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What do V1 neurons tell us about saccadic suppression?

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Abstract

A series of neurophysiological experiments on the responses of V1 neurons during saccadic eye movements were carried out. Strong suppression followed by rebound was observed in post-saccadic neural activities. These results showed that although the reduction in perceptual sensitivity during rapid eye movements was largely due to the smearing of visual stimuli during saccades, there exist saccade-related extra-retinal signals mediating saccadic suppression at the neuronal level.

Keywords: Saccadic suppression; Orientation tuning; Primate visual cortex; Eye movement

1. Introduction

Saccadic suppression, a decrease in perceptive ability associated with rapid eye movements (saccades), has been known to the psychophysics community for a long time [3]. Using detection threshold and recognition rate as measures of perceptive ability, psychophysicists have concluded, by results from numerous experiments, that the smeared retinal stimulation during saccades (Fig. 1) gives a substantial contribution to the phenomenon of saccadic suppression. Nevertheless, they also recognized that there might still be a residual extra-retinal cause since saccadic suppression was found to precede the saccade onset and outlast the completion of the saccade [3]. As

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Fig. 1. Scene, retinal image during a fixation and a saccade.

early as the turn of this century, Dodge [1] proposed a central inhibition mechanism operating as to ignore stimuli that would be disturbing to clear vision. However, the existence of such central mechanism has since been debated and where this central inhibition might originate and how it works remain elusive.

Wurtz's [2,4,5] pioneering experiments on this issue showed that at the level of V1, many neurons' responses were suppressed following saccadic eye movements, and that stimulus movement produced similar neural response. The authors concluded that the suppression was an effect due to stimulus movement and that there was no evidence for extra-retinal corollary discharge in V1. We conducted a more quantitative study to re-examine this issue. We recorded from about 40 neurons in the primate visual cortex in an awake behaving monkey using epoxy-coated tungsten electrodes through a surgically implanted well overlying the V1 operculum, while a monkey was performing a saccade or a fixation task. Our results suggest that there are residual extra-retinal signals in mediating saccadic suppression at the neuronal level.

2. Results

In all our saccade experiments,[1] the first fixation spot was turned off once the second fixation spot was turned on at a distance of 6–12° away from the first fixation spot. The monkey made saccades to the second fixation spot as soon as possible.

We found that when the monkey was making saccades on a textured screen, the post-saccadic responses of V1 neurons generally exhibited a suppression phase followed by a rebound phase (Fig. 2A). We compared the response of V1 cells in the saccade condition with the fixation condition (Fig. 2B) in which the monkey was fixating a stationary red dot on the screen and then the textured background was suddenly scrolled for an equivalent distance. The eye and the background movement velocities were about 200°/s and 100°/s, respectively. We found that the V1 neurons' responses were similar in these two conditions, confirming Wurtz's finding. This result suggests that the saccadic suppression of neuronal activities is mainly caused by the smearing of the retinal image.

[1] In our experiment diagrams, saccades are illustrated by an arrow pointing from the first to the second fixation spot (both are red dots of radius 0.1°). Dashed circles illustrate our V1 neurons' receptive fields with respect to the fixation spots. Neither the dashed circles nor the arrows were part of the stimuli. In all raster and histogram plots, the top and bottom ticks on each ordinate show the peak value of the histogram and the total number of rasters (or trials), respectively.

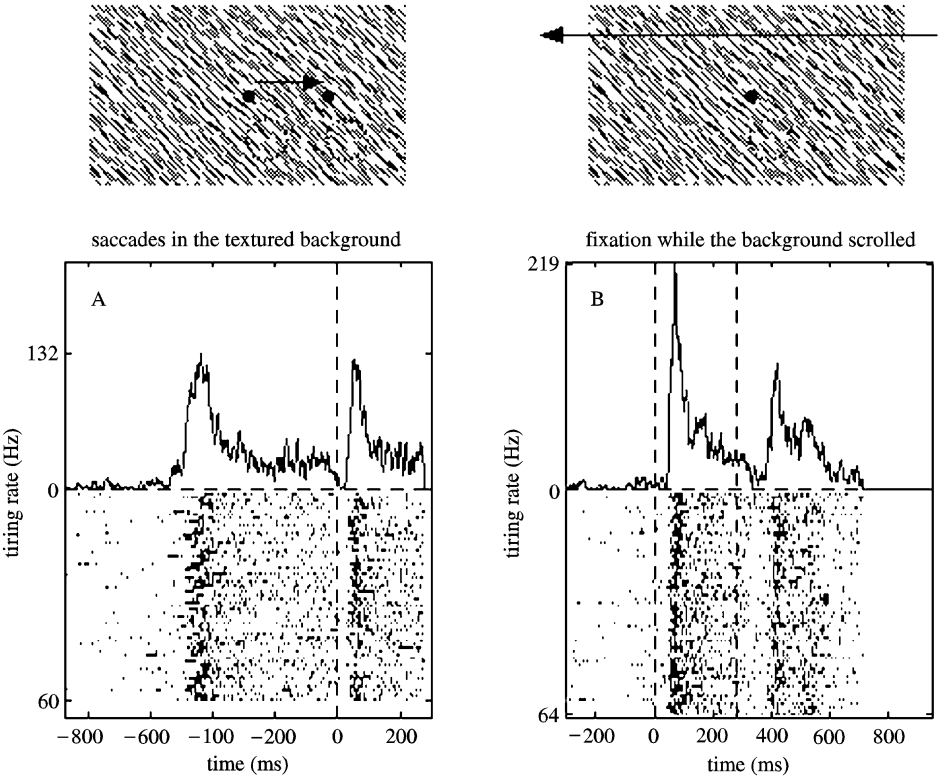

Fig. 2. Suppression and rebound were observed in a V1 cell's response (cell B073098) due to either eye movements or stimulus movement. (A) The monkey made 6° saccades in the textured background. All trials were aligned with the completion of saccades (the vertical line). (B) The monkey was fixating a red dot while the textured background was scrolled over a distance of 6°. All trials were aligned with stimulus onset (the first vertical line) and the second vertical line indicated the time when the texture started to scroll.

However, it is not certain whether the perceptive ability of V1 neurons is changed during saccades and thereby contributing to the saccadic suppression as well. We pursued this issue further by comparing the orientation tuning properties during fixation, during horizontal and vertical saccades (Fig. 3). The textured figure did not fall on the receptive field when the monkey was looking at either the initial fixation spot or the second fixation spot. It was only briefly encountered by the cells' receptive fields during saccades. Since the effect on the retinal stimulation caused by eye movements in a particular direction is equivalent to smoothing the textured stimuli along that direction, we can simulate the stimulus that the retina received during horizontal and vertical saccades. When the direction of eye movements is parallel (orthogonal) to the orientation of the texture, we refer the eye movements as parallel (orthogonal) saccades accordingly. Of particular interest here is that, the texture orientation of the image resulted from such directional smoothing remains the same during parallel saccades but is reversed during orthogonal saccades.

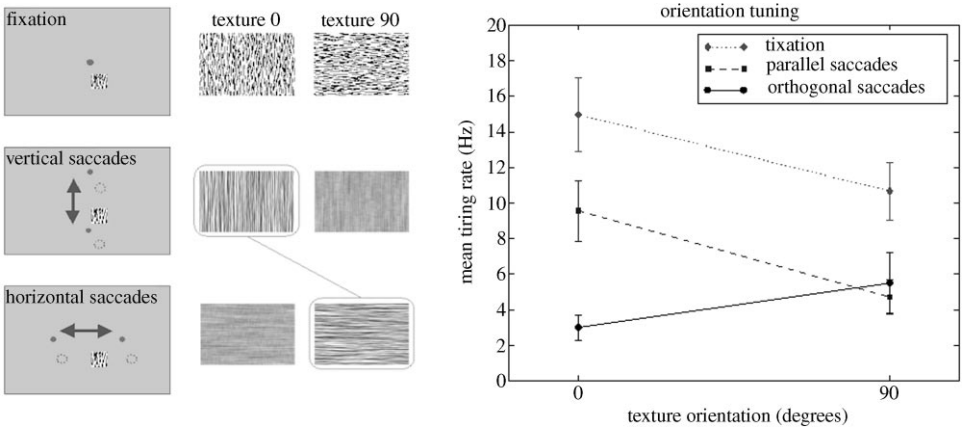

Fig. 3. Orientation selectivity of V1 neurons did not change during saccades. The left column illustrates the paradigms for the fixation, vertical and horizontal 12° saccades. The middle two columns show two texture stimuli as well as the equivalent retinal images seen during vertical and horizontal saccades. The two linked images are those seen during the parallel saccades. The rightmost figure shows the mean firing rate ± standard error of an orientation-selective V1 neuron (cell B061098) to the two stimuli under these conditions.

Indeed, Fig. 3 shows not only the reduction of response due to the weakened retinal input during saccades, but also the preservation of the orientation selectivity during orthogonal saccades as well as parallel saccades. The reversal in the orientation tuning curve for the orthogonal saccades was due to the reversal in the perceived orientation of the texture. Of 17 V1 neurons we recorded on these conditions, this result was robust for all 7 highly orientation-selective neurons. For the other 10 neurons, we found that the p-value (from the t-test on the responses to the two stimuli being the same) distribution for parallel saccade condition was very similar to that for the fixation condition. The p-value distribution for the orthogonal saccades was shifted rightward than that for the fixation condition, reflecting the fact that more texture details were lost due to smearing during orthogonal saccades.

However, we still cannot rule out the central mechanism that might contribute to the reduction of the response magnitude. When the monkey saccaded to follow a red dot on a blank gray screen, the saccadic suppression and rebound could still be observed in the activity of individual cells as well as in their population average (Fig. 4).

Assuming that the retinal stimulation did not change significantly when the eyes moved across a gray screen during saccades, this result suggests that some extra-retinal control signals must be at work to produce the saccadic suppression and rebound in the neurons' post-saccadic responses. Although it is not impossible that the mechanical properties of the eyes and the receptivity of the retina change during saccades, altering the incoming stimulation at a very early stage, we think that an extra-retinal signal, originating from some central mechanism associated with eye movement control, is likely involved in causing this suppression and rebound in the neuronal responses.

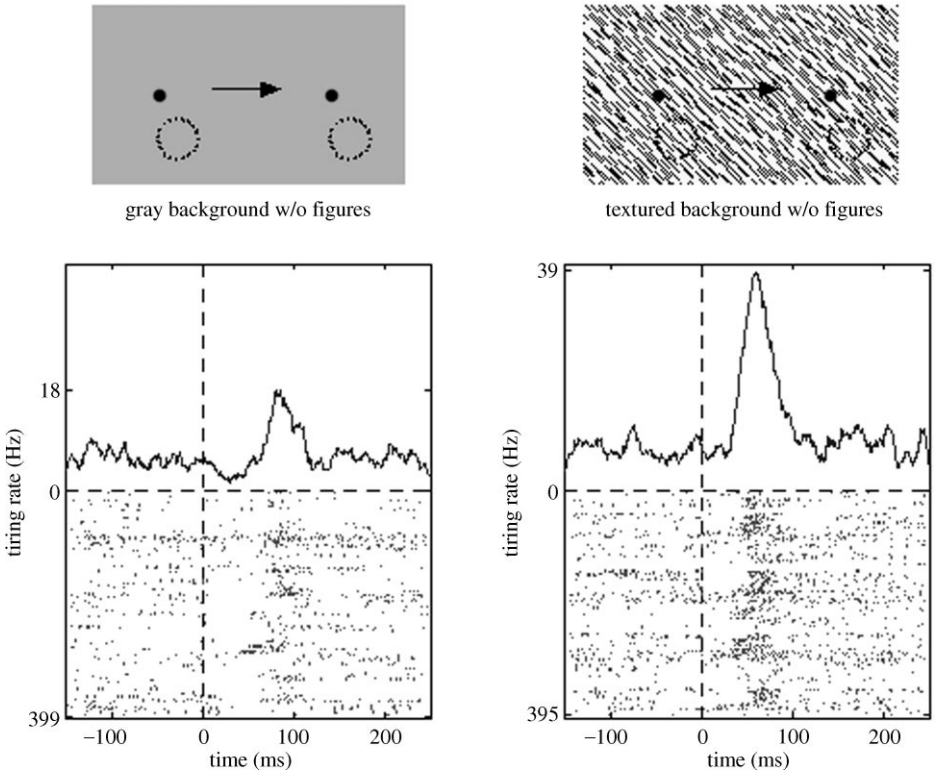

Fig. 4. 12° saccades in blank background also produced suppression and rebound in the post-saccadic response as saccades in textured background did. The former seemed to have longer response latency. These 794 trials came from 8 single-unit V1 neurons (cell B060598–B062398) and were aligned with the completion of saccades (the vertical line).

With this hypothesis in mind, we did another experiment to evaluate the impact of saccades on V1 neurons' processing of visual information. The monkey was required to make an 8° horizontal saccade in gray background. A $2^\circ \times 2^\circ$ color stimulus, covering neurons' receptive field (diameter: 0.8°–2°, eccentricity: 2.5°–13°) when the monkey was looking at the second fixation spot, was presented at different times with respect to the second fixation. In particular, we consider the cases when the stimulus was turned on long before saccades (A), during saccades (B), right after saccades (C), and long after saccades (D) (Fig. 5).

When the stimulus was turned on 300 ms after the second fixation, the eyes had already stabilized. During the 100 ms time window from the stimulus onset, the average maximum deviation of the eye movements and its standard error were 0.046° and 0.0015°, respectively. Compared to the response in this fixation condition (D), the initial responses in A, B and C were all smaller, suggesting the suppression of initial neuronal response to the stimulus seen by the receptive fields of the cells during and right after saccades.

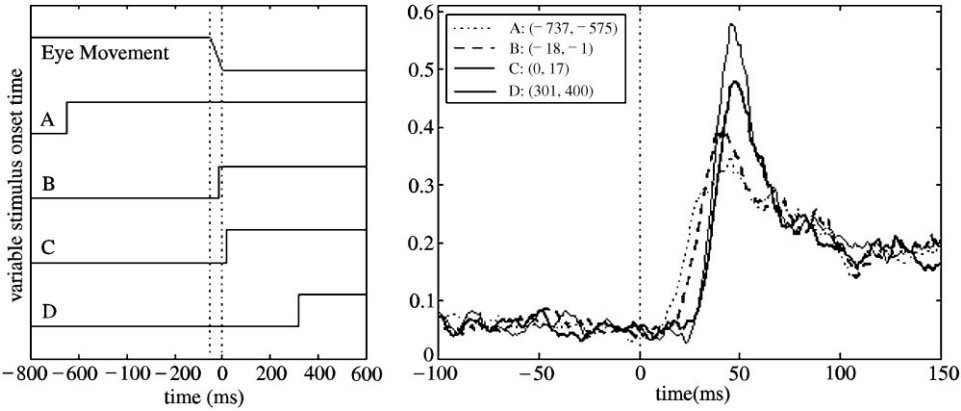

Fig. 5. V1 neurons showed different post-saccadic responses to the stimulus turned on at different times. The left illustrates the stimulus onset timing with respect to the second fixation. In condition A, the stimulus was turned on at least 575 ms before the second fixation. In condition B, the stimulus was turned on during saccades. In condition C, the stimulus was turned on right after the landing of the saccades. In condition D, the stimulus was turned on at least 300 ms after the second fixation. The right shows normalized population histograms over 18 V1 neurons (cell C081399–C082599) for the four conditions. For A and B, time 0 in the graph is the second fixation; for C and D, time 0 is the stimulus onset time. The numbers in the legend give the range of stimulus onset time.

However, we need to be cautious of confounding factors. The earlier response onset in A and B than C and D could arise from the stimulus entering the receptive field of the cells before the eyes reached the second fixation spot. In fact, the difference in response latency was greatly reduced for smaller stimuli ($0.2^\circ \times 0.2^\circ$). The reduction of the responses in A and B might be caused by the motion blurring of the stimuli, which could potentially wash out the sharp temporal edge of the visual input. Condition C had less smearing problem than A and B, but it was still possible that the reduced response was due to the continuing slow motion of the eyes before they came to a full stop after saccades. During the 100 ms time window after the stimulus onset, the average maximum deviation of eye movements and its standard error were 0.1° and 0.0039°, respectively. Though these eye movements were relatively small compared to the stimulus size and the receptive field size, they were significantly larger than those in condition D. We are not sure whether these factors can account for all the difference in neuronal responses between A, B, C and D.

3. Conclusion

First, the retina provides the dominant input to V1 neurons. Eye movement or stimulus movement could lead to qualitatively similar neuronal responses. Saccadic suppression is therefore mainly caused by the diminished visual input during eye movements. Secondly, V1 neurons continue to process visual input during saccades and the orientation selectivity of V1 neurons does not change with saccadic eye

movements. Finally, by controlling retinal input and eye movements, we showed some evidence for the existence of extra-retinal signals mediating saccadic suppression in V1. However, the potential effects due to small eye movements should be taken into account in interpreting the post-saccadic processing of visual information.

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