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Review article

Suppression of the magnocellular pathway during saccades

John Ross [a], David Burr [b,c,*], Concetta Morrone [b]

[a] *Department of Psychology, University of Western Australia, Perth WA 6009, Australia*
[b] *Istituto di Neurofisiologia del CNR, Via S. Zeno 51, Pisa, Italy*
[c] *Dipartiment di Psicologia, Università di Roma 'la Sapienza', Via dei Marsi 78, Rome, Italy*

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Abstract

Saccades create two problems for the visual system: they cause fast (but resolvable) motion of the retinal image and a change in the relationship between retinal and external spatial co-ordinates. In this review, we examine the first of these problems, of why there is no disturbing sense of motion during saccades. Recent evidence from a range of sources suggests that during saccades, the magnocellular pathway is selectively suppressed, while the parvocellular pathway is functionally unimpaired, or even enhanced. The suppression seems to occur early, possibly in the lateral geniculate nucleus, where the pathways are well separated. It is possible that the suppression shares similar mechanisms to those responsible for contrast gain control.

1. Introduction

Saccades, the rapid ballistic movements of the eyes that we make to foveate what catches our attention, pose at least two related problems for visual science. The first is why we notice no image motion; the second is why we do not notice any image shift. This is, of course, not the end of the list. Other questions abound: How do we initiate saccades? How do we aim them? How do we combine saccades with other eye movements, like smooth pursuit and vergence (even other saccades), and with movements of the body and the head? (Imagine the saccading acrobat!)

This review will concentrate on the first problem, why we notice no motion when we make saccades, and on evidence suggesting that the visibility of motion is selectively suppressed by suppressing activity in the magnocellular pathway during saccades.

2. Displacement and motion

So closely related are the first two problems mentioned, movement and shift, that they have been taken to be one and the same. Even Zeno and other classical philosophers would not doubt that to see something move is to see it shift position, and to see something shift position is to see it move. But they are distinct. Things may shift position while escaping our motion detectors, and excite them while going nowhere. If a disc is ruled with a grating fine enough, or dotted with spots small enough, and the contrast is low enough, it may be rotated yet seem not to move [14]. Yet, paradoxically, though no motion is seen, the bars of the fine grating can be seen always to remain parallel to those on a companion disc, ruled more coarsely and at high contrast, which appears to move vigorously as it is rotated at the same speed. So the shift in position can be appreciated, even though there is no direct sense of motion.

Equiluminance provides another example. If a grating is made by modulating colour, while keeping luminance constant, it can be drifted without seeming to drift [18]. But once again, we can keep track of shifts of position and notice the grating keeping up with a more conventional companion, which is seen to move. It is also the same for the hour and minute hands of a clock. The rate at which they move is too slow to be detected, but we count on seeing their shift in position so that we can tell the time.

In both cases, the explanation is the same. Our special stimuli, the fine, low contrast patterns, or the equilumi-

* Corresponding author.

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nant ones, and the minute and hour hands of a clock are outside the range to which our motion detectors are tuned. They therefore escape detection on the basis of being seen to move, but we catch them out by noticing (by other means) that they did not stay in the same place.

The simplest examples of motion being seen without there being a shift in position are the motion aftereffects: the waterfall illusion, where the cliff face near a waterfall seems to move upwards when we transfer our gaze to it from the waterfall, the spiral that seems to rotate backwards and shrink after it stops spinning, and so on. Here motion detectors are adapted by motion in such a way that something that normally would not excite them does so, if only briefly. We respond to their signals by seeing false motion. A brief flash also excites them, and seems to be accompanied by motion, of a somewhat ambiguous kind. We also see cinematic (and TV raster) motion as real, when what is presented to us is a series of stationary (or raster-swept) images. Indeed, under some circumstances, such as brief exposures of complex gratings [22] and the so-called 'reversed-phi motion' [3], motion may be seen in the opposite direction to the direction of the physical displacement.

So we can find examples on both sides, of shift without motion, and motion without shift, thus decoupling what might be thought to be indissolubly coupled. There is, however, an asymmetry. If we detect a shift, we are not thereby caused to sense motion; but if we detect motion, we do sense a shift, even if none has occurred. Motion carries an imperative that shift of position does not.

3. Is there really a problem of motion during saccades?

Having established that motion and image displacement are not necessarily the same, let us examine why the motion created by fast saccades is not sensed, and does not disturb vision. One idea is that image motion in saccades poses no problem because it is too fast to be seen. It was first proposed by Dodge [23], echoed by Woodworth [60], and confidently reiterated by Matin [37]. Campbell and Wurtz [15] claimed motion at saccadic speeds produced a 'grey-out', and proposed that it was not seen because it was masked by clear perceptions before and after a saccade. The idea was repeated as recently as 1994 by Bridgeman et al. [7]. But simple though the idea is, there is convincing evidence to show that it is not the case. The peak velocity in large saccades (20° and above) is about 500 degrees per second (deg/s) [17], very fast, but not fast enough to smear the image into a grey blur. Burr and Ross [10] show that we can see perfectly well at such image velocities, provided that there are sufficient low spatial frequencies in the image. High frequency components are indeed wiped out, but components of lower spatial frequency, inconspicuous when the image is still, become visible with image motion. Fig. 1 summarises these results. They show that there is no loss of visual sensitivity at saccadic image velocities; just a shift in what we see well, from high on the spatial frequency scale to low, where energy in natural images happens to be greatest ([25]: reproduced in Fig. 1B).

So if vision operated normally during saccades we would be startled every time we made one by a rush of motion, just as we are by jerky camera motion or a bumped projector. There is a problem to be solved.

One of the earliest solutions was proposed in 1866 by von Helmholtz [26], who suggested that we do indeed sense motion during saccades, but do not notice it because it provides information that is absorbed into our perception. Just as we use image size and shape to perceive distance and slant, Helmholtz asserted, so we use image motion to keep track of the motion of our eyes, correct for it, and so keep the world stable. We can see in Helmholtz's solution the germs of two later, closely related ideas, efference-copy [27] and corollary discharge [54]. The idea is that signals from image motion are cancelled by signals concurrent with, or generated by the saccade itself, with the result that no motion is sensed and a record of where the eye is pointing is updated continually throughout the saccade.

It is difficult to gainsay either Helmholtz's solution or the ideas of efference-copy and corollary discharge, but as we understand more and more about how motion is processed they become increasingly implausible.

4. Sensing motion

There are now several models of motion detectors. Some are based on the idea of correlation of activity at two different sites, input from one site being delayed (e.g., [46,48]). Some are based on the idea of filters oriented in space-time (e.g., [1,8,12]). Still others are based on the idea that motion is sensed from spatiotemporal gradients (e.g., [24,53]). These models differ in important respects, but all are capable of responding to apparent rather than real motion, and can explain certain motion illusions, like reversed phi-motion. Nothing actually has to move for them to respond.

5. The magnocellular pathway

There are two major pathways reaching from the retina deep into the brain (see [57]), the magnocellular (M) pathway and the parvocellular (P) pathway. Ninety percent of all retinal ganglion cells in monkeys feed in to one or the other of these pathways, the majority (80%) to the P-pathway, and the minority (10%) to the M-pathway.

In the retina, M-cells have higher contrast gain and

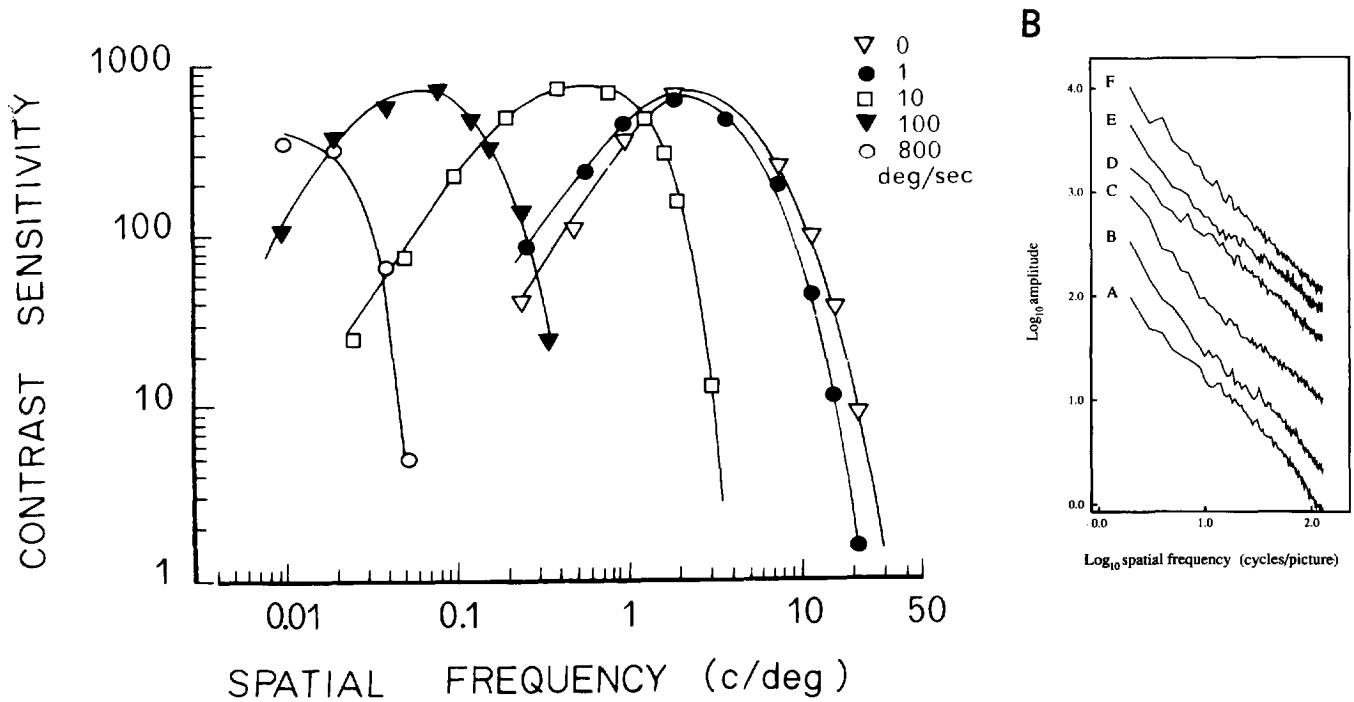

Fig. 1. A: the 4 similar inverted-U curves are taken from Burr and Ross [10]. They show how contrast sensitivity changes with velocity, indicated by labels in the figure. There is no loss of sensitivity, even at image velocities of 800 deg/s, but as velocity increases, the spatial frequency at which vision is most sensitive slides down the spatial frequency scale. B: the right hand curve is taken from Field [25]. It indicates the amplitude found in typical natural images over a range of spatial frequencies. Since amplitude is greater at lower frequencies than at high, the effect of increasing image velocity is to increase motion power.

lower contrast threshold than P-cells ([19,31,45]). This difference persists in the LGN [30] and in V1, where contrast-response functions are steeper, and neurones show an active adaptation [49]. In the retina, and at later stages, M-cells have shorter latencies than P-cells [43]).

In the retina, M-cells, but not P-cells, respond so as to convey useful information about low spatial frequencies at high temporal frequencies. Information about motion at high velocities is thus carried by the M-, but not by the P-pathway (see [32,40] for review). Fig. 2, taken from [39], shows estimates of the spatiotemporal contrast sensitivities of the M- and P-pathways, derived from experiments with ibotenic lesions in the P- and M-layers of the LGN. The high temporal and low spatial frequencies (i.e., high velocities) are subserved primarily by the M-pathway.

From the retina, both pathways proceed to the lateral geniculate, where separate layers subserve each pathway. Both pathways then pass through V1 and V2, M via layer 4B of V1 and the thick stripes of V2, P via the blobs and inter-blobs of V1 and the thin stripes and inter-stripes of V2. Thereafter M proceeds to mainly to the MT (the putative motion centre) and on to the posterior parietal cortex, and P mainly to V4 (the putative colour centre) and the inferior temporal cortex.

That the M-pathway provides the major input from the LGN to the MT is established by the fact that damage to the M-layers of the LGN consistently reduces responses in the MT, while damage to the P-layers has little or no effect [38]. But the separation between the two pathways is not as clean as the description above might imply: there is cross-talk at various stages. Nor is the division of the labour of information processing as clean as is sometimes supposed (M is for where, P is for what, M is for motion, P is for position, and so on). Yet it is safe to conclude that most motion at saccadic velocities (certainly motion of components orthogonal to the path of motion) is, in normal vision, handled entirely by M, since those velocities place most image content outside the range that P can handle (see Fig. 2).

What we know of these pathways comes mainly from work on the brains and the vision of monkeys, but it seems safe, from genetic and psychophysical similarities between ourselves and them, to conclude that this knowledge applies to ourselves. There is clinical evidence for this conclusion. A patient studied by Zihl et al. [62] sees normally in most respects, but has difficulty seeing

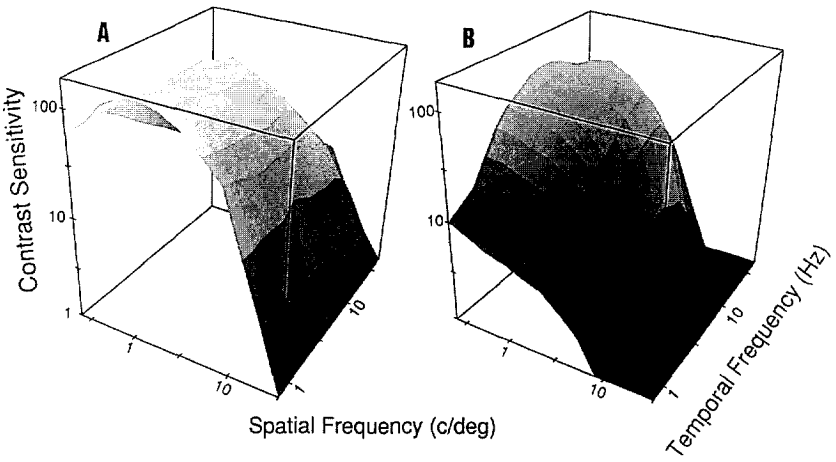

Fig. 2. Estimates of the spatiotemporal contrast sensitivity of the P-pathway alone (A) and the M-pathway alone (B), derived from selective lesions to the P- and M-layers of the geniculate (reproduced with permission from [39]).

motion, suggesting that motion perception is separately processed in the human brain, as it is in monkeys. This patient has a lesion, as positioned by computer tomography and magnetic resonance imaging, likely to fall in the human analogue of the monkey MT. Recent studies with functional magnetic resonance imaging have shown this region to be selectively active, both during real motion and the motion after-effect [55]. Other imaging techniques, such as PET and MEG show similar results ([2,61]).

6. Vision during saccades

Holt [28] proposed that vision simply shut down during saccades, by a process he called 'central anaesthesia'. However, this brute force solution can be dismissed by numerous studies that show that vision is possible during saccades, and even by casual observation, such as making a saccade across a star or similar bright object. Under some conditions, during saccades, we can actually make things visible that normally are not, like the spokes of a moving car's wheel, the blades of a fan, or sleepers viewed from a moving train (see also [41]).

Latour [34] and Volkman [58] measured saccadic suppression for a very brief flash of white light, and reported a maximum suppression of 0.5 log units, often less. MacKay [36] reported sensitivity losses of this order in normal vision for a spot flashed on a moving background, leading to the suggestion that it was the image motion causing the suppression, not a central non-visual signal. Others have found no suppression at all during saccades [33]. Certainly, the limited suppression revealed by the early studies was not enough to explain the lack of motion during saccades.

Burr et al. [11] tested vision during saccades with luminance-modulated gratings extending well beyond the path of the saccades. The saccades ran parallel to the bars of the gratings, so the images of the gratings themselves were not moved by the saccades (Fig. 3). (No other stimulus arrangement, except a blank field, allows the simplification of providing unchanging stimulation to the saccading eye.) The gratings did not move, but because they were flashed briefly (and therefore had ample energy at high temporal frequencies) they would, under normal circumstances, activate the M-system, provided their spatial frequency was low enough to be within its acceptance range. (Note that they have a wide spread of temporal frequencies, and so do not exclusively activate the M-system.)

Sensitivity was reduced during saccades, but selectively. It was not simply that vision was worse. At very low spatial frequencies much more contrast was needed to see a grating flashed in a saccade than one flashed when the eye was still. But at higher spatial frequencies, there was no difference, or even a slight enhancement of sensitivity by saccades. This can explain the variability of earlier measures of sensitivity loss, since it shows that the size and duration of saccadic stimuli are crucial. It also suggests that it is the M-system that is disadvantaged in saccades, and that the P-system is not disadvantaged at all, perhaps is even advantaged because of lack of competition from its usual companion.

A supplementary finding of the Burr et al. [11] study was that the perception of interrupted motion was blunted by saccades. A horizontal grating of low spatial frequency drifted down a screen. Abruptly, either on a signal or during a saccade, it jerked backwards then continued drifting as before. It had to be jerked much more when it occurred during a saccade to be visible than when it occurred during normal vision. Perhaps more significantly, a jerk within a saccade caused no startle, whereas normally it causes much.

This may be because the M-pathway not only carries motion signals to the MT, the putative motion centre, but also leads to other areas implicated in attention.

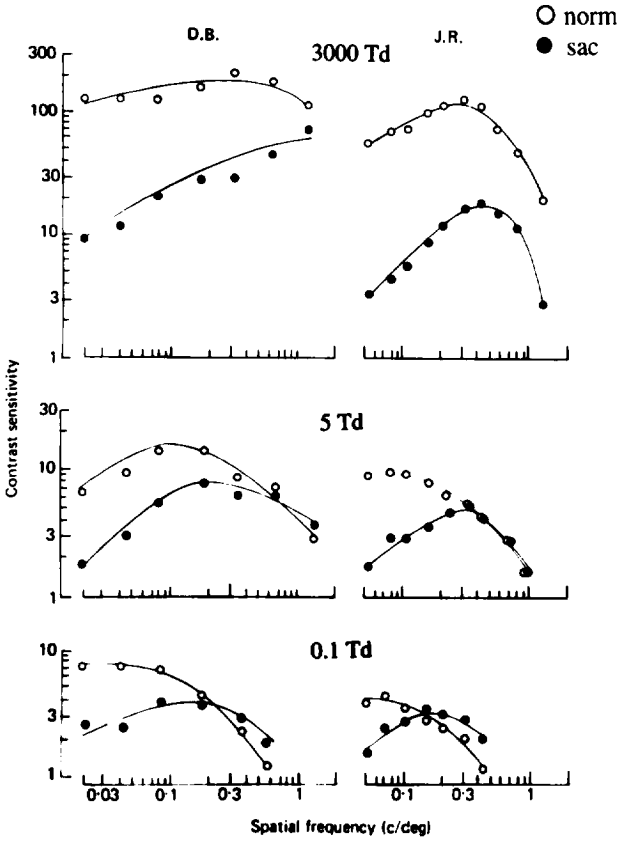

Fig. 3. Contrast sensitivity for detecting a briefly presented (20 ms) horizontal sinusoidal grating during normal viewing (open symbols) and during large (30°) horizontal saccades (reproduced with permission from [11]). As the saccades ran parallel to the grating bars, they produced no effective image motion. Contrast sensitivity was measured as a function of spatial frequency, for 3 different levels of ilumination. At all illuminations, sensitivity was suppressed during saccades, but only at low spatial frequencies. At higher spatial frequencies, the suppression becomes progressively less. At lower illumination levels, the range of frequencies suppressed during saccades becomes less. At the very lowest illumination, sensitivity was actually better during saccades than during normal vision. All these results are consistent with a dampening of motion perception during saccades.

Thus the usual effect of large images moving rapidly, to alert attention, may be diminished, along with the strength of motion signals themselves, by suppression of activity in the M-pathway.

Failure to see motion, or greatly diminished sensitivity for it within saccades, has been confirmed by Shiori and Cavanagh [52], and by Ilg and Hoffmann [29]. Shiori and Cavanagh displayed a field of random dots. When observers made saccades, the whole field was moved up, down, left or right either before, during or after the saccade. The finding was that judgements of the direction of the shift were at chance levels if the shift occurred during, or shortly before the saccade. At other times it was easy to see. Ilg and Hoffmann also displayed fields of random spots up, down, left or right, but they compared thresholds for seeing a shift when it was within a saccade with thresholds for normal viewing. Thresholds during saccades were greatly elevated.

More recently, Burr et al. [13] again used gratings with bars parallel to saccades, to avoid the effects of retinal smear, but this time modulating either in luminance as in their 1982 study, or in colour at equiluminance, so as to favour the parvo system (Fig. 4). The earlier finding was confirmed for luminance modulation: loss of sensitivity was found only at low spatial frequencies. The striking new finding was that there was no loss of sensitivity at equiluminance whatever the spatial frequency. That is to say, detection of modulation in colour was at normal levels or better during saccades.

Indeed, under some circumstances, colour was seen better during saccades than it normally is. Fig. 5 shows sensitivity for discriminating colour (green from red) or luminance (white from black) as a function of delay after

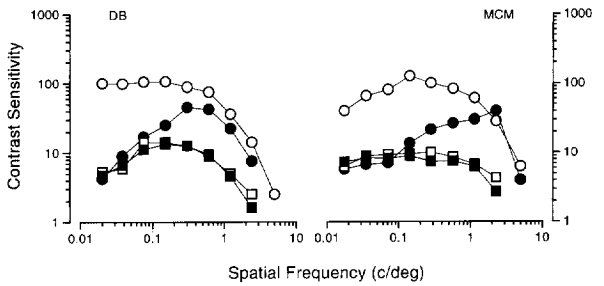

Fig. 4. Contrast sensitivity for detecting a briefly presented (16 ms) horizontal sinusoidal grating during normal viewing (open symbols) and during large (40°) horizontal saccades, for stimuli modulated in luminance (circles) or colour (squares) (adapted with permission from [13]). For luminance contrast, there was strong saccadic suppression at low spatial frequencies, as before. For colour contrast, however, there was no suppression at any spatial frequency.

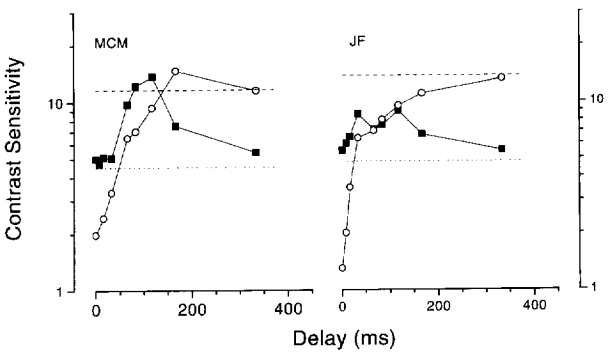

Fig. 5. Contrast sensitivity for discriminating whether a horizontal Gaussian strip was brighter or darker (open symbols) or redder or greener (filled symbols) than the background, as a function of latency after the beginning of a large horizontal saccade (reproduced with permission from Burr et al. [13]). The dashed and dotted lines show the sensitivities for the luminance and colour discriminations in normal viewing. The luminance discrimination is greatly impaired during saccades, and gradually returns to baseline conditions after 150 ms or so. The chromatic discrimination, on the other hand, is actually better soon after the saccade. Subject J.F. was naive as to the aims of the experiment.

the onset of a saccade. Discrimination of colour was at normal levels near the beginning of the saccade, but actually improved for a period afterwards. This is strong evidence that the parvo system is spared during saccades. Indeed it seems to work better than ever.

Convincing confirmation for this conclusion is provided by Uchikawa and Sato [56] using a technique of a very different kind. They measured incremental spectral sensitivities for circular targets on a white background both in normal viewing and in saccades. During saccades, they established the clear presence of a Sloan's notch, a signature of chromatic opponency, and hence of P-system function. Fig. 6 shows these results superimposed on sensitivity measures of M- and P-ganglion cells, taken from Zrenner [63]. Clearly, the change in the dependence on wavelength during saccades parallels closely the behaviour of M- and P-cells.

Interestingly, similar effects occur during blinks. Spectral sensitivity curves measured just before the eyes close during an eyeblink are similar in form to the filled squares of Fig. 6 [47], reinforcing previous suggestions that blink and saccadic suppression share the same mechanisms [59]. As the problems created by blinks are similar to those of large saccades (a large, low-frequency luminance transient), it would be parsimonious for the same mechanism to work in both cases.

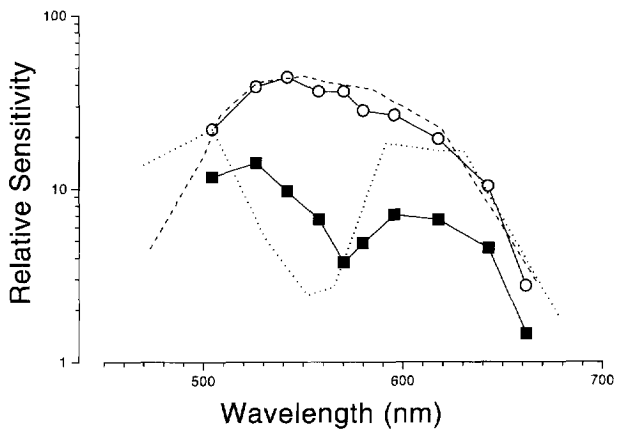

Fig. 6. The symbols show psychophysical increment thresholds for a briefly presented monochromatic light of various wavelengths, against a white background, in normal viewing (open circles) and during 6° saccades (filled squares) (adapted from [56]). Apart from an overall suppression, the form of the curve changes during saccades, to reveal the classic 'Sloan's notch', a dip in sensitivity for lights near 570 nm. Sloan's notch is the signature for colour opponent mechanisms. The broken lines show sensitivity measurements for macaque monkey retinal ganglion cells: the dashed are the average of *X* M-cells, and the dotted line the average of *Y* P-cells (taken from [63], and scaled in absolute sensitivity to best fit the psychophysical data). While the psychophysical thresholds in normal vision follow quite closely, the sensitivities of the M-cells, those in saccades are closer to that of the P-cells, with the clear dip indicating chromatic opponency (the thresholds are not identical, but many factors could explain that). This again suggests that only the magno pathway is suppressed during saccades.

Bridgeman and Macknik [6] have yet another line of evidence leading to the same conclusion. They measured the detectability of the shift during a saccade of a red square standing on a black background (to give a luminance contrast) or on an equiluminant green background (to give pure colour contrast). They found detectability to be better during saccades in the equiluminant condition, whereas in normal viewing, detectability is then worse.

This review has concentrated on studies that attempt to isolate the stimuli from extraneous visual cues, and ensure that the saccade produced no actual retinal motion. However, in natural conditions, the retinal image moves, and is preceded and succeeded by sharp stationary images, which may 'mask' the sensation of motion, as suggested by many (e.g., [36]). Indeed there is good experimental evidence that this sort of masking can occur, even with spatially distinct stimuli. Interestingly, these masking effects follow a similar pattern to those of saccadic suppression: they are confined to low frequency stimuli, modulated in luminance ([20,21]). More recently, it has been shown that forward and backward masking can change the spectral sensitivity curves, in a similar way to that which occurs during saccades shown in Fig. 6 [50]. This would suggest that in natural viewing conditions, both visual and non-visual factors co-operate in a similar way to suppress the magnocellular pathway.

7. Why, where and how?

Suppressing the M-system prevents what would otherwise be an alarming rush of motion every time we made a saccade, particularly a large one where image velocity can be 500 deg/sec or more and the traverse at least 40°. This provides a motive for the suppression.

But where is the suppression applied and how is it achieved? One likely site for a suppression mechanism to act is early, in the lateral geniculate, where P- and M-systems are conveniently separated out into layers. Another possibility is that suppression occurs later, blocking the access of information to the MT. Burr et al. [13] attempted to decide between early and late mechanisms using a masking technique. Both a test grating patch and a mask grating were presented, either simultaneously within the saccade (simultaneous masking), or with the mask within the saccade and the test after (forward masking) or the test within and the mask after (backward masking).

The results favour an early suppression. A mask within a saccade acts as if it has already suffered the effects of suppression, and so does a test. Since extensive evidence locates contrast masking effects in V1 ([5,42,44]), suppression must have had its effects at an earlier stage,

and, if we exclude the retina, the lateral geniculate nucleus becomes the most likely suspect.

At this stage, there is no direct evidence as to how suppression of M-function may be achieved during saccades. However, a possibility raised by us recently is that saccadic suppression may act through the mechanisms that regulate the contrast gain of geniculate and cortical visual cells [9]. In the monkey, retinal M-cells (but not P-cells) show dynamic contrast gain control (e.g., [4]): gain decreases at higher levels of contrast, increasing the working range of the cells. Automatic gain control mechanisms at all levels of the visual system seem to have two characteristic signatures: the attenuation is divisive (e.g., [16]), and the impulse response accelerates at higher contrasts (lower gain levels: e.g., [51]). The masking studies showed that saccadic suppression tended to be divisive, attenuating sensitivity at all mask levels by a constant scaling factor [13]. More recent studies show that during saccades, the temporal impulse response for luminance (but not for colour) becomes faster [9], by an amount that agrees quantitatively with the behaviour of M-cells ([4,35]). As M- but not P-cells show contrast gain, this study provides further support for the notion that the M-system is selectively suppressed during saccades. But it also suggests the intriguing possibility that saccadic suppression is achieved by an extra-retinal signal setting the gain control mechanisms of the thalamus, or early cortex. This would certainly be a parsimonious solution for the visual system. But whatever the mechanism, the evidence presented in this review all suggests that the magnocellular pathway is selectively suppressed during saccades, thereby blunting the otherwise disturbing sense of motion that would accompany every saccade.

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