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Eye Movements: Keeping Vision Stable

Dispatch

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A long-standing problem for vision researchers is how our perception of the world remains stable despite the continual motion of our eyes. Three recent studies begin to shed light on how the visual system suppresses the motion generated by these eye movements.

Our eyes are seldom still: they dart incessantly from one object to another, up to three time per second (more often than our heart beats) with a series of rapid ballistic movements called saccades. Each saccade causes rapid motion of the retinal image and brings a different view of the world into central vision: yet we are unaware of both the fast motion and the continual changes, and usually of the very fact that our eyes move. Our world remains perceptually stable and constant, despite the distinctly unstable platform on which our sensors are mounted.

This problem has fascinated many visual scientists, from the 11th century Persian scholar Alhazen, to Helmholtz, Sherrington, Sperry and many others. A variety of explanations have been offered, but most researchers now agree that there must be some form of 'corollary discharge' accompanying a saccade that somehow blunts its disruptive effects. The effects of the corollary discharge are widespread (reviewed in [1]): they include suppression of vision (particularly of visual motion), remapping of receptive fields, transient perceptual compression, and even profound changes in binocular rivalry and other bi-stable phenomena [2].

In this issue, Kleiser *et al.* [3] report a functional magnetic resonance imaging (fMRI) study in humans, in which they examined the 'BOLD' responses to brief stimuli presented either before or during a voluntary saccade. Using an event-related paradigm, they were able to show that many visual areas respond significantly to these stimuli, and that in some areas activation is reduced during saccades. As the authors point out, a generic reduction is open to many possible artefacts: signals resulting from saccadic planning or execution, from positional uncertainty, smearing, or some non-specific attentional or visual masking effects. They controlled for this possibility intelligently by taking advantage of psychophysical studies showing that equiluminant chromatic stimuli are not suppressed during saccades [4], assuming that any difference in the suppression of luminance or chromatic stimuli would reflect stimulus-selective saccadic suppression. As shown in Figure 1, they observed stimulus-selective saccadic suppression in three distinct neural areas: areas hMT+ (also called V5), V7 and V4. In all three areas, the response to luminance gratings was significantly reduced, while that to chromatic gratings remained unchanged.

Area hMT+ is a part of the human brain equivalent to areas MT and MST in the macaque brain, which are specialised for visual motion and known to receive strong input from the so-called 'magnocellular' dorsal stream. Saccadic suppression of this area is consistent with psychophysical studies suggesting that suppression is specific to the magnocellular stream [4], and also that motion-selective mechanisms are severely damped during saccades [5–7]. It is also consistent with the electrophysiological measurements from single units of MT in macaques, which often show strong suppression during saccades [8].

V7 is a parietal area of the dorsal stream which is also related to eye movements and which also receives a strong magnocellular input. The suppression in V4 is less expected, as it is part of the ventral stream and thought to be involved mainly with form and colour processing. However, V4 also receives a substantial magnocellular input [9], consistent with the suppression of this stream. And although V4 is not generally considered to be a motion area, fMRI studies in macaque demonstrate strong direction selectivity in V4, stronger even than in MT [10]. Kleiser *et al.* [3] also observed some suppression in other areas, such as V1, V2 and V3, but this either did not reach statistical significance, or did not differ significantly for chromatic and luminance gratings. They correctly conclude that, while they have no firm evidence for saccadic suppression in these areas, they cannot exclude this possibility from their non-significant results.

As they obtained different results for different cortical areas, Kleiser *et al.* [3] conclude that saccadic suppression does not occur early in visual processing, but rather at multiple cortical sites within the magnocellular stream. This conclusion goes against many psychophysical studies which have suggested that saccadic suppression occurs early in the visual system, at or before the site of contrast masking [5], which probably occurs in the primary visual area V1, and before low-level motion processing occurs [8].

In another recent study, Thilo *et al*. [11] addressed this question more directly with a clever electrophysiological technique. Replicating an old study of Riggs *et al.* [12], they have shown that visual phosphenes produced by electrical stimulation of the eye are suppressed during saccades (circles in Figure 2). But phosphenes of cortical origin — V1 or V2 — generated with the modern technique of transcranial magnetic stimulation (TMS) are not suppressed (squares in Figure 2). This strongly suggests that saccadic suppression occurs early, before the site of generation of cortical phosphenes, probably within the visual thalamus — the lateral geniculate nucleus (LGN) — but perhaps within V1 itself.

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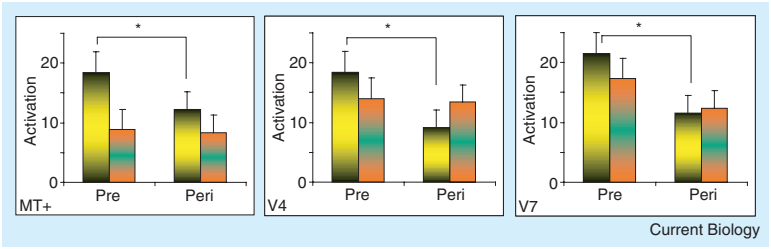

Figure 1. Cortical areas in which significant selective suppression of luminance stimuli was observed in the recent study by Kleiser *et al*. [3].

The yellow–black bars refer to responses to luminance modulation, the red–green bars to chromatic modulation. 'Pre' refers to measurements made before the saccades, 'peri' to measurements during saccades. In all cases the luminance response was significantly suppressed during saccades (indicated by the asterisks) but the chromatic response was not.

How do the two recent studies [3,11] relate to the evidence from animal physiology? There have been extensive electrophysiological studies in cat and monkey, far too many to mention here (reviewed in [1]). It is worth noting, however, that there exist strong connections between brainstem and thalamus that are activated during saccades [13], providing a plausible mechanisms for saccadic modulation of thalamic response. Many studies have demonstrated such modulation of LGN activity, but perhaps the most comprehensive study is that of Reppas *et al.* [14]. Using a sophisticated statistical technique, they calculated impulse response functions of monkey LGN cells in response to quasi-random sequences of large-field flicker. Voluntary saccades induced profound changes in the responses, particularly of magno cells: activity was depressed around the time of the saccade, followed by a larger and longer-lasting enhancement. This suggests that saccadic suppression occurs, at least in part, in the LGN, consistent with Thilo *et al.*'s [11] result and with earlier psychophysical data.

Many other parallels with the psychophysics were found, including a similar timecourse [15], a stronger effect on magno than parvo cells [4], and an acceleration of the impulse response function [16]. One notable difference is that the neural responses of primate LGN cells undergo a very strong post-saccadic enhancement, much stronger than the suppression [14]. Most studies on humans — including that reproduced in Figure 2 — show suppression rather than enhancement (although post-saccadic enhancement has been reported for stimuli of high spatial frequencies [5] or for chromatic modulation [4], and sensitivity is greater shortly after a real than a simulated saccade [15]). There could be many reasons for a general failure to show enhancement, including possible ceiling effects in absolute sensitivity, so the enhancement is not seen at threshold (similar to attentional effects [17]). This would certainly be an interesting line to explore.

But can we conclude that suppression occurs only in the visual thalamus? Of course not. The problem of saccadic suppression has proven to be so elusive it is highly likely to occur in different forms at multiple sites. The differential attenuation of BOLD activity in various areas [3] points to additional post-thalamic processes. And a recent study by Thiele *et al.* [8] shows that MT cells display very interesting behaviour during saccades that cannot all be ascribed to an early suppression: some are suppressed, while others reverse their preferred direction selectivity, presumably to cancel motion information carried by other cells, and help keep the world still.

It is not surprising that saccadic suppression should occur at different levels. Many basic sensory phenomena, such as gain-control, do not occur at a single site but at virtually every possible location: photo-receptors, retinal ganglion cells, LGN cells and cortex [18]. Indeed the parallels between saccadic suppression and contrast gain control are so strong it has been suggested that saccadic suppression may use the contrast gain control mechanisms to regulate sensitivity [15,16] (also explaining why the magnocellular pathway is most affected, as magno cells have stronger gain-control mechanisms than parvo cells

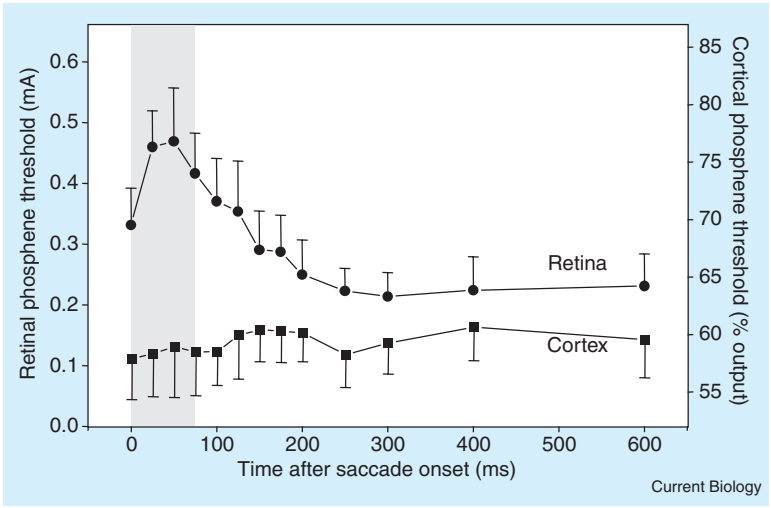

Figure 2. Thresholds for generating retinal (circle symbols) or cortical (square symbols) phosphenes, as a function of time after saccade.

The thresholds for the retinal phosphenes were greatly increased during the saccades (indicated by the grey region), while the cortical phosphenes were unaffected. This indicates that saccadic suppression precedes the generation of cortical phosphenes, occurring either in visual thalamus or in V1 itself.

[19]). This would certainly be an elegant and economic solution to the problem of saccadic suppression, taking advantage of mechanisms already in place for other functions.

What is certain is that, while significant gains have been made over the past few years, we are far from a complete understanding of all the mechanisms that lead to the stable and seamless perception from our continually moving sensors. This area will provide a rich source of research questions of the next few years, probably giving general insights of visual function.

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