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Changes in visual perception at the time of saccades

John Ross, M. Concetta Morrone, Michael E. Goldberg and David C. Burr

We frequently reposition our gaze by making rapid ballistic eye movements that are called saccades. Saccades pose problems for the visual system, because they generate rapid, large-field motion on the retina and change the relationship between the object position in external space and the image position on the retina. The brain must ignore the one and compensate for the other. Much progress has been made in recent years in understanding the effects of saccades on visual function and elucidating the mechanisms responsible for them. Evidence suggests that saccades trigger two distinct neural processes: (1) a suppression of visual sensitivity, specific to the magnocellular pathway, that dampens the sensation of motion and (2) a gross perceptual distortion of visual space in anticipation of the repositioning of gaze. Neurophysiological findings from several laboratories are beginning to identify the neural substrates involved in these effects.

Saccades are ballistic movements of the eyes that reposition our gaze, three times a second on average. They can be deliberate, but normally are automatic and not noticed. A person watching a sporting event, conversing with a companion, looking at a television or reading a book usually makes many saccades without noticing that they have occurred. Not only does the actual movement of the eyes escape notice, but also the motion of images as they sweep across the retina and the fact that gaze itself has been repositioned go unnoticed. The world appears to 'stay put'. By contrast, comparable image motion produced externally, as opposed to by movements of an observer's own eyes, has an alarming effect on the observer's sense of stability.

Helmholtz[1] was one of the first to address the problem of why image motion caused by saccades and other eye movements passes unnoticed and why stability is maintained in spite of shifts in image position. He argued that image motion resulting from eye movements is sensed, but not perceived. Image motion is used, in addition to 'the effort of will involved in trying to alter the adjustment of the eyes'[1], to enable constancy of visual direction to be maintained both during and after saccades. Sperry[2] and Von Holst and Mittelstaedt[3] formalized Helmholtz's ideas in the 1950s in two closely related theories. They suggested that saccades were accompanied by a 'corollary discharge'[2] or an 'efference copy'[3] of the motor signal and that this information was used to cancel image motion caused by saccades. This idea was popular, but has become less plausible with the realization that motion might be sensed by specialized mechanisms and cannot simply be annulled by a contrary displacement signal.

An alternative potential source of extra-retinal information about eye position is proprioceptive signals from extra-ocular muscles (Sherrington's 'inflow' theory[4]). However, this theory has serious

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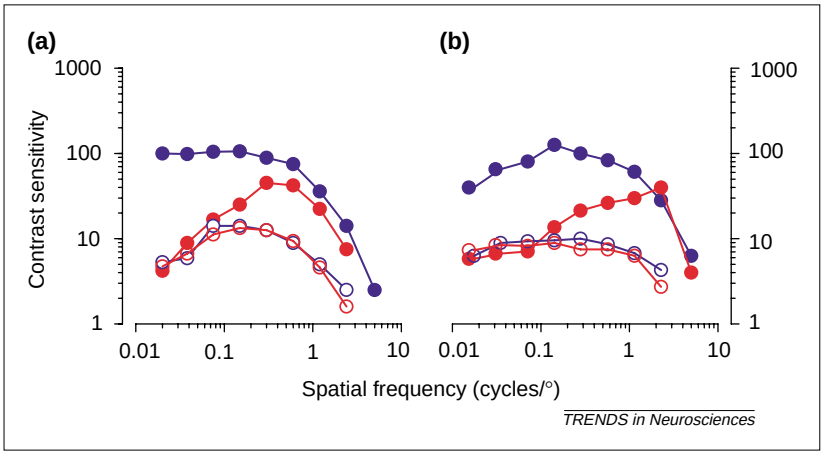

Fig. 1. Selective saccadic suppression of low frequency luminance-modulated stimuli. Contrast sensitivity was measured in two subjects (a) and (b) who were required to detect briefly flashed horizontal gratings with variable spatial frequencies during fixation (blue symbols) and shortly after the onset of a large horizontal saccade (red symbols). Either the luminance (filled circles) or equiluminant colour (open circles) of the gratings could be modulated. For luminance modulation, there was a large difference in sensitivity between normal and saccadic viewing, but only at low spatial frequencies. At frequencies >0.5 cycles/°, sensitivity was virtually identical in the two conditions. For chromatically modulated stimuli, however, there were no differences in sensitivity. Adapted, with permission, from Ref. 24.

difficulty explaining perceptual effects that precede eye movement, given the latencies involved. Nevertheless, recent evidence suggests that proprioceptive feedback might contribute to stability, although the feedback loop has a high threshold and low gain[5,6] and is certainly not sufficient to completely annul the effects of saccadic image motion.

Historical accounts of early work and the debate between inflow and outflow theories are to be found elsewhere (for example, Refs 7,8).

Saccadic suppression

Another idea to emerge early in the last century was that visual sensitivity is actively reduced during saccades. Holt[9] concluded that saccades 'condition a momentary visual central anaesthesia', that is, a complete loss of sensitivity. However, evidence for suppression by saccades is contradictory. Many researchers[10–12] have reported weak threshold elevation for detecting spots of light flashed briefly during saccades (two to threefold) and Krauskopf *et al.*[13] found no threshold elevation. By contrast, Bridgeman *et al.*[14] reported a strong reduction in sensitivity for detecting displacement during saccades when the displacement occurred at about the same time as the start of a saccade. There are many commonplace instances where vision is manifestly clear during saccades. For example, when looking at the track from a fast-moving train, the sleepers become visible only when we saccade against the motion of the train, thereby stabilizing their image on the retina (see also Ref. 15). In the exploratorium in San Francisco, USA, a horizontal saccade across the 'lightstick' (a vertical strip of appropriately flickering lights) produces a clear and detailed sketch of a human eye. Clearly, not all visual functions are suppressed during saccades, so the 'anaesthesia' or loss of sensitivity might be specific to particular stimulus configurations.

Dodge[16] and Woodworth[17] concluded that there was no requirement for a central change in visual functions, arguing that image motion during saccades was too rapid to be seen and caused what Campbell and Wurtz[18] later termed a 'greyout'. Is this really the case? Measurements of contrast sensitivity during normal vision[19] show that when moving at saccadic speeds, gratings with high spatial frequencies become invisible or 'grey out', as had been suggested. However, gratings with low spatial frequencies not only remain visible but also become significantly more conspicuous[19]. Thus, during saccades, high spatial frequencies (i.e. details) of images should not be resolvable, but the normally invisible low spatial frequencies that predominate in natural scenes[20] should become abruptly salient. Therefore, if saccadic suppression occurs at all, it should be strongest at low spatial frequencies.

This is exactly what has been observed in studies of contrast sensitivity for gratings that are flashed briefly during saccades[21–24]. The filled symbols in Fig. 1 are an example of how contrast sensitivity for gratings that are flashed briefly at the beginning of a saccade or in normal viewing vary as a function of spatial frequency. The curves are virtually identical at high spatial frequencies, but diverge strongly at low spatial frequencies, revealing a tenfold reduction of sensitivity at 0.02 cycles/°. The reduction in sensitivity is specific at frequencies that would otherwise be visible during saccades. This selectivity might also explain some of the conflicting data from earlier studies. Loss of sensitivity should depend on the spatial frequency content of the experimental stimuli, which are typically high (e.g. small spots of light) in the luminance threshold-based studies[10–12], but low (large targets) in displacement studies[14].

Recent anatomical and physiological advances have shown that vision, at least in the early stages of visual analysis, is processed through two largely independent streams: the magno- and parvocellular systems (for example, Ref. 25). Although these two systems are not completely separate, parvocellular function can be favoured by using equiluminant stimuli, that is, stimuli that are modulated in colour but not in luminance. The open symbols in Fig. 1 show how sensitivity to briefly presented equiluminant stimuli is affected by saccades[24]. There is little suppression of equiluminant stimuli, irrespective of the spatial frequency and in some circumstances stimuli can even be enhanced. These results imply that saccadic suppression is specific to the magnocellular pathway with the parvocellular pathway being unimpaired. Other experiments have shown that displacements during saccades show little suppression if the stimuli are equiluminant[26].

Using a different technique Uchikawa and Sato[27] have provided convincing support for this conclusion. They measured incremental spectral sensitivity for coloured disks against a white background during

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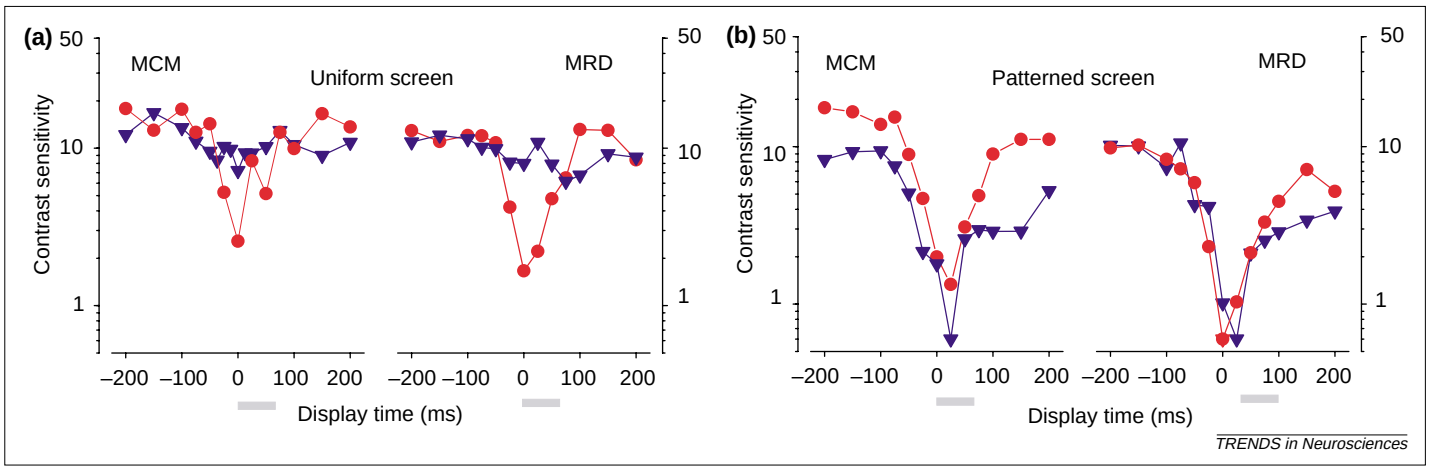

Fig. 2. Contrast sensitivity for detecting the presence of a broad 'difference-of-gaussian' horizontal bar. The bar was flashed briefly at two subjects (MCM, MRD) at various times relative to the onset of a real horizontal saccade (red symbols) or one simulated by a rapidly rotating mirror (blue symbols). During real saccades, there was always a large and temporally localized loss of sensitivity of about one order of magnitude at the onset of a saccade. When the background was homogeneous, simulated saccades (mirror motion) caused little suppression (a), whereas a strongly patterned background caused suppression that was comparable in strength and lasted longer than that caused by a real saccade (b). Shaded bars represent the duration of the eye movement. Thus, image motion might be important for saccadic suppression but it is not sufficient to explain either the amplitude or the timecourse of the suppression that occurs during real saccades. Adapted, with permission, from Ref. 31.

normal viewing and saccades. During saccades, the spectral sensitivity curve showed a marked decrease at ~570 nm (known as Sloan's notch), a clear signature of the presence of spectrally opposed mechanisms, which are typical of the parvocellular system. In normal viewing, this decrease was absent (for brief stimuli), suggestive of magnocellular function.

A fundamental question provoked by these studies is whether saccadic suppression results from a central signal, such as a corollary discharge[2,3], or whether the visual motion caused by the eye movement itself masks vision during saccades[28,29]. There is good evidence that image motion of the kind caused by saccades can mask brief stimuli[18,29,30], but is this the only, or indeed the principal, mechanism at work?

One way in which visual and non-visual effects can be distinguished is to measure sensitivity during 'simulated' saccades in otherwise identical conditions. Figure 2 shows contrast sensitivity for detecting grating stimuli on a display viewed through a mirror that stimulates saccades by abruptly deflecting the display at a speed, amplitude and acceleration comparable to 'real' saccades[31]. The image motion altered thresholds, but the pattern of the results was different from those observed during real saccades. For sinusoidal gratings displayed on an otherwise blank screen, simulated saccades had little effect on thresholds, whereas real saccades produced greater effects (Fig. 2a). However, when a high-contrast random pattern was added to the display to provide strong visual references (Fig. 2b), the simulated saccade produced a suppression that was comparable in magnitude and lasted longer than that produced by the real saccade. For both the real and simulated saccades, suppression preceded and outlasted the saccade, as previously reported[32], but recovery to the simulated saccade with patterned background was much slower than recovery to the real saccade.

Visual masking, therefore, might be important for saccadic suppression, but clearly it is not the only mechanism involved. There must also be a signal of non-visual origin that accompanies each real saccade that decreases sensitivity to low frequency, luminance-modulated stimuli. The similarity between the timecourse of saccadic suppression and visual masking could indicate that these two phenomena have a common site of action. One possibility is that both act on mechanisms that regulate the gain of cortical or geniculate cells. Decreasing the gain of these cells should decrease sensitivity, as previously observed[23,24,31,32] but it should also cause these cells to fire more transiently. Indeed, measurements show that the impulse response function increases during saccades[33], consistent with this prediction. Other evidence suggests that saccadic suppression precedes the site of contrast masking[24] and motion analysis[34], again indicating an early action.

The selectivity of the suppression of the magnocellular pathway suggests that suppression might be specific to motion signals, an idea first proposed by Burr *et al.*[23]. This suggestion is supported not only by the selectivity of suppression for low spatial frequency luminance-modulated gratings, but also by the dependence of suppression on luminance. At low luminance levels the range of frequencies over which sensitivity is dominated by the transient motion channel shifts to lower frequencies. Low luminance has the same effect on saccadic suppression, lowering the spatial frequency range at which it occurs, suggesting that only the transient system is suppressed, irrespective of the luminance level. More-direct measurements, such as thresholds for detecting a change in speed, also point to a strong suppression of motion mechanisms[23] and several other laboratories[35,36], using different techniques, have confirmed that motion sensitivity is greatly reduced during saccades. Although it is difficult to measure motion during the short duration of saccades (a motion stimulus requires integration over time),

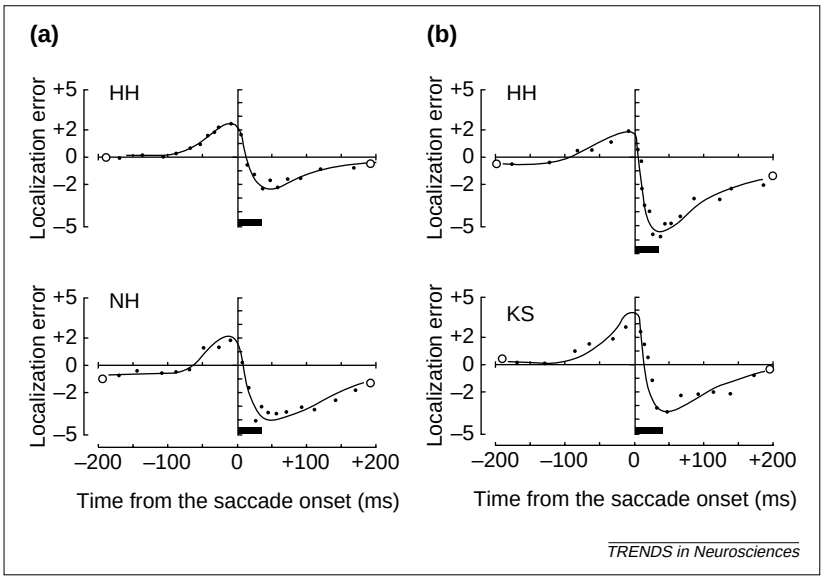

Fig. 3. Examples of perceptual mislocalization around the time of saccades. Subjects (HH, NH, KS) made 4° saccades, either downwards (a) or towards the right (b) and indicated the apparent position of a small, briefly flashed light in otherwise dark conditions. Positive errors refer to mislocalization in the direction of the saccade. Shaded bars represent the duration of the eye movement. Adapted, with permission, from Ref. 74.

these studies all show that motion-related tasks are severely impaired during saccades.

Castet and Masson[37] recently challenged the hypothesis that suppression of motion occurs during saccades. They showed that under certain conditions, saccades in the direction of a rapidly moving, high-contrast grating can improve not only the detection of the grating, but also the discrimination of the direction of its motion. However, this is not surprising because the suppression of visual function during saccades occurs over a brief period and is never total. Thus, under normal conditions, when stimuli are not presented transiently at an optimal moment, visual function (including motion perception) might continue during saccades. This is illustrated by the previously cited example of saccading against the motion of a train[15]. Indeed, the data presented by Castet and Masson[37] can be modelled by assuming a transient saccadic desensitization combined with a reduction in the physical speed of the retinal image[38]. However, these studies highlight the important point that centrally driven saccadic suppression attenuates visual motion but does not eliminate it, suggesting that other mechanisms, such as masking, might come into play under natural conditions.

Some qualitative changes that have been observed during saccades might also be relevant for understanding motion perception during saccades. In one study[23], observers viewed a large screen from a short distance on which a scene was back-projected by a mirror system. When the scene was displaced abruptly at saccadic speeds and amplitudes (by mirror deflection), observers reported a strong sensation of motion that instantly commanded attention, reporting the sensation of being 'startled'. However, if the displacements of the scene were the result of a saccade, the motion was sensed (if the contrasts were sufficiently high) but lacked the saliency of fast motion in normal viewing (subjects observed that the image had been displaced, but did not report feeling 'startled'). This could simply be the result of imperfect attenuation of motion mechanisms during saccades, but it might also implicate other factors. One such factor could be that the visual system 'expects' a rapid displacement during saccades and is therefore not 'surprised' when this occurs. This might be achieved by reducing attention, particularly to motion, which has recently been shown to be under strong attentional control[39,40]. Interestingly, parietal cortical neurones that are involved in both attention and saccadic selection, respond weakly to motion induced by saccades or if a saccade brings a stable stimulus to their receptive field. However, if a monkey attends to the stimulus for another reason – either because it is relevant to a task or because it begins abruptly and immediately before the saccades – the cells fire, as if the modulating factor was attention, instead of motion in the absence of attention[41].

The studies discussed above all refer to moderate-to-large saccades and might not be applicable to the microsaccades (saccades ≤2.0°) that are normally made, together with slow drifts, around the fixation point. Most evidence suggests that small saccades cause little or no threshold elevation[13,42], indicating that the effects of the image tremor must be controlled by other means. Murakami and Cavanagh[43] recently proposed that the retinal motion generated by microsaccades is eliminated by subtracting a baseline speed, estimated from the minimal retinal 'jitter', from the velocity signals of local-motion detectors. Evidence favouring this model is derived from the observation that if a region of the retina is adapted to jittering motion and a static pattern is subsequently inspected, the unadapted (but not the adapted) region appears to jitter. These authors claim that the reduction in motion sensitivity caused by adaptation reduces the estimate of the baseline jitter and that with the baseline thus lowered, the motion caused by eye-jitter becomes super-threshold in the unadapted region and hence visible. This idea is particularly interesting in the context of older theories that suggested stabilization is achieved by subtraction of extra-retinal signals. In such cases, there is subtraction of a speed scalar (not a spatial displacement vector) from velocity estimates that have been extracted by specialized motion detectors.

Physiological studies

Whereas psychophysical studies indicate an early site for the action of saccadic suppression, perhaps as early as LGN, direct physiological evidence is less clear. In humans, visual activity during saccades has been studied using evoked potentials (VEPs) and, more recently, with fMRI and positron emission tomography (PET) imaging. Early studies using VEP (Refs 44,45) showed a strong (>80%) attenuation of response amplitude to stimuli that were presented at about the same time as the start of a saccade. More recent studies (see, for example, Refs 46,47), using multiple arrays of electrodes, have confirmed this. It is difficult to draw conclusions about the site or

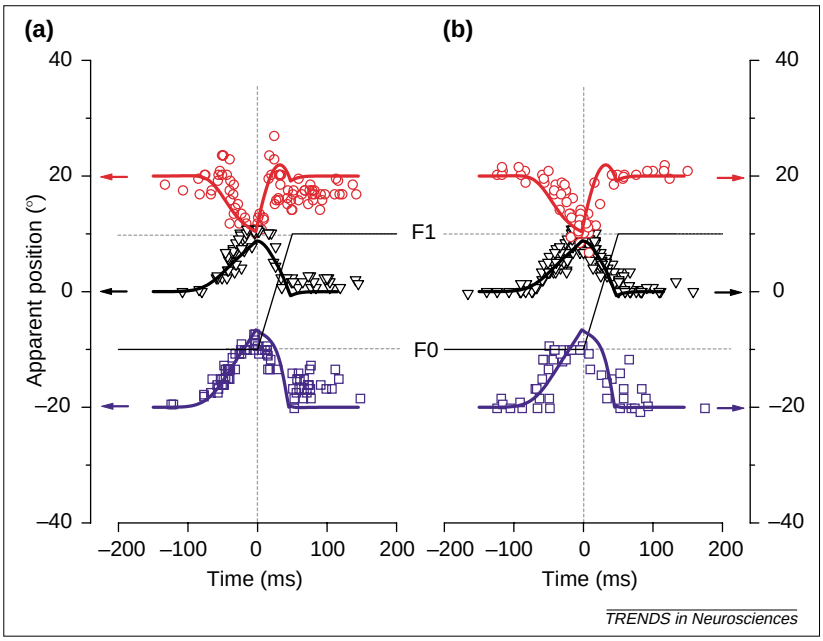

Fig. 4. Mislocalization of the position of a bar. A bar was briefly presented around the time of a horizontal saccade from F_0 to F_1 (–10° to 10°). The ordinate shows the external position judged by the subjects (a) and (b) at the various display times relative to the onset of a saccade (where 0° refers to the centre of the screen). The bar was actually displayed in one of three positions on the screen (0° and ±20°, indicated by the arrows). Subjects systematically mislocated the bar, depending both on the time of presentation and its actual position. For bars presented at –20° or 0°, there was a strong mislocation in the direction of the saccade at about the same time as the onset of a saccade. This suggests that the perceptual system anticipates the saccade and starts to compensate for its effects (see also Fig. 5). For bars presented at +20° (past the position of the saccadic target), however, the mislocation was in the opposite direction (that is, against the direction of the saccade), leading to an effective compression of visual space. Adapted, with permission, from Ref. 80.

selectivity of the suppression from these results, however they all point to neural changes in the visual response at about the same time as the onset of a saccade that is mediated by an extra-retinal signal.

fMRI studies also show a strong modulation of activity in V1 when saccades are made in complete darkness[48,49]. Interestingly, earlier PET studies[50] also show a clear diminution of cerebral blood flow (CBF) activity in V1, V2 and parietal cortex when subjects make saccades in the dark, with the diminution being directly proportional to the frequency of the saccades. The inhibition of activity in these cortical regions strongly supports the psychophysical evidence for suppression of the magnocellular pathway[24].

However, direct recording from V1 in monkeys has yielded conflicting results. Wurtz[51] and Fischer *et al.*[52] reported that striate cortical cells responded identically to motion produced by saccades and stimulus motion when the eyes were not moving, suggesting that suppression is the result of a visual, as opposed to an oculomotor, corollary process. By contrast, Battaglini *et al.*[53] found a small number of cells (~12%) in V1 that respond to external motion, but not to saccade-induced motion. A recent evoked potential-based study in alert cats also provided evidence for a corollary discharge modulation of activity in V1 (Ref. 54).

Similar controversial results have been obtained in V2 and V4 (reviewed in Refs 55,56). By contrast, recent studies, using brief stimuli, showed a much stronger inhibitory effect of saccades in middle temporal area (MT) and medial superior temporal area MST neurones[57]. One study reported little suppression, but found a reversal in the directional selectivity of MST neurones[58]. It is possible that this change in selectivity could be the mechanism that blunts the sensation of motion.

The influence of saccades on lateral geniculate nucleus (LGN) activity has been studied in alert cats. The results show that saccades have both inhibitory and excitatory effects on LGN responses, which differ between X- and Y-cells (Refs 59,60) (these cells are not necessarily homologues of parvo- and magnocellular pathways in primates). Many of the suppressive effects observed can be reproduced either by visual stimulation at saccadic speeds outside the receptive field[61] or by playing back the effective saccadic stimulus[60]. These findings suggest that visual signals play a role in suppressing visual activity during saccades. This is consistent with the psychophysical results shown in Fig. 2, illustrating that when strong visual contrasts are present, visual masking effects might be equivalent to or greater than central effects. However, this does not preclude the existence of central effects during saccades, which become evident when visual effects are minimized by minimizing contrast. The action of an extra-retinal signal in cat X-cells has also been directly demonstrated by inactivating the pre-tectum showing that this normally exerts a strong excitatory signal on X-cells during real but not simulated saccades[62]. Extra-retinally mediated saccadic suppression has also been shown in the pulvinar of both the monkey, where many cells are suppressed during saccades in the dark[63], and the cat, where 50% of cells that normally respond during simulated saccades did not respond to stimulation during real saccades[64].

Neural activity has also been measured in several brain regions during microsaccades[65–67]. Martinez-Conde *et al.*[67] showed that when a monkey explores a stable environment, V1 neurones are probably active when a microsaccade moves the stimulus in the receptive field. These authors postulate that loss of this microsaccade-related activity is responsible for the fading of visual perception during image stabilization. By contrast, Leopold and Logothetis[65] found a microsaccade-related depression of activity in V1, excitation in V2 and V4 and no consistent effect in inferior temporal cortex. In addition, Bair and O'Keefe[66] found no difference between real and microsaccade motion in MT neurones.

In conclusion, although human psychophysics and physiology data clearly suggest an extra-retinal suppression of early visual activity during saccades, the underlying neural mechanisms are yet to be identified using physiological studies.

Perceived position

Retinal motion is not the only problem introduced by saccades. A related (but not identical) problem is how

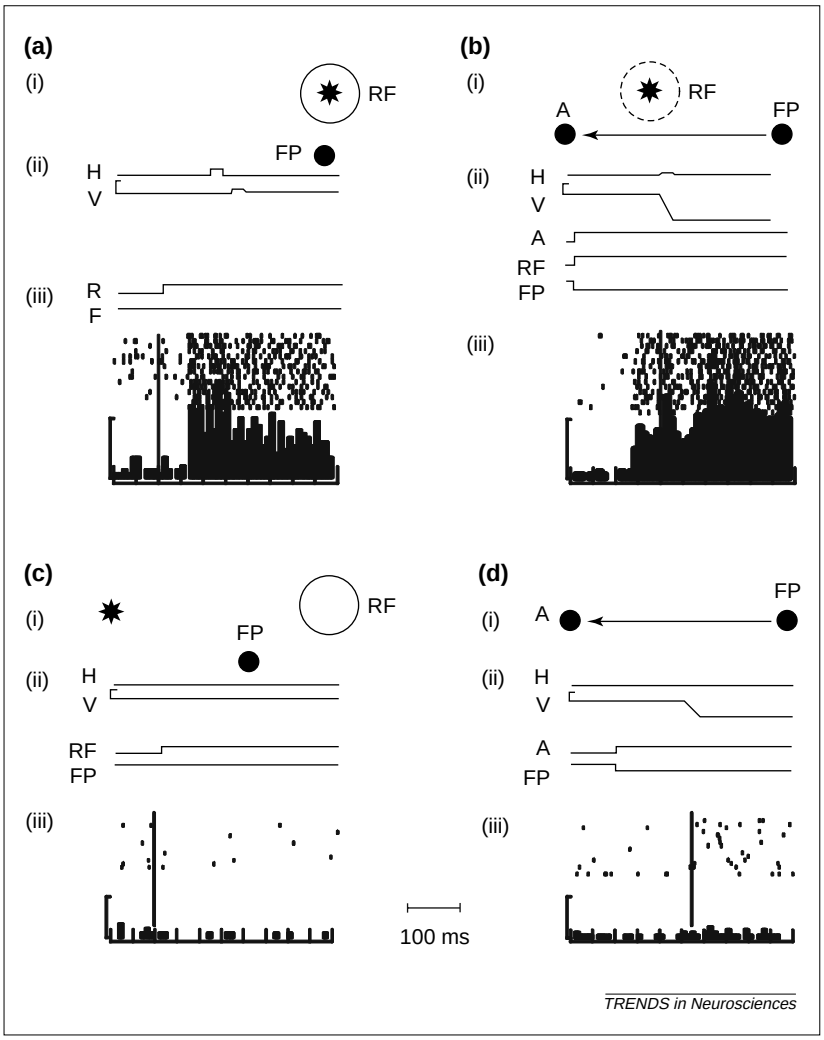

Fig. 5. Saccadic remapping of the visual receptive field by a lateral intraparietal area (LIP) neurone. (a)–(d) part (i) shows the position of the receptive field (RF) and fixation point (FP). (a)–(d) part (ii) shows the horizontal (H) and vertical (V) eye position traces and RF stimulus and FP artifacts. (a)–(d) part (iii) shows the neuronal response displayed as a raster plot. Each point signifies an action potential and each line illustrates a separate trial. Successive lines are synchronized with the appearance of the stimulus (vertical line). The histogram beneath the raster illustrates the cumulative sum of the trials in the raster plot. (a) Shows the response of the neurone to a behaviourally irrelevant stimulus in its receptive field (RF) during a fixation task. (b) When the monkey makes a saccade that brings a stimulus into its RF, the neurone fires before the saccade begins. At the time that the neurone begins to fire, the stimulus is not in the RF of the neurone as determined in the fixation task. In part (i) A represents the saccade target and the arrow signifies the saccade. (iii) The raster and histogram synchronized with the beginning of the saccade. (c) The neurone does not discharge when the stimulus appears at the same spatial location (the future RF). (iii) The histogram and raster are synchronized with the start of the stimulus. (d) The neurone does not discharge when the monkey makes the saccade, but no stimulus appears in the future RF. (iii) The raster and histogram synchronized with the start of saccade appearance. Adapted, with permission, from Ref. 95.

to perceive a stable external world from extremely unstable retinal images. Helmholtz[1] believed that the constancy of perceived position was maintained during and after saccades, because both extra-retinal (the 'effort of will') and retinal (sensed but not perceived image motion) information were used to recalibrate the direction of gaze.

Leonard Matin and colleagues[68–71] and Bischoff and Kramer[72] were among the first to find errors in localization at the time of eye movements. These and subsequent studies[73–78] revealed that the perceived position of a target that was flashed before, during and after a saccade was not its veridical position was systematically mislocalized. The apparent position of flashed targets changes over a 200 ms period, starting less than 100 ms before the eyes begin to move and reaching a maximal effect at about the same time as the onset of saccades. Presumably these displacements compensate for the actual shift in retinal position brought about by the saccade. Figure 3 shows a clear example of the timecourse of errors in localizing a spot that is briefly flashed at about the same time as the saccades occur[74]. Before the saccade, the spot seems to be displaced in the direction of the saccade, suggesting an anticipatory shift. After the eyes start to move, there is a rebound effect, suggesting that the shift is slower than the saccade itself. The maximum shift is approximately half the size of the saccade, suggesting a relative gain of 0.5, as observed by others[79].

If the errors in localization are to compensate for eye movements, they should always be in the same direction as the saccade. However, this is not always the case. Figure 4 shows reports of perceived positions of visual targets presented in one of three spatial positions, before, during and after saccades[80]. Each of the three targets tends to be mislocalized at about the time of a saccade, an effect that is greatest at the onset of the saccade. However, the size and sign of such errors strongly depend on the position of the target within the visual field. Targets are not simply displaced in the direction of the saccade, but tend to converge towards the saccadic target, which results in 'compression' of the visual world. This compression is powerful enough to remove vernier offsets for line targets that are flashed at about the time of the saccade onset and can create offsets for collinear line targets flashed at different times (75 ms apart). For example, compression causes four bars, flashed in such a way as to straddle the saccadic target, to merge into a single bar and severely distorts natural scenes. Because the relative distance between objects does not depend on retinal coordinates in these conditions, this emphasizes how large the perceptual compression can be.

However, not all studies have reported compression (for example, Ref. 78). In an attempt to reconcile the divergent results from various laboratories, Lappe and co-workers[81] studied saccade-induced mislocation errors under various conditions. They found strong compression towards the saccadic target, replicating previous findings[80,82], but only when visual references were available. In complete darkness, all mislocation errors were in the direction of the saccade and similar in magnitude (in agreement with Ref. 78). Furthermore, for compression to occur, the visual reference had to be available after the saccade. Other studies have shown that visual references also modify the gain of pre-saccadic displacement[83].

Similar to saccadic suppression, a question of interest is whether shifts in position and spatial

compression have a central or a retinal origin. Two lines of evidence point to a central origin. Simulated saccades (produced by the mirror technique described earlier) cause a different pattern of mislocalization, with a different timecourse and magnitude[82,84]. More significantly, no compression occurs with simulated saccades, because all targets are displaced in the same direction and to the same extent, irrespective of their position in the visual field[82]. Other evidence was provided by a study by Bahcall and Kowler[85], who used an adaptation technique[86] to alter the gain of saccades (so that they were systematically shorter or longer than intended). They showed that the location errors are determined by the intended as opposed to the actual saccadic amplitude, suggesting that the compensatory shift for the saccade is not determined by the visual motion *per se*, but instead has a central origin.

Whereas almost all researchers agree that saccades cause strong mislocalizations in perceived position (either compression or uniform displacement), several reports suggest that errors do not occur in motor responses such as hammering[87] and eye pointing[88]. These results are intriguing because they imply that two visual representations exist: one that remains veridical during saccades and another that becomes transiently distorted. This is consistent with the longstanding[89] and recently revived[90] idea that separate systems are responsible for conscious perception and for direct interactions with our environment. However, it should be pointed out that more recent studies, using double-saccade or pointing paradigms, show displacement errors for motor responses in the direction of saccades[77,91,92], casting doubt on the conclusions of the earlier studies. We recently repeated the experiment shown in Fig. 4, asking subjects either to point to or report verbally the position of targets presented just before a saccadic eye movement[93]. Verbal reports produced the same results to those shown in Fig. 2, even when all visual references were removed by obscuring the screen at the time of the report. However, when asked to point to where a bar had been seen (again with references obscured), the pattern of pointing was virtually veridical. This supports the idea that there are two separate visual representations with separate remapping during saccades.

Other lines of evidence also suggest that veridical information might be available at the time of saccades and can be used under certain conditions. For example, Deubel *et al.*[94] repeated Bridgeman *et al.*'s[14] measurements of sensitivity to line displacements at the time of saccades. The saccade typically raises thresholds by more than a factor of three. However, if a line is briefly blanked at the time of the saccade and reappears ~100 ms later, subjects can detect displacements with unimpaired accuracy. This suggests that information about position is not lost during the saccade and that mechanisms exist to maintain continuity between fixations.

It is interesting to note that the timecourse of perceptual compression[82] is similar to that for loss of sensitivity[31] (compare Figs 2–4). Specifically, both begin more than 50 ms before saccades start, are maximal at or shortly before the start of saccades, diminish during saccades and disappear only after saccades have ended. The timecourse of loss of sensitivity during saccades has been successfully modelled by assuming the existence of a signal spike that coincides with the start of a saccade and sets a high contrast gain. When added to a stimulus and convolved with an impulse response function, this extra-retinal or corollary discharge spike can account for the magnitude and the timecourse of sensitivity loss, including those stimuli that precede saccades. The same impulse signal, temporally shaped by the filter properties of the visual neurone, might dictate the timecourse of saccadic compression. However, the presence of frames of reference and the visual activity that they stimulate might be important in synchronizing the suppression and compression with the time of the saccade.

Physiological mechanisms

Compression cannot be explained by a slow extra-retinal position signal, but instead it requires changes in the properties of receptive fields or position codes associated with them. Duhamel *et al.*[95] showed that there are early changes in the receptive field properties of some neurones in the lateral intraparietal area (LIP) of monkeys making saccades that anticipate their consequences (Fig. 5). LIP neurones begin to respond up to 80 ms before the onset of a saccade to stimuli that will fall within their classical receptive field after the saccade is completed. This predictive effect might represent a mechanism by which the visual and oculomotor systems combine to calculate a spatially accurate image of the world, in spite of the eye moving. This could enable neurones to respond immediately at the end of a saccade to stable stimuli that enter the receptive field by virtue of the saccade, without having to 'wait' for retinal reafference[41].

During the perisaccadic interval some, although not all, LIP neurones show the predictive shift of receptive field in the direction of the saccade. Other neurones continue to respond to the presaccadic position and some respond to stimuli in both positions[57,96]. Areas that receive input from the LIP (including the frontal eye field and the superior colliculus) might thus interpret stimuli arising over a large area (comprising pre- and post-saccadic receptive fields) as being in the same position. This could result in compression, particularly of the positions of stimuli that are flashed briefly during psychophysical experiments. Perisaccadic receptive field shifts are not unique to the LIP, but have been found in other eye movement-related areas (e.g. the superior colliculus[97] and the frontal eye field[98]), a medial parietal area that is

associated with reaching[99] and even in earlier stages in the cortical visual system, V4, V3a and V2 (Ref. 100). The multiple representations in all these areas could contribute towards the observed compression.

Although data suggest a functional relationship between receptive field 'stretching' and transient mislocalization, there is no direct evidence available to link these two phenomena. Clinical cases, however, suggest that the mechanism by which the brain calculates spatial location across a saccade is by compensating for each eye movement using a shifting receptive field strategy. Patients with right parietal lesions cannot compensate for leftward saccades in a double-step task. Furthermore, in the context of that task, such patients cannot accurately localize stimuli that appear in their right (ipsilateral) visual field by using a rightward (ipsilateral) saccade, although their performance of rightward saccades is generally more accurate than leftward saccades[101].

Concluding remarks

Saccades result in several physiological events in anticipation of their threat to visual stability. They suppress visual motion, an effect that is not entirely the result of 'visual masking' (although the motion of patterned stimuli might play an important role). Suppression is selective and particularly strong for rapid, low frequency luminance modulation, suggesting that only magnocellular function is suppressed, whereas parvocellular function is spared or even slightly enhanced. Suppression starts before and is maximal at the start of a saccade and outlasts saccades. Saccades also alter the apparent position of targets appearing at about this time, causing both displacement and compression. The timecourse of the displacement and compression closely parallels that of suppression.

What emerges from the psychophysics and neurophysiology is the idea that an extra-retinal signal, which can be termed corollary discharge[2] or efference copy[3], coordinates two main functional changes that preserve visual stability during saccades. The first is a change of gain that selectively reduces sensitivity, making image motion less visible. The second is a transitory expansion of receptive fields that enables a smooth shift of coordinates at the price of transitory spatial compression that affects only targets that flash into view at the time of the onset of a saccade.

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