

93 Visual Perception during Saccades

DAVID C. BURR AND M. CONCETTA MORRONE

WE FREQUENTLY REPOSITION our gaze by making rapid ballistic eye movements called *saccades* in order to focus the fovea on visual areas under scrutiny. While the strategy is highly efficient for the visual system, allowing it to analyze the whole visual field with the high resolution of the fovea, it poses several problems for perception. Saccades cause rapid large-field motion on the retina, potentially confusable with large-field motion in the external world. They also change the relationship between external space and retina position, confounding information about visual direction. Much effort has been made in recent years to attempt to understand the effects of saccades on visual function. Evidence suggests that saccades trigger two distinct neural processes: a suppression of visual sensitivity, specific to motion analysis, probably mediated by the magnocellular pathway, and a gross perceptual distortion of visual space just before the repositioning of gaze. While the evidence for these phenomena is strong, their functional role for perception is less clear, leaving several areas of inquiry still open.

Saccades are ballistic movements of the eyes made to reposition our gaze. They can be deliberate, but normally they are automatic and go unnoticed. Not only do the actual eye movements escape notice, but so does the image motion they cause and the fact that gaze itself has been repositioned. The first to document this problem was probably the Persian scholar Alhazen: “For if the eye moves in front of visible objects while they are being contemplated, the form of every one of the objects facing the eye . . . will move on the eye as the latter moves. But sight has become accustomed to the motion of the objects’ forms on its surface when the objects are stationary, and therefore does not judge the object to be in motion” (Alhazen, 1083, p. 194; see Howard, 1996). Much later, Helmholtz (1866) argued that image motion caused by eye movements is *sensed* but not perceived. He further claimed that, together with “the effort of will involved in trying to alter the adjustment of the eyes,” eye movement-induced retinal motion is used to maintain constancy of visual direction during and after saccades. Sperry (1950) and Von Holst and Mittelstädt (1954) expanded on Helmholtz’s ideas in the 1950s with two closely related theories. They suggested that saccades were accompanied by a *corollary discharge* (Sperry, 1950) or an *effference copy* (Von Holst and Mittelstädt, 1954) of the motor signal and that this information was used to cancel image motion caused by saccades. This idea was popular, but it has become less

plausible with the realization that motion is probably analyzed by specialized mechanisms distinct from those signaling spatial position, and can therefore not simply be annulled by a contrary displacement signal (Burr and Ross, 1986).

An alternative potential source of extraretinal information about the eye position is proprioceptive signals from extraocular muscles: Sherrington’s (1918) *inflow* theory. However, this theory has serious 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 (Bridgeman and Stark, 1991; Gauthier et al., 1988) and is certainly not sufficient to annul completely the effects of saccadic image motion. Historical accounts of early work and the debate between inflow and outflow theories are to be found elsewhere (e.g., Bridgeman et al., 1994; Carpenter, 1977).

Saccadic suppression

Another idea to emerge early in the twentieth century was that visual sensitivity is actively reduced during saccades. Holt (1903) concluded that saccades “condition a momentary visual central anaesthesia,” a loss of sensitivity. However, evidence for suppression by saccades is contradictory. Many researchers (Krauskopf et al., 1966; Latour, 1962; Zuber and Stark, 1966) have reported weak threshold elevation for detecting spots of light flashed briefly during saccades (two- to threefold), and Krauskopf et al. (1966) found no threshold elevation at all. By contrast, Bridgeman et al. (1975) 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.

Dodge (1900) and Woodworth (1906) 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 (1978) later termed a *grayout*. However, measurements of contrast sensitivity (during normal vision) show that this idea cannot be generally true. Although stimuli of high spatial frequency become unresolvable at saccadic speeds, stimuli of low spatial frequency can be seen only in motion but become considerably more visible at saccadic speeds (Burr and Ross,

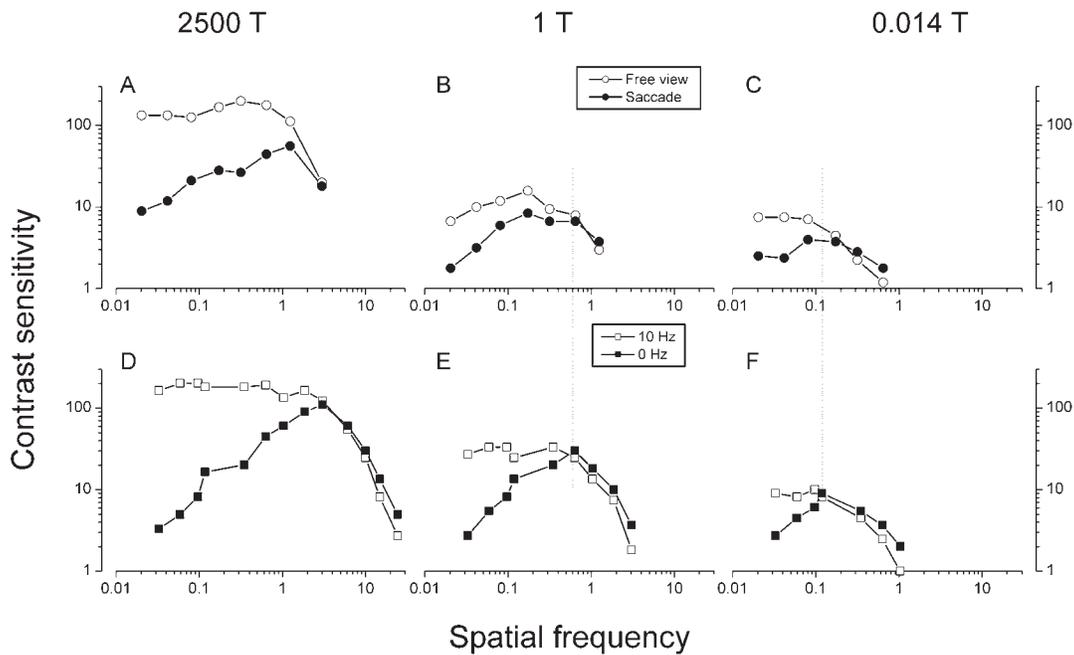


FIGURE 93.1. *A–C*, Contrast sensitivity for detecting a horizontal grating briefly displayed either at the beginning of a large (30 degrees) horizontal saccade (*filled circles*) or during free viewing (*open circles*). Measurements were made for 2500 trolands, 1 troland, and 0.014 troland. Sensitivity is greater during normal than saccadic viewing at low spatial frequencies, but the two curves converge at high frequencies; the point of convergence decreases steadily with retinal illuminance. Note that at the higher spatial frequencies of the lowest illuminance (*C*), contrast sensitivity was actually higher during saccades than during normal vision (see also Fig. 93.2). Measurements were not made above 3 *c/deg*, as the large

saccade would cause smearing of the grating. *D–F*, Contrast sensitivity for continuously displaced horizontal gratings, either stationary (*filled squares*) or drifting at 10 Hz (*open squares*). There is an advantage for drifting gratings at low spatial frequencies. The spatial frequency at which the motion curve peels away from the stationary curve changes with illuminance and corresponds very closely to the point at which the sensitivity during saccades (*above*) diverge from normal sensitivity, strongly suggesting that motion mechanisms are selectively suppressed during saccades. (Data reproduced with permission from Burr et al., 1982.)

1982). Thus, during saccades, the normally invisible low spatial frequencies that predominate in natural scenes (Field, 1987) should become abruptly salient, posing a potential problem for vision.

It seems that at least part of the reason motion is not seen during saccades is that the low spatial frequencies that would normally be so conspicuous are suppressed during saccades (Burr et al., 1982, 1994; Volkman et al., 1978). An example of this frequency-dependent suppression is shown in Figure 93.1*A–C*, which compares contrast sensitivity for detecting briefly flashed horizontal gratings during saccades (*filled symbols*) with that for fixation (*open symbols*). Sensitivity is very similar at the higher spatial frequencies, but at low spatial frequencies sensitivity during saccades is reduced sharply, reaching a tenfold reduction of sensitivity at 0.02 *c/deg*; these are the very frequencies that would otherwise be visible and highly conspicuous during saccades. The selectivity for spatial frequency might explain some of the conflicting data from earlier studies. Loss of sensitivity should depend on the spatial frequency content of the experimental stimuli, typically high (e.g., small spots of light) in the luminance threshold studies (Krauskopf et al., 1966;

Latour, 1962; Zuber and Stark, 1966) but low (large targets) in displacement studies (Bridgeman et al., 1975).

Burr et al. (1982) also reported qualitative changes in motion perception during saccades. Observers viewed at close distance a high-contrast scene back-projected through a deflectable mirror. Displacing the scene abruptly at saccadic speeds and amplitudes caused a strong sensation of motion that instantly commanded attention. However, if the displacements of the scene were the result of a saccade, the motion was sensed, but lacked the salience and the alarming sensation that usually accompany fast motion in normal viewing: subjects observed that the image had been displaced, but they did not report feeling startled. This qualitative impression, together with Bridgeman et al. (1975) demonstrations of large desensitization to displacement during saccades, suggested that the frequency selectivity of the suppression may reflect desensitization of motion mechanisms.

A good deal of evidence points to motion desensitization during saccades. However, it should be pointed out that it is difficult to test motion directly during saccades, for several important technical reasons. One is that, by definition,

motion requires moderately long stimulus durations, necessarily exceeding the duration of maximal saccadic suppression. Another is that the movement of the eyes will introduce image motion, which is difficult to take into account accurately in calculating the real retinal velocity of external motion. However, it is possible to infer the action of motion mechanisms using brief stimuli that contain a wide range of temporal frequencies and hence will excite motion mechanisms (tuned to all directions) as well as mechanisms that respond best to stationary stimuli.

An attempt to do this is shown in Figure 93.1. As mentioned earlier, sensitivity during saccades becomes progressively compromised at low spatial frequencies, while at higher spatial frequencies, above $3c/\text{deg}$, there is virtually no suppression at all. Figure 93.1*D-F* shows steady-state sensitivity measurements for stationary and drifting (10 Hz) gratings measured under comparable conditions. As had been well documented previously (Burr and Ross, 1982), gratings in motion are more conspicuous at low spatial frequencies. Importantly, the spatial frequency at which sensitivity for drifting gratings begins to exceed that of stationary gratings is very similar to the spatial frequency at which saccadic suppression begins (illustrated by the vertical dashed lines). As brief stimuli comprise a wide range of temporal frequencies, the similarity in frequency ranges suggests that it is the motion mechanism that is selectively impaired during saccades. To be certain that the correspondence in spatial frequency was not merely a coincidence, the measurements were repeated at two lower levels of illumination. At all three levels of illuminance, both sets of curves—sen-

sitivity to brief displays in normal and saccadic viewing and sensitivity to continuously drifting or stationary stimuli—peeled apart at about the same spatial frequency, consistent with the suggestion that the mechanisms suppressed during saccades are those responsible for the higher sensitivity to motion in normal viewing.

Despite the problems in examining motion perception directly during saccades, there have been several attempts to do so. For example, thresholds for detecting an abrupt change in the speed of a drifting grating are far poorer during saccades than during normal vision (Burr et al., 1982). The discrimination of motion of random-dot patterns is also severely impaired during saccades (Ilg and Hoffmann, 1993; Shiori and Cavanagh, 1989). And discrimination of two-frame motion sequences is severely impaired when one frame is presented near saccadic onset (Burr et al., 1999).

What may be the mechanism whereby motion sensitivity is reduced? 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 (see Chapter 30). Although these two systems are not completely separate, parvocellular function can be probed by using equiluminant stimuli, which are modulated in color but not in luminance. The magno system there may respond spuriously to some equiluminant stimuli, but it is known to be incapable of color discriminations (Merigan, 1989). Figure 93.2 shows forced-choice discriminations of either the color of equiluminant red-green stimuli or the luminance of equichromatic yellow-

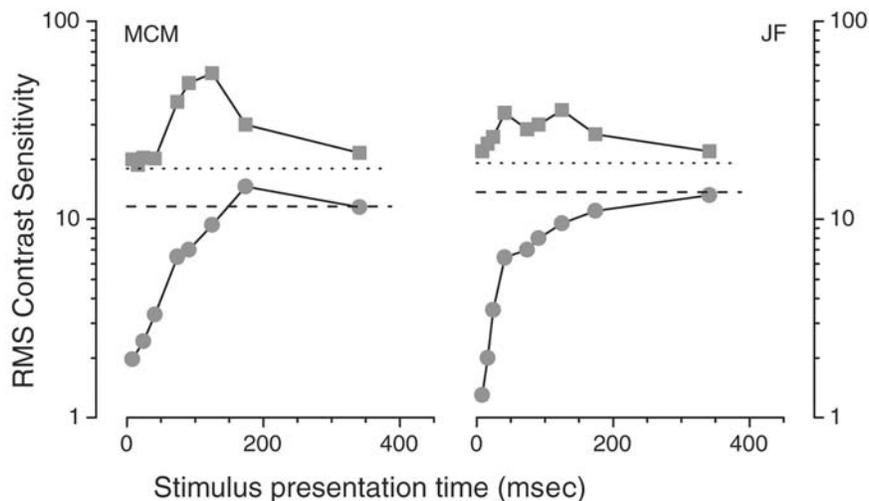


FIGURE 93.2. Contrast sensitivity for discriminating the color or the luminance of a broad horizontal bar briefly presented at a given time after the onset of a saccade (abscissa) for two subjects. Bars were modulated either in color (equiluminant red-green: *square symbols*) or in luminance (yellow-black: *circle symbols*). Sensitivity is expressed as the inverse of rms cone contrast. The dotted line

shows the chromatic sensitivity in free viewing, the dashed line the luminance sensitivity. Note that not only is there no desensitisation for chromatic discrimination just after the saccade, there is actually a marked increase in sensitivity for a period of up to 200 msec after the saccade. (Data reproduced with permission from Burr et al., 1994.)

black stimuli (both of very low spatial frequency) as a function of time after saccadic onset. Luminance discrimination (*filled circles*) was severely impaired just after saccadic onset, by one log unit, steadily improving to normal levels over a 200 msec period. Chromatic sensitivity (*filled squares*), on the other hand, was not at all impaired at around the time of the saccade, and actually improved over the period following the saccade, by approximately a factor of 2. This is an example of saccadic enhancement of contrast sensitivity, similar to that previously observed by Burr et al. (1982) for relatively high frequencies of luminance modulation (see Figure 93.1C) that presumably stimulate the same P-pathways.

These results, showing that equiluminant stimuli (irrespective of the spatial frequency) are not suppressed during saccades and can actually be enhanced, imply that saccadic suppression is specific to the magnocellular pathway. The parvocellular pathway, essential for chromatic discrimination, is left unimpaired. Using a different technique, Uchikawa and Sato (1995) arrived at a similar conclusion. They measured incremental spectral sensitivity for detecting monochromatic discs displayed against a white background during normal viewing and saccades. They showed that during saccades, the spectral sensitivity curve showed a marked decrease at ~570 nm (known as *Sloan's notch*), a clear signature of the spectrally opposed mechanisms of the parvocellular system. In normal viewing, this decrease was absent (for brief stimuli), suggestive of magnocellular function. Their results are replotted in Figure 93.3, together with representative measurements of responses of P and M retinal ganglion cells of macaque monkey. The psychophysical detection thresholds during normal viewing follow closely the responses of M cells, while during saccades they are more like those of P cells.

A fundamental question provoked by these studies was whether saccadic suppression results from a central signal, such as the corollary discharge proposed by Sperry (1950) and Von Holst and Mittelstädt (1954), or whether the visual motion caused by the eye movement itself masks vision during saccades (Campbell and Wurtz, 1978; Castet et al., 2001; MacKay, 1970, 1973). There is good evidence that image motion of the kind caused by saccades can mask brief stimuli (Campbell and Wurtz, 1978; Derrington, 1984; MacKay, 1973), but is this the only, or indeed the principal, mechanism at work? Diamond et al. (2000) simulated visual saccades by optically deflecting the display at suitable speed, amplitude, and acceleration, and measured contrast sensitivity to briefly displayed gratings. When the target gratings were displayed on an otherwise blank screen, simulated saccades had little effect on thresholds compared with real saccades. However, when a high-contrast random pattern was added to the display to provide a strong spurious visual motion signal, the simulated saccade produced a suppression

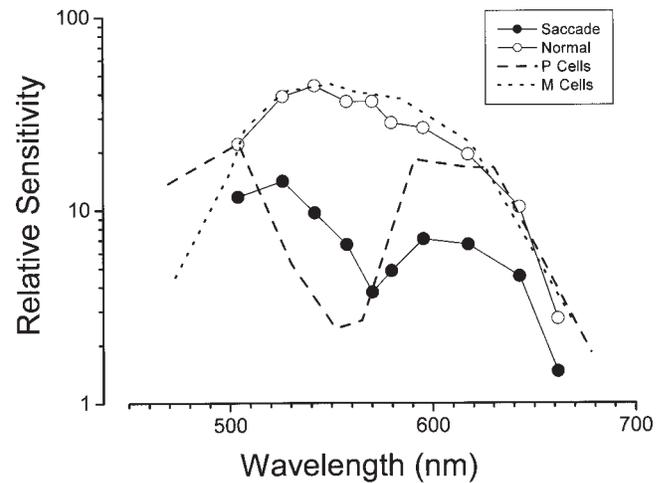


FIGURE 93.3. Sensitivity (in relative units) for detecting a monochromatic bar briefly presented on a white background in normal viewing (*open circles*) or at the onset of a 6 degree saccade. In normal viewing, the curve has a broad peak around 550 nm and closely follows the sensitivity of a sample of retinal M cells in the macaque monkey (taken from Zrenner, 1983). During saccades, however, the form of the curves changes dramatically to reveal Sloan's notch, a sharp dip in sensitivity at around 570 nm. The dashed lines show the average response of a population of P cells. While not following the human sensitivity data exactly, they show the same characteristic dip for middle wavelengths. This is very strong evidence for suppression of magnocellular activity during saccades. (Adapted with permission from Uchikawa and Sato, 1995, and Zrenner, 1983.)

that was comparable in magnitude to and lasted longer than that produced by the real saccade. This result suggests that visual masking can be important for vision at around the time of saccades but that it is not the only mechanism. There must also be a signal of nonvisual origin that accompanies each real saccade to decrease sensitivity to low-frequency, luminance-modulated stimuli. Other evidence for a non-visual suppression signal is that visual phosphenes generated by applying weak electrical signals to the eye in darkness are suppressed during saccades by a comparable amount to real light images (Riggs et al., 1974).

The similarity between the time course of saccadic suppression and visual masking observed by Diamond et al. (2000) could indicate that these two phenomena have a common site of action. This seems reasonable, given the evidence that saccadic suppression occurs early, preceding the site of contrast masking (Burr et al., 1994) and motion analysis (Burr et al., 1999). One interesting possibility is that both saccadic suppression and masking act on *contrast gain mechanisms* of cortical and/or geniculate cells. This idea predicts that saccades should not only decrease sensitivity, as shown in Figures 93.1 and 93.2, but should also cause the system to respond more rapidly. This is what occurs. During saccades, the temporal impulse response function

becomes more rapid (Burr and Morrone, 1996), as many models of contrast gain (e.g., Shapley and Victor, 1981) would predict.

The fact that the impulse response accelerates rather than decelerates during saccades suggests that although strongly attenuated, the magno system remains active during saccades (a parvo-dominated response should be slower). This is consistent with experiments demonstrating that under certain conditions, saccades in the direction of a rapidly moving, high-contrast grating can improve direction discrimination of that stimulus (Castet and Masson, 2000; Garcia-Perez and Peli, 2001). While these demonstrations clearly do not refute the existence of a centrally driven suppression mechanism, they do highlight the important point that centrally driven saccadic suppression only attenuates motion detection, not eliminating it completely, leaving an important role for other mechanisms, such as masking, in natural (usually visually rich) viewing conditions (Campbell and Wurtz, 1978; Derrington, 1984; Diamond et al., 2000; MacKay, 1973).

The studies discussed above all refer to moderate to large saccades and might not be applicable to the microsaccades that are normally made, together with slow drifts, around the fixation point. Most evidence suggests that small saccades cause little or no threshold elevation (Krauskopf et al., 1966; Sperling, 1990), indicating that the effects of the image tremor may be controlled by other means. Murakami and Cavanagh (1998, 2001) 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 favoring 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. Murakami and Cavanagh claim that the reduction in motion sensitivity caused by adaptation reduces the estimate of the baseline jitter so that the motion caused by eye jitter becomes superthreshold in the unadapted region and hence visible. This idea is particularly interesting in the context of older theories that suggested that stabilization is achieved by subtraction of extraretinal 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.

In conclusion, human psychophysical data clearly suggest an extraretinal suppression of early visual activity during saccades. There is also much neurophysiological evidence pointing to the underlying neural mechanisms of suppression, but there is no clear consensus on this: some studies show clear evidence of suppression, but others do not. Still others point to more complicated effects, such as an inversion of directional selectivity of MT cells during saccades

(Thiele et al., 2002). For a recent review of some of this literature, readers are referred to Ross et al. (2001).

Perceived position

Retinal motion is not the only problem introduced by saccades. A related problem is how the visual system manages to maintain a stable sense of visual direction while the retinal input is constantly changing. Helmholtz (1866) believed that the constancy of perceived position was maintained during and after saccades, because both extraretinal information (the *effort of will*) and retinal information (sensed but not perceived image motion) were used to recalibrate the direction of gaze.

Leonard Matin and colleagues (Matin, 1972; Matin and Pearce, 1965; Matin et al., 1969, 1970) and Bischof and Kramer (1968) were among the first to find errors in localization at the time of eye movements. Figure 93.4A replots data from Bischof and Kramer showing the perceived position of a stimulus briefly flashed at a variable interval after observers initiated a 16 degree saccade. There is a strong mislocalization immediately after the saccade onset in the direction of the saccade, about half of its amplitude. Halfway through the saccade, the direction of the misperception inverts before returning to veridical. This experiment has since been replicated by many observers, including Honda (1989, 1991, 1993), Schlag and Schlag-Rey (1995), and Dassonville et al. (1992). The curve in Figure 93.4 was replotted in a more recent study by Ross et al. (1997) that measured the perceived position of a bar briefly flashed at various times over a period preceding and following the saccade. The results are quite similar to those of Bischof and Kramer (except for the rebound after the saccade in this instance, although it has been observed under other conditions). They also show that the mislocalizations precede the saccade, before the eye has begun to move, reaching a maximum at around saccadic onset. The maximum shift is again about half the size of the saccade, suggesting a relative gain of 0.5, as observed by others (Bridgeman, 1995).

If the errors in localization are to compensate for eye movements, they should be in the same direction as the saccade. However, this is not always the case, as first observed by Bischof and Kramer (1968) and studied systematically by Ross et al. (1997). Figure 93.5A shows reports of perceived positions of visual targets presented just before a saccade as a function of the actual position of the bar. The small symbols show data on when the bars were flashed during control fixation conditions: localization was veridical and accurate. However, when the same bar was flashed to the stationary eye just prior to a saccade, it was seen to be displaced, but the direction of the displacement depended critically on its position. Bars flashed to the left of the

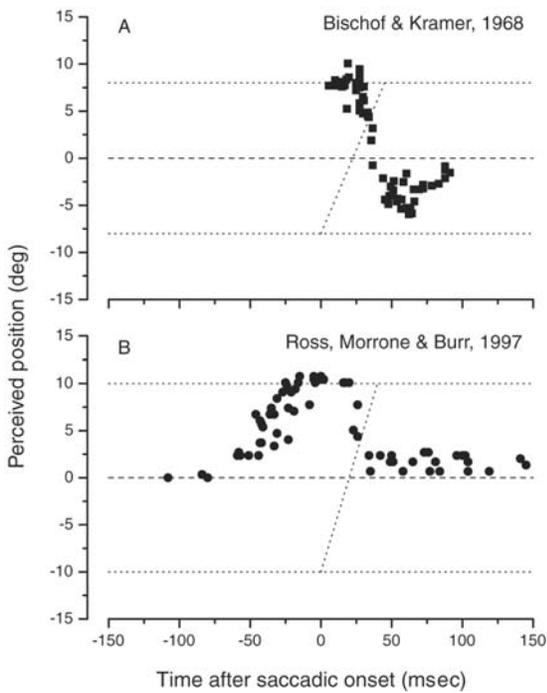


FIGURE 93.4. Perceived positions for stimuli briefly presented at various times relative to the onset of a rightward horizontal saccade (shown by the dotted lines). The dashed line shows the veridical position of the target. The upper curve show data reproduced from Figure 3 of Bischof and Kramer (1968), the lower curves from Figure 1 of Ross et al. (1997). Both data show a strong mislocalization of maximal strength near the onset of the saccade.

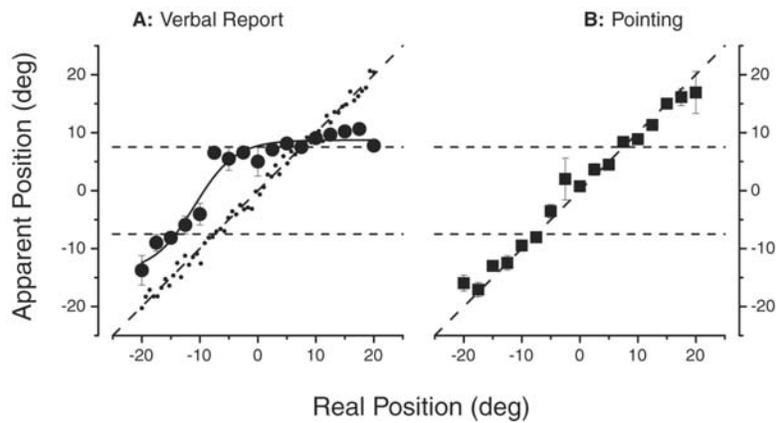


FIGURE 93.5. Perceived positions of bars briefly flashed to stationary eyes in the 40 msec before observers made a 20 degree saccade. The large filled circles in *A* show results for verbal reports in which subjects called out a number from a remembered scale. In this example (taken from Burr et al., 2001), the subject closed her eyes immediately after making the saccade. The curve follow-

saccadic target were displaced rightward, in the direction of the saccade. However, bars flashed beyond the saccadic target were displaced leftward, against the direction of the saccade, resulting in a general compression. This compression is powerful enough to remove vernier offsets for line targets that are flashed at about the time of saccade onset and can create offsets for collinear line targets flashed at different times (75 msec apart). Compression can even cause four bars, flashed 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.

Compression does not always accompany saccades. For example, Cai et al. (1997) reported no compression occurring under the conditions of their experiment. The issue was at least partially resolved by Lappe et al. (2000), who reported compression only in conditions where visual referents were perceived after the saccade. Another major difference is that in order to remove visual referents, Lappe et al. performed their experiments at very low levels of illumination, near darkness, as did many of the previous investigations that report no compression (Cai et al., 1997).

As with saccadic suppression, it is important to understand if the apparent shifts and compression are caused by the visual events associated with the saccade or if they have a central origin. As before, simulating saccades by rapid

ing the data is the output of a model for the shift and compression effects (Morrone et al., 1997). The small circles show control data taken under identical conditions during fixation (from Ross et al., 1997). The filled squares in *B* show results for blind pointing to the remembered positions, again with the eyes shut. (Adapted with permission from Burr et al., 2001.)

rotation of a mirror provides crucial information. Mislocalizations can occur during simulated saccades, but they have a very different time course and magnitude than those during real saccades (Morrone et al., 1997). More significantly, no compression occurs with simulated saccades: all stimuli are displaced in the same direction and to the same extent, irrespective of their position in the visual field (Morrone et al., 1997).

There exists a good deal of evidence for the involvement of extraretinal eye position information in judging apparent visual direction, although some of it is contradictory. Helmholtz (1866) noted that causing the eye to move by applying pressure with the finger (creating an eye movement without an “intention to move” signal) caused an apparent displacement on the visual world. However, the inverse experiment, attempting to move the eyes under conditions of total paralysis, did not create a sense of apparent motion of the visual scene (Brindley et al., 1976; Stevens et al., 1976), even though there should have been a mismatch between the extraretinal signal and the retinal movement (or lack thereof). The issue has been readdressed by Matin et al. (1981), who showed that with partial systemic paralysis, observers make enormous errors in visual localization, on the order of 20 degrees. In darkness, partially paralyzed (but not normal) observers misperceive completely the median plane and make gross errors in pointing to visual targets. Most significantly, when using an auditory tone to localize a visual target, partially paralyzed observers grossly overestimate the eccentricity (Fig. 93.6). With full room illumination, the perception of the median plane and the pointing performance to visual targets return to normal, but the auditory-to-visual matches remain as distorted as they are in darkness (Fig. 93.6). This experiment provides clear evidence for the role of extraretinal eye position information in judging apparent visual direction. When the muscular response is weakened through partial paralysis, the motor command for a given response must be increased. The fact that the increase in gain of the motor command produces a commensurable increase in perceived eccentricity suggests that the extraretinal signal combines with retinal information in determining visual location. It seems that when present, visual information can dominate in determining the medial plane (possibly explaining the previous null results of Brindley et al., 1976, and Stevens et al., 1976); but the visual illumination is not sufficient to override the erroneous eccentricity estimates in the auditory-to-visual matches.

Motor responses during saccades

Although there is overwhelming evidence for perceptual mislocalizations during saccades, there is also good evidence that subjects maintain a veridical spatial map, and that this map can be used to guide motor activity such as second sac-

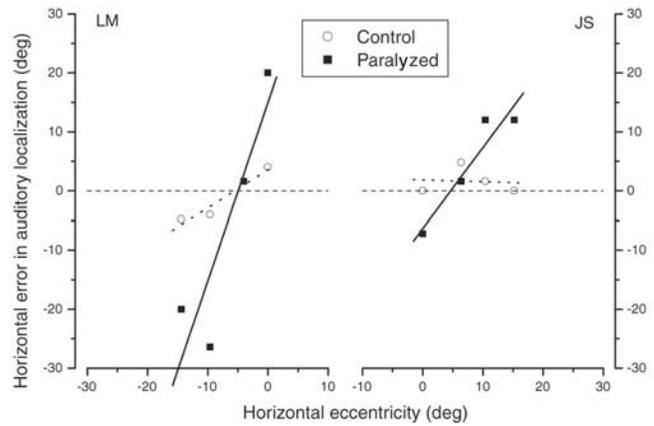


FIGURE 93.6. Error in auditory localizations of fixed visual targets in observers that have been partially paralyzed by curare (*filled squares*) compared with control measurements in the same observers (*open circles*). During paralysis, observers overestimated the distance of the light from the median by more than a factor of 2. These results suggest that extraretinal information used to control eye position is used in estimating eccentricity. These results were the same in darkness as in normal lighting conditions. (From Matin et al., 1981, with permission.)

cades and hammering (Hallett and Lightstone, 1976a, 1976b; Hansen and Skavenski, 1977, 1985). Bridgeman et al. (1975) also reported that subjects can point accurately to targets that were displaced perisaccadically, even though the subject did not perceive the change in target position. 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 long-standing (Trevarthen, 1968) and recently revived (Goodale and Milner, 1992) idea that separate systems are responsible for conscious perception and for direct interactions with our environment.

While the early evidence was clear, some recent experiments on pointing and secondary saccades have failed to replicate the original dissociation between motor accuracy and perceptual mislocalization during saccades, reporting localization errors for both tasks (Bockisch and Miller, 1999; Dassonville et al., 1992, 1995; Honda, 1991; Miller, 1996; Schlag and Schlag-Rey, 1995). However, it should be pointed out that localization errors for motor responses vary considerably both in size and in dynamics in different laboratories: some show errors as large as the perceptual errors, starting well in advance of saccadic eye movements (Dassonville et al., 1992, 1995; Schlag and Schlag-Rey, 1995); others show very small errors (4 to 5 degrees for 15 degree saccades), mainly for targets presented at the completion of the saccade (Bockisch and Miller, 1999; Miller, 1996). The errors have been reported to be similar for finger indication and for secondary saccades. Unfortunately, all these studies used different experimental paradigms, with various levels of dark adaptation, visual references from the eye recording systems,

and possible visual cues introduced from diffuse or reflected light from the apparatus (Bockisch and Miller, 1999; Dassonville et al., 1992; Miller, 1996).

We have recently revisited the problem to see whether the compression of visual space that occurs with verbal reports also occurs with blind pointing under matched conditions (Burr et al., 2001; see also Husain and Jackson, 2001). When asked to report verbally the position of a bar flashed just before a saccade, subjects made strong compression errors, as mentioned before and plotted in Figure 93.5A. This occurred even when all visual references were removed by obscuring the screen at the time of the report. However, when subjects were asked to point ballistically to the location where a bar had been seen (again with transient darkness caused by eye shutting), the pattern of pointing was virtually veridical (Fig. 93.5B). This supports the idea that there are two separate visual representations with separate remapping during saccades. Importantly, when subjects were asked to point with visual cues available (but the hands obscured from view), the pattern of errors was like that for verbal reports. This suggests that, when available, the perceptual representation can override the motor representation of visual space. It is also possible that this result explains the conflicting results in the literature. Hansen and Skavenski (1977, 1985) used the technique of transient darkness (with a light-adapted eye) to remove visual cues, and they reported accurate motor response, similar to those in Figure 93.5B. On the other hand, fully dark-adapted subjects, viewing stimuli through eye sensors that provided clear visual references (Schlag and Schlag-Rey, 1995), made substantial errors on secondary saccades in the direction of the primary saccade.

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. (1996) repeated Bridgeman et al.'s (1975) measurements of sensitivity to line displacements at the time of saccades. The saccade typically raises thresholds by more than a factor of 3. However, if a line is briefly blanked at the time of the saccade and reappears ~100 msec 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. Interestingly, even under conditions where observers are unable to report correctly the direction of the displacement, they frequently make an accurate second saccade toward the displaced target (Deubel et al., 1998).

In conclusion, the bulk of the evidence suggests that veridical pointing and second saccades can occur with perisaccadic targets under conditions where perception is severely distorted. However, in order to demonstrate veridical motor responses, care must be taken to render the visual signals unreliable (that does not only entail removal of visual

references), or else vision will dominate. The results suggest the existence of separate representations for perception and action that are affected differently during saccades. However, there is clearly considerable interaction between the representations, and when there is conflict, the perceptual system seems to dominate.

Physiological mechanisms

In an important paper, Duhamel et al. (1992) showed that the receptive fields of some neurons in the lateral intraparietal area (LIP) of monkeys change position before each saccadic eye movement, effectively anticipating its consequences. Up to 80 msec before the onset of a saccade, LIP neurons start to respond to stimuli that will fall within their receptive field after the saccade is completed. This predictive effect might represent a mechanism by which the visual and oculomotor systems combine and calculate a spatially accurate image of the world in spite of the eye movement. This could enable neurons 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 (Gottlieb et al., 1998).

Perisaccadic receptive field shifts are not unique to the LIP but have been found in other eye movement-related areas—for example, the superior colliculus (Walker et al., 1995) and the frontal eye field (Umeno and Goldberg, 1997), a medial parietal area that is associated with reaching (Batista et al., 1999)—and even in earlier stages in the cortical visual system, V4, V3a, and V2 (Nakamura and Colby, 2002). The remapping of receptive fields in all these areas is consistent with visual mislocalization in the direction of the saccade. It is not, however, obvious how it may explain the observed compression: compression requires a shift that is dependent on the position of the stimuli.

One possibility is that during the perisaccadic interval, not all LIP neurons (and other neurons in the parietal stream) show the predictive shift of receptive field in the direction of the saccade. Other neurons continue to respond to the pre-saccadic position, and some respond to stimuli in both positions (Kusunoki et al., 1997; Nakamura and Colby, 2000). 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, resulting in compression, particularly of the positions of stimuli that are flashed briefly during psychophysical experiments. The fact that many areas show anticipatory shifts in receptive fields, to a greater or lesser extent, could mean that multiple representations in all these areas in some way contribute to the observed compression.

Neural correlates of perisaccadic mislocalization have also been observed in MT and MST neurons. Using a

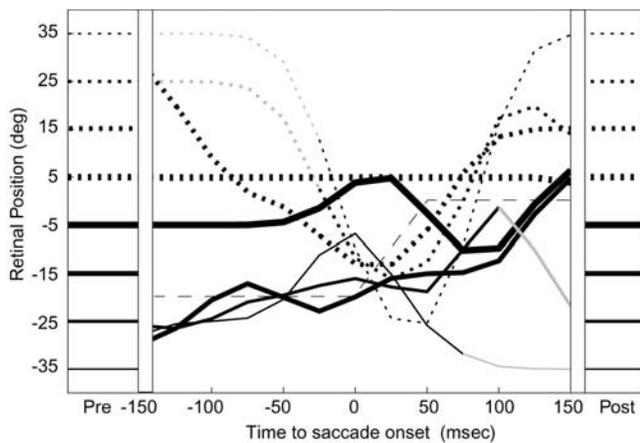


FIGURE 93.7. Perisaccadic compression of space in areas MT and MST. The curves of various widths and line types indicate the predictions of the perceived stimulus position of a population of MT and MST neurons, as a function of time relative to saccadic onset, for bars stimulated at various positions (actual positions are indicated at the left and right of the graph). The animal made 20 degree leftward saccades, as indicated by the faint dashed black curves. Well before and well after saccadic onset, the model prediction from the cell population is very close to veridical. However, at times near saccadic onset, the population code tends to converge near the saccadic target, implying compression. (From Krekelberg et al., 2003, with permission.)

Bayesian analysis, Krekelberg et al. (2003) show that a population of MT and MST cells can encode veridically the position of briefly flashed bars. However, analysis of the same neurons just prior to a saccade (using the weights derived from free viewing) shows a gross mislocalization in the population response, shown in Figure 93.7. The mislocalization is in the direction of compression, as observed with human observers. Interestingly, both the magnitude of the compression and the time course are very similar to those observed with human observers (Morrone et al., 1997; Ross et al., 1997).

While the work is new and very much in progress, it is fair to say that strong neural correlates exist between the psychophysical and electrophysiological single-unit findings, to account for both the systematic shift in the direction of the saccade and the compression toward the saccadic target. The areas involved include V3, LIP, VIP, MT, MST, and superior colliculus.

Future directions

At the time of saccades, vision is profoundly modified. There is a transient desensitisation of low-frequency stimuli modulated in luminance together with gross distortions of perceptual (but not motor) space. The time course of perceptual compression is very similar to that for loss of sensitivity: both begin more than 50 msec before saccades start, are maximal

at or shortly before the start of saccades, diminish during saccades, and disappear only after saccades have ended. These psychophysical findings, summarized in this chapter, have been confirmed in many laboratories and have spurred the successful search for neural correlates in the single neurons of nonhuman primates.

What is the functional role of these perisaccadic changes? How do they enable the visual system to perceive a steady world from input arriving from such unsteady sensory platforms? As discussed earlier, the transient suppression of low spatial frequency luminance information probably serves to dampen the motion system (or one part of the motion system), blunting the otherwise disturbing sensation of movement the rapid displacement should cause. But as we have seen, the suppression is very brief, lasting for only about 50 msec, and by no means total, 1 log unit at most. How can this brief, partial suppression obliterate the extended motion that occurs with each eye movement? Indeed, we know that motion is not totally suppressed in some cases (Castet and Masson, 2000) and that part of the magnocellular system remains operative at reduced gain (Burr and Morrone, 1996). One fruitful line for future research will be to simulate saccadic vision by large-field motion and spatial blur to investigate whether this transient partial attenuation of luminance, but not color, is sufficient to blunt the motion sensation or whether other factors are involved.

The perceptual distortions are even more perplexing: a shift in the direction of the saccade combined with gross compression of perceptual space toward the saccadic target. There is clear neural activity in the parietal pathway correlated with these two processes. But what is their function? Do the shift and compression facilitate directly the perceptual transitions from one fixation to another or is it an epiphenomenon of some more subtle, still not understood process?

Finally, there is now very good evidence that at the time of these gross perceptual distortions, veridical information remains available during saccades, information that can be used to guide ballistic motor acts and secondary saccades. What is the nature of the spatial representation used in this case? Why is this information normally unavailable to perception? Which visual pathway mediates the spatial map used by the action system? How can the retention of this ability be reconciled with the psychophysical and physiological demonstrations of massive suppression of the magno (dorsal) pathway's activity during saccade?

While much progress has been made in documenting the psychophysical and electrophysiological events that accompany saccades, many of the important questions about visual stability remain unsolved and will provide a fertile terrain for research for the next few decades.

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