reason. From the work of Murphy, Haddad and Steinman (1974), and Zeevi, Peli and Stark (1979) we knew that normal humans can use peripheral retina to fixate targets as well as they can using their fovea. Together, these facts suggested that adaptation of fixation to foveal loss simply meant switching on a capacity or skill that was already present.

What about other kinds of movements? Heinen et al. (1992) examined the accuracy of saccades made to track 10 deg arc target steps in the dark. The story was about the same as for fixation but the recovery was much slower. Specifically, trajectories of saccades made in the first few weeks after the retinal lesions brought the scotomatous foveae to the target. Then, the subjects made an upwards saccade to put the PRL onto the target. Over time, the saccades become more accurate but it was quite a sluggish recovery process. This was surprising to us since animals could adopt a strategy that the foveal lesion was equivalent to rotating the eye on the muscle

pair surgically. Optican and Robinson (1980) have shown that patients adapt to this condition within a couple of days. Also, Hallett (1978) had shown that subjects could readily make saccades in the direction opposite to a target step suggesting a high degree of flexibility in saccade control. Hallett's (1978) observation suggested that the retinal error signal for the saccades perhaps could be computed based on arbitrary retinal loci. However, if that were true then our monkeys should have adapted quite rapidly. The fact that they didn't suggested that the retinal error signal is normally calculated using the fovea as the origin of that vector and that it is extremely difficult to change that origin. Whittaker, Cummings and Swieson (1991) found that macular degeneration patients, who had adapted to their long standing disease made saccades of long latency and slow speeds. Those authors suggested that the saccade generation mechanism of the patients differed from the mechanism used to generate foveating saccades in normal subjects.

Although saccade adaptation was slow, substantial recovery ultimately took place and saccade behavior became fairly normal. Next, we examined smooth pursuit tracking of a small target against a totally dark background. Pursuit was as good as it is in normal subjects and some of that data is reproduced in Fig. 3. At this point, our experiments showed no substantive eye movement deficits in our animals. What is going on? Our confusion about the nature of the deficit was deepened by


Figure 2. Representative 2-dimensional eye movement recording of a foveally lesioned monkey tracking a pseudorandom sine wave target movement. Tick marks show one second intervals on the *TIME* axis and 5 deg arc rotations on the *POSITION* axis. *HS* and *VS* are the horizontal and vertical position of the target respectively. *HE* and *VE* are horizontal and vertical position of the eye.



visual psychophysical experiments by Rubin and his colleagues at Hopkins which revealed that fovea doesn't have privileged access to language centers. Therefore, there must be an eye movement deficit in the maculopathy patient.

The possibility arose that using visible backgrounds which simulate more natural scenes might reveal an oculomotor abnormality. We began by recording smooth pursuit with sine wave gratings as backgrounds because they were easy to describe. The motion of the tracking stimulus was a pseudorandom sine or triangle waveform obtained by adding two or more frequencies. Tracking was mostly smooth and good when the background was dark. However, as the luminance of the background sine wave grating was increased, the tracking started to deteriorate dramatically. This is illustrated in Fig. 2 which shows a representative record of the eye tracking a target 1.9 log units brighter than the space average luminance of the background. This was the brightest of the backgrounds we could use and still get tracking. Nevertheless, it appeared that the smooth tracking nearly disappeared and the tracking became very saccadic.

In fact, there was smooth motion when the background was 1.9 log units dimmer than the target which was quantitatively assessed by taking out the saccades and measuring displacement of the eye by smooth movement alone. We divide that amplitude of the smooth eye movement by the target displacement to get gain. When

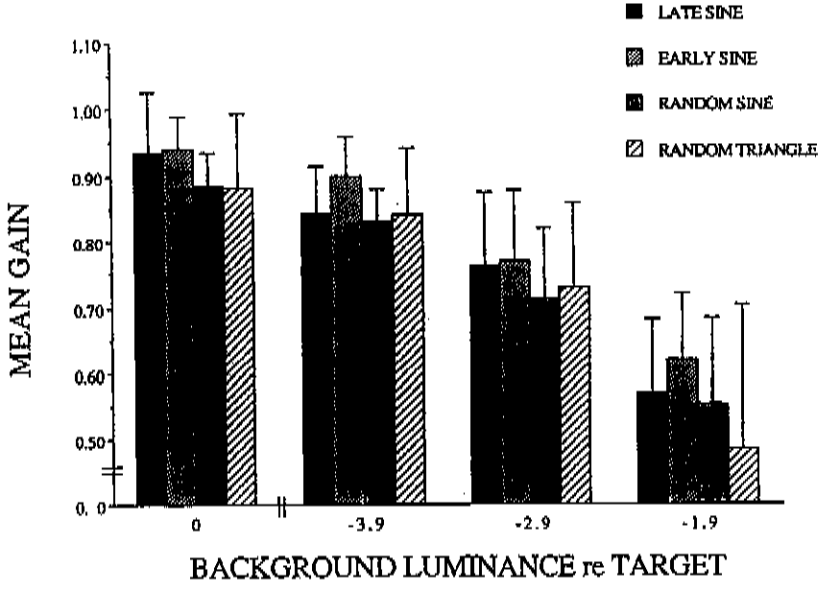


Figure 3. Bar graph showing *MEAN GAIN* of the smooth tracking component of eye motion for *BACKGROUND LUMINANCE* ranging from total dark ( 0 ) up to 1.9 log units dimmer than the tracked target. Error bars show one standard deviation. Target motion was sinusoidal or pseudorandom sine or triangle motion. *EARLY SINE* shows gain soon after retinal lesion and *LATE SINE* shows gain measured one year later.

the background was made even brighter so as to approach a real scene, the smooth eye movement components go to zero amplitude so we didn't measure those records. The bar graphs in Fig. 3 show the gain of the tracking as function of background luminance for 3 different types of target motion.

Clearly, there was a marked deterioration in the monkey's ability to follow the smooth motion as the background became more visible. Although we have decreased the salience of the target by adding the background, it is still brighter than the background and is very clearly visible. At higher background luminance the tracking becomes purely saccadic. By itself this means that the target remained visible to the monkey because they were tracking it with saccades but its motion was not effective in generating smooth pursuit.

We pursued the issue of target salience further by increasing target salience by making it bigger or changing its color to red. This caused only a modest increase in gain. We also took the step of darkening the background around the region where the target moved. Consequently, the target moved horizontally within a darkened zone in the center of the background. The set of histograms reproduced in Figure 4 show that none of these procedures caused improvement in tracking gain because gain continued to decrease when the background luminance increased.

Clearly the deterioration in smooth pursuit was not arising from the degradation of salience or visibility of tracking targets. Instead, it appeared that the

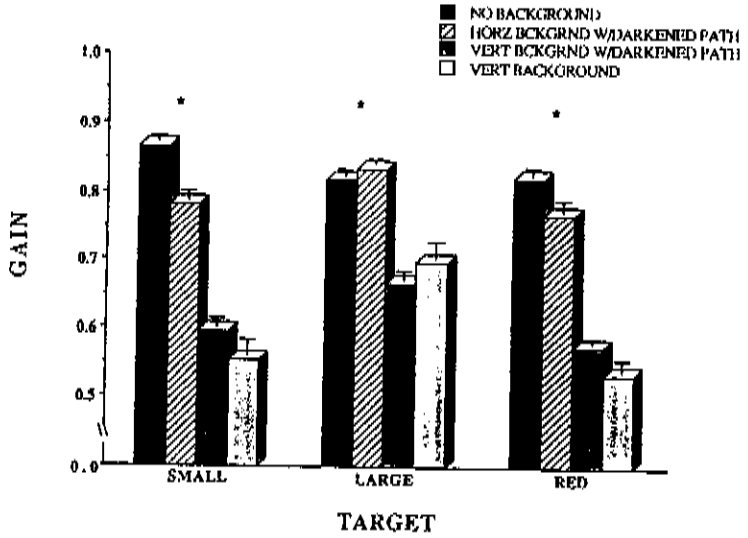


Figure 4. Bar graphs showing mean *GAIN* of the smooth component of eye motion made in tracking 3 different *TARGETs* in the presence of 4 different backgrounds. *SMALL* and *LARGE* target refer to 0.1 deg arc and 0.5 deg arc diameter targets 1.9 log units brighter than the background. *RED* target was 0.5 deg arc diameter and 1 log unit brighter than the background. The * indicate that tracking with no background or with horizontal bars resulted in statistically reliably larger gain (tested by ANOVA and *a posteriori* Sheffe test).



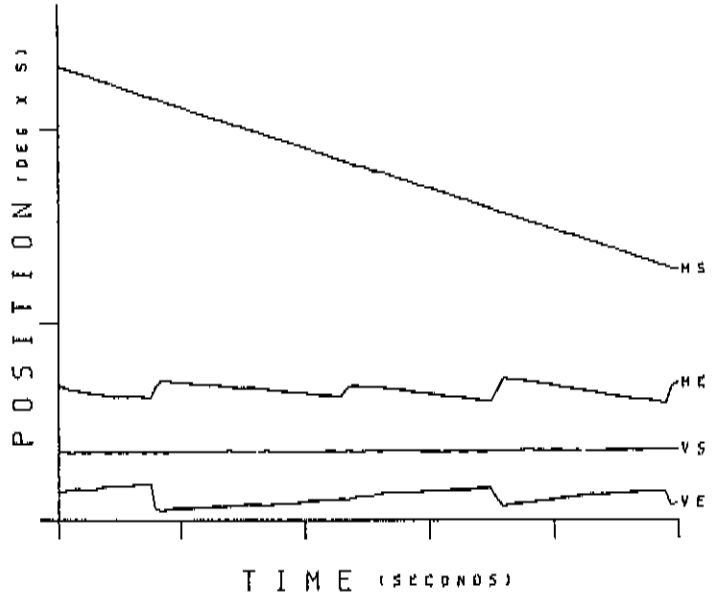


Figure 5. Representative 2-dimensional eye movement recording showing fixation of a stationary spot in the presence of moving background. The *HS* trace shows background drifting right at about 1 deg arc per second. The *HE* trace shows that the eye is dragged along with the background but at reduced speed. Other features as in Fig. 2.

retinal image slip of the background caused by the smooth pursuit could not be overcome by the motion of the target sensed by peripheral retina in the absence of a fovea. This result was quite different than the modest 20% gain reduction Collewijn and Tamminga (1984) obtained with peripheral tracking in people with intact foveae. It was also much worse than peripheral tracking that Keller and Khan (1976) obtained in monkeys with intact fovea in the step ramp paradigm with visible backgrounds.

We next examined fixation with backgrounds made up of 22 min arc checkerboard patterns or of random dots. Figure 5 shows fixation of a stationary target with the background drifting right about 1 degree per second. With moving backgrounds, the eye tended to be pulled along with the background which contributed substantial retinal slip of the object the subject was trying to fixate. That in turn degraded its visibility. What was surprising to us was that even a stationary background contributed to sloppiness of fixation. To illustrate, a background that was 2 log units dimmer than the fixation target caused the variability of eye position to go from a standard deviation of about 8 min arc to about 18 min arc on both meridia. Likewise, the speed of drift increased from about 30 min arc/sec to about 45 min arc/sec. This was very sloppy fixation!

Lastly, we examined the accuracy of saccadic tracking against lighted backgrounds. This tracking appeared very much like that seen with totally dark backgrounds described earlier. There was often some error at the end of primary saccades. This was more likely to elicit a secondary saccade in the dark. However, overall final eye position does not differ substantially with the lighted background compared to completely dark backgrounds.

**Conclusions**

This story isn't complete yet but it seems likely that the work which failed to reveal any breakdown in eye movements in patients with foveal lesion did so because they used too unnatural a stimulus to study the eye movements. When visible backgrounds are added, it seems that the smooth movement control systems that are normally accustomed to using the fovea have a great deal of difficulty in using the periphery to guide their movements. The breakdown in fixation control and saccades is not so severe that they would cause visual deficits by themselves. However, its likely that they cumulate such that the sum effect is more than the parts. In other words, there is minor deterioration of fixation, coupling with somewhat sloppier saccades, and the net loss makes it difficult for the patient to acquire and hold targets. When motivation is limited, which is often the case in the older patient, they simply give up trying. The problem becomes acute when the target begins moving and the smooth pursuit system is completely unable to follow it except under very reduced background conditions that aren't normally encountered in every day viewing.

This latter observation is of most interest to this audience, who seeks understanding of oculomotor control, because it is the first experiment I've encountered which suggests that you must have stimulation to the fovea to smooth pursue. That is, it is essential that the fovea is intact to be able to generate a smooth pursuit movement in the presence of visible backgrounds even when the smooth motion is initiated by target motion in the peripheral retina. For example, an explanation of the large difference between the gain reduction obtained by Collewijn and Tamminga (1984) in intact humans tracking peripheral targets and our study is most likely to be that our monkeys had no fovea. Why the fovea must be there even when it is not directly stimulated is not clear.

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