66 Interaction between Eye Movements and Vision: Perception during Saccades

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Vision is always clear and stable, despite continual saccadic eye movements that actively reposition our gaze, two to three times a second. Saccades may be made deliberately, but normally they are automatic and pass unnoticed. Not only does the actual movement of the eyes escape notice, so too does the motion of images as they sweep across the retina and the fact that gaze itself has been repositioned. The world seems to stay put. Comparable image motion produced externally, rather than by movements of the observer's own eyes, has an alarming effect on the observer's sense of stability. The problem of visual stability is an old one that has fascinated many scientists, including Descartes, Helmholtz, Mach, and Sherrington, and indeed goes back to the eleventh-century 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 eyes 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 objects to be in motion" (Alhazen, 1083). However, only recently have the tools become available to monitor eye movements accurately and to measure their effects qualitatively.

The problem of visual stability during eye movements can be broadly divided into two separate issues: Why do we not perceive the *motion* of the retinal image produced as the eye sweeps over the visual field—and how do we cope dynamically "online" with the continual changes in the retinal image produced by each saccade to construct a stable representation of the world from the successive "snapshots" of each fixation? Although the problem of visual stability is far from solved, tantalizing progress has been made over the last few years. This chapter highlights the progress made in understanding visual perception during large saccades: For small saccade, drift and pursuit eye movements, please refer to chapters 60–65.

SACCADIC SUPPRESSION

Part of the general problem of visual stability is why the fast motion of the retinal image generated by the movement of the eyes completely escapes notice: Comparable wide-field motion generated externally is highly visible and somewhat disturbing (Allison et al., 2010; Burr et al., 1982). It has long been suspected that vision is somehow suppressed during saccades (Holt, 1903), but the nature of the suppression has remained elusive. Now it is clear that the suppression is neither a "central anaesthesia" of the visual system (Holt, 1903), nor a "grey-out" of the world due to fast motion (Campbell & Wurtz, 1978; Dodge, 1900; Woodworth, 1906), as this motion is actually visible, extremely so at low spatial frequencies (Burr & Ross, 1982). What happens is that some stimuli are actively suppressed by saccades while others are not: Stimuli of low spatial frequencies are very difficult to detect if flashed just prior to a saccade while stimuli of high spatial frequencies remain equally visible (Burr et al., 1982; Volkmann et al., 1978). *Equiluminant* stimuli (varying in color but not luminance) are not suppressed during saccades and can even be enhanced (Burr, Morrone, & Ross, 1994), implying that the parvocellular pathway, essential for chromatic discrimination, is left unimpaired while the magnocellular pathway is specifically suppressed (Castet & Masson, 2000; Shioiri & Cavanagh, 1989).

Saccadic suppression follows a specific and very tight time course, illustrated in figure 66.1A (replotted from Diamond, Ross, & Morrone, 2000), very different from saccadic enhancement for equiluminant stimuli (figure 66.1C, replotted from Knoll et al., 2011). Sensitivity for seeing low-spatial-frequency, luminance-modulated stimuli declines 25 ms before saccadic onset, reaching a minimum at the onset of the saccade, then rapidly recovering to normal levels 50 ms afterwards. The suppression effect is multiplicative and is homogeneous at all eccentricities (Knoll et al., 2011), in contrast to what

was previously postulated (Mitrani, Mateeff, & Yakimoff, 1970). Does the suppression result from a central nonvisual "*corollary discharge*" signal (discussed in chapter 66), or could it result simply from visual "masking" effects? This would seem unlikely, as great care was taken to ensure a uniform surround. However, the question is important. In order to be certain that the saccade itself was essential for the suppression, we simulated saccadic eye movements by viewing the stimulus setup through a mirror that could be rotated at saccadic speeds. When the background was uniform, with minimal visual references, the simulated saccades had little or no effect on sensitivity (open symbols of figure 66.1A).

However, that is not to say that under more natural conditions masking does not occur. When the test stimulus is embedded within a textured screen, simulated saccades do decrease contrast sensitivity (see figure 66.1B). Indeed, the maximum suppression is nearly as great as that caused by real saccades and lasts for much longer. This suggests that after the saccade, sensitivity is greater than that expected with comparable motion without the saccade, possibly implying a postsaccadic facilitation. The timing for this postsaccadic facilitation is very similar to that observed for equiluminant stimuli, but for these stimuli the simulated and real saccade produce the same effects (closed and open symbols of figure 66.1C), indicating that the facilitation is related

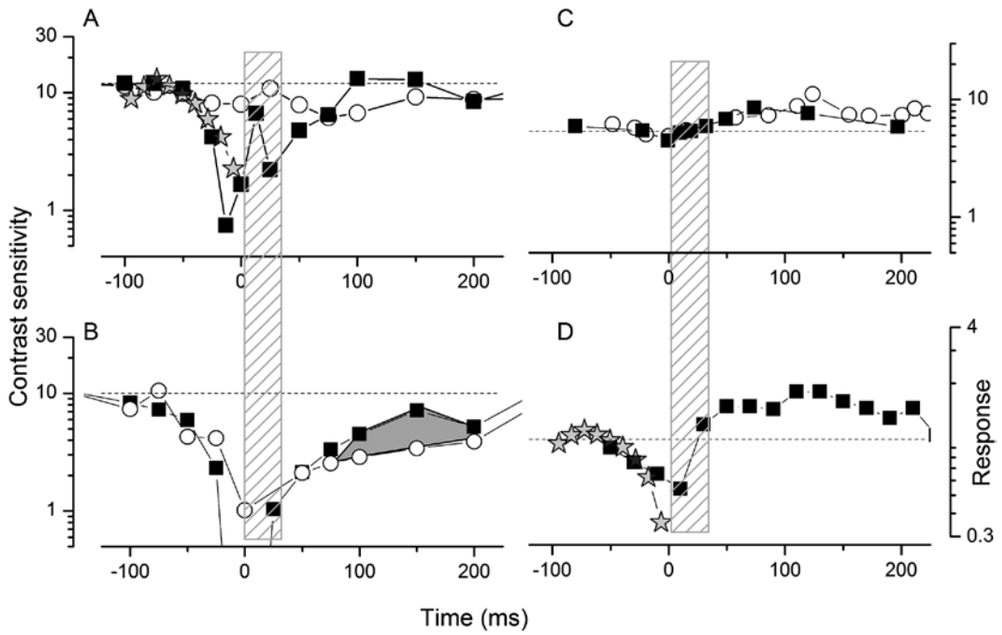

FIGURE 66.1 The effect of saccades on human contrast sensitivity, human blood-oxygen-level dependent (BOLD) response, and firing rate in monkey middle temporal visual cortex (MT; reproduced with permission from Diamond, Ross, & Morrone, 2000; Ibbotson & Cloherty, 2009; Knoll et al., 2011; Vallines & Greenlee, 2006). (A) Filled squares show contrast sensitivity for discriminating (in two-alternative forced choice) the brightness of a brief, low-frequency, luminance-modulated grating patch, as a function of time relative to saccadic onset. The background was of mean luminance, with very few visual referents present. Sensitivity is severely reduced (by more than a log-unit) at saccadic onset. The open circles show measurements made in identical conditions, but instead of making a saccade, a mirror moved at the same speed and amplitude as the saccade; this had very little effect on sensitivity. The stars plot the exponential of the BOLD amplitude of a portion of V1 representing a small patch of grating presented briefly before a saccade. The exponential transformation is applied under the hypothesis that BOLD amplitude is proportional to log contrast sensitivity. (B) As for (A) except that the background was a high-contrast random check pattern. With a structured background, the simulated saccade did reduce visibility, presumably by masking, with the effect lasting longer than it did for a real saccade. The gray shaded area indicates the region where sensitivity was greater during the saccade than in fixation. (C) As for (A) except that the stimulus was a small patch of equiluminant red–green sinusoidal grating presented 5° rightwards and 2° upward of presaccadic fixation. Contrast sensitivity is the inverse of threshold cone contrast. The open symbols report data for the simulated saccade. Note the twofold facilitation in both sets of data. (D) Firing rate of a typical MT neuron in awake monkey in response to stimulation with a brief stimulus (squares) and human V1 BOLD activity (stars), as a function of time relative to saccadic onset. The pattern of the response is similar to the psychophysical results of (A). The enhancement after the saccade may allow for the more rapid recovery from masking during real rather than simulated saccades (difference between filled and open symbols in part B).

to the spurious retinal motion and not to an active mechanism associated with the saccade.

That real saccades cause a different pattern of luminance-contrast sensitivities from simulated saccades shows that suppression results at least in part from an active, extraretinal signal. Interestingly the amount of suppression varies with age, being much stronger in adolescent children than in adults (Bruno et al., 2006), even though motion perception and masking are largely adult-like by that age (Maurer, Lewis, & Mondloch, 2005; Parrish et al., 2005). This indicates that the mechanisms mediating suppression are still developing into late adolescence. As the saccadic motor system is also not completely mature during adolescence (Fischer, Biscaldi, & Gezeck, 1997), this is further evidence that the extraretinal signal responsible for mediating the saccadic suppression may be linked to the motor system.

Psychophysical studies indicate that saccadic suppression occurs early in the visual system (Burr, Morrone, & Ross, 1994), at or before the site of contrast masking (Watson & Krekelberg, 2011), and before low-level motion processing (Burr, Morgan, & Morrone, 1999). Thilo et al. (2003) addressed this question more directly with a clever electrophysiological technique. Replicating an old study by Riggs, Merton, and Morton (1974), they showed that visual phosphenes produced by electrical stimulation of the eye are suppressed during saccades. However, phosphenes of cortical origin—V1 or V2—generated with the technique of transcranial magnetic stimulation (TMS) were not suppressed. This strongly suggests that saccadic suppression occurs early, before the site of generation of cortical phosphenes, probably within the lateral geniculate nucleus (LGN) or perhaps within V1 itself. A functional magnetic resonance imaging (fMRI; Sylvester, Haynes, & Rees, 2005) study that measured blood-oxygen-level-dependent (BOLD) activity of LGN while subjects made saccades over a field of constant illumination (to avoid the generation of spurious retinal motion) showed a clear suppression in both LGN and V1, reinforcing early suggestions of saccadic suppression in the dark in V1 (Bodis-Wollner, Bucher, & Seelos, 1999; Paus et al., 1995). Interestingly, the amplitude of the BOLD responses in V1 decreased as the stimuli were presented closer to the saccadic onset, following a dynamic similar to that observed psychophysically (see the stars in figure 66.1A, taken from Vallines & Greenlee, 2006), again suggesting an early site of action.

There is also fMRI evidence for postthalamic modulation by saccades. The BOLD response to luminance stimuli is relatively suppressed compared with that to chromatic stimuli during saccades, but the attenuation varies across areas (Kleiser, Seitz, & Krekelberg, 2004), strong in middle temporal visual cortex (MT)—as expected—but also strong in V4, a cortical area receiving more parvocellular than magnocellular input. That some form of suppression can take place after form analysis is also suggested by the psychophysical result that a suppressed line can influence perception of a form presented postsaccadically (Watson & Krekelberg, 2009). There is also evidence of suppression in higher neural levels, in areas normally associated with attention (Bristow et al., 2005; Kleiser, Seitz, & Krekelberg, 2004). This is interesting, as it could be the suppression of the high-order "attention related" areas that prevents the sense of motion from entering into awareness, causing startle (Allison et al., 2010; Burr et al., 1982).

The electrophysiology of saccadic suppression is more complex. Electrophysiological studies show that the majority of cells in V1 respond vigorously to the movement created by saccades; however, some cells do not respond to saccade-generated motion, but only to real motion in the external world. These cells are the minority, about 10% in V1, 15% in V2, and 40% in V3A (Galletti & Fattori, 2003; Wurtz, 2008). Reppas, Usrey, and Reid (2002) have shown that voluntary saccades induce profound changes in the response of LGN cells, particularly magnocellular cells: Activity is depressed around the time of the saccade, and there is also a larger and long-lasting enhancement after the saccade. There is also clear evidence for strong suppression in the colliculus and pulvinar, which may be important for the suppression of fast motion (see also chapter 65, Wurtz, 2008).

Perhaps the data that can be most readily compared with the psychophysical sensitivities are those in Ibbotson, Crowder, Cloherty, Price and Mustari (2008) and Bremmer et al. (2009), who measured responsiveness of MT/medial superior temporal (MT/MST) cells to a brief stimulus, similar to that used in the psychophysics experiments. Data from a cell (taken from Ibbotson & Cloherty, 2009) are replotted in figure 66.1D. This cell showed a very strong and robust suppression before the start of the saccade, followed by a clear enhancement lasting some 200 ms after the termination of the saccade. While it is difficult to make a quantitative comparison between psychophysical threshold measurements (figure 66.1A) and firing rates of one representative MT cell (figure 66.1D), it is interesting that modulation of MT/MST response follows a similar time course to sensitivity for a brief low-spatial-frequency stimulus. It also follows V1 BOLD activity (stars) (Vallines & Greenlee, 2006), presumably reflecting responses from magnocellular/MT–MST pathways (Bremmer et al., 2009) before the saccade. The very strong postsaccadic enhancement of MT cells, which is present also in total darkness,

could explain the relatively higher sensitivity after real saccades compared with after simulated saccades (the difference between open and closed symbols of figure 66.1B). Another interesting result reported for MT neurons is that, in addition to being suppressed, many neurons seem to reverse their preferred direction selectivity (Thiele et al., 2002). This odd behavior could be important in "canceling" the spurious motion information generated by the eye movement, helping to keep the world still. Other areas like ventral intraparietal and lateral intraparietal (LIP) cortex do not show a suppression with similar time course to that observed in MT (Bremmer et al., 2009), pointing again to a specific suppression of the M-pathways and of motion perception (Allison et al., 2010; Burr et al., 1982; Burr, Morrone, & Ross, 1994; Shioiri & Cavanagh, 1989).

To conclude, it is not surprising that saccadic suppression should occur at different levels. Many basic sensory phenomena, such as gain control, do not occur at a single site but at virtually every possible location: photoreceptors, retinal ganglion cells, LGN cells, and cortex (Shapley & Enroth-Cugell, 1984). Indeed, the parallels between saccadic suppression and contrast gain control are strong, suggesting that they may share similar mechanisms. During saccades, the temporal impulse response to luminance, but not to equiluminant stimuli, becomes faster and more transient (Burr & Morrone, 1996). Both LGN (Reppas, Usrey, & Reid, 2002) and MT/MST (Ibbotson et al., 2008) cells show a similar response pattern, with faster and more transient impulse response functions during saccades. These results suggest that saccadic suppression may act by attenuating the contrast gain of the neuronal response, causing a faster impulse response (Shapley & Victor, 1981). Changing contrast gain makes neurons less responsive to low-contrast stimuli, decreasing the effectiveness of the spurious signals caused by the saccade, hence facilitating the recovery to normal sensitivity. That saccadic suppression operates via gain-control mechanisms is consistent with the selective suppression of the magnocellular pathway, as M-cells have much stronger gain control than P-cells (Sclar, Maunsell, & Lennie, 1990). This would certainly be an elegant and economical solution to the problem of saccadic suppression, taking advantage of mechanisms already in place.

The idea that gain control explains both the suppression and the rapid recovery during saccades has been implemented in a model that simulates quantitatively the time course of contrast sensitivity in normal and simulated saccades (Diamond, Ross, & Morrone, 2000). Interestingly, changing response gain is one of the few mechanisms that can explain simultaneously many saccadic suppression properties. It can account for the similarity in sensitivities of real and simulated saccades in the presence of a noise background, but not with a homogeneous background; and it can explain the dependence of suppression from input noise (Watson & Krekelberg, 2011). It can also explain the postsaccadic enhancement, the change of the impulse response function (Burr & Morrone, 1996), and also the change in the strength of masking between brief pre- and postsaccadic stimuli (Burr, Morrone, & Ross, 1994). By operating on gain-control mechanisms, saccadic suppression would serve two important roles: the suppression of image motion, which would otherwise be disturbing, and the rapid return to normal sensitivity after the saccade.

DYNAMIC UPDATING OF INTERNAL SPATIAL MAPS

Besides the (relatively) simple problem of suppressing the motion caused by the fast-moving image on the retina, the brain must also take into account the saccadic movement when determining the instantaneous position of objects in space. Like Alhazen, Helmholtz (1866) also recognized that "the effort of will involved in trying to alter the adjustment of the eyes" could be used to help stabilize perception. Models based on similar ideas of compensation of eye movements were proposed by Sperry (1950) in the 1950s with the concept of corollary discharge and by Von Holst and Mittelstädt (1954) of efference copy: The effort of will of making the eye movements (corollary discharge or efference copy) is subtracted from the retinal signal, to cancel the motion produced by the eye movement and stabilize perception. Now we know that retinal motion signals cannot be easily compensated, given the sophisticated analysis performed by motion detectors. However, there is evidence for the existence of a corollary discharge signal that must be instrumental in maintaining visual stability (for detailed discussion of corollary discharge see chapter 65).

Considerable psychophysical evidence exists for a corollary discharge in humans, going back to the 1960s when Leonard Matin and others reported large transient changes in spatial localization at the time of saccades. When asked to report the position of a target flashed during a saccade, subjects mislocalized it, primarily in the direction of the saccade (Honda, 1991; Mateeff, 1978; Matin & Pearce, 1965). The localization error is typically in the order of half the saccadic size. Later, Mateeff and Honda measured the time course of this effect and showed that the error starts about 50 ms

before the saccadic onset and continues well after fixation is regained. The error before the saccadic onset has been taken as an indication of the existence of a slow and sluggish corollary discharge signal that compensates partly for the eye movement: The internal representation of the position and the actual position of the gaze do not match, resulting in errors in the localization of a briefly presented visual target.

We have examined saccadic mislocalization in photopic conditions using equiluminant stimuli (that remain visible during saccades). This approach revealed a bizarre result: At the time of saccades, visual space is not so much shifted in the direction of the saccade but *compressed* toward the saccadic target (Morrone, Ross, & Burr, 1997; Ross, Morrone, & Burr, 1997)—see figure 66.2A and figure 66.2C. Objects flashed at saccadic onset to a range of positions, from close to fixation to positions well beyond the saccadic target, are all perceived at or near the saccadic target. The effect is primarily parallel to the saccade direction (Ross, Morrone, & Burr, 1997) although a small compression is also observed in the orthogonal direction (Kaiser & Lappe, 2004). These results are intriguing because they indicate that the process described mathematically as a simple translation of the internal coordinate system is not plausible: Perhaps the system cannot perform the transformation of space without additional perceptual costs.

The perisaccadic compression of space is so strong that four bars, spread over 20°, are perceived as fused into a single bar (Ross, Morrone, & Burr, 1997). Discrimination of shape (Matsumiya & Uchikawa, 2001) or colors (Lappe et al., 2006) of the bars is still possible, but counting them and perceiving them in separate positions is not. Sometimes the shape or orientation of the flashed object can also change, appearing smaller and more vertical (for horizontal saccades), although these effects have been harder to quantify. The fact that the feature itself is not lost or compressed suggests that the mislocalization occurs at a relatively high level of analysis, after feature extraction.

It has been suggested that saccadic compression occurs only when visual references are present and is absent in the dark (Lappe, Awater, & Krekelberg, 2000). However, subsequent studies (Awater & Lappe, 2006) have shown that this is not necessarily true. In the dark, or in transient dark conditions achieved by a brief blackout immediately following the saccadic onset, compression does occur but can be obscured because in these condition there is also a mislocalization of the saccadic target (Morrone, Ma-Wyatt, & Ross, 2005). When this is taken into account, compression occurs in both light and dark, with and without visual references. Several studies have shown that visual references per se (like scattered points on the monitor) do not affect compression. However, presenting the same brief stimulus twice perisaccadically, even to different retinal locations, greatly reduces mislocalization (Morrone, Ross, & Burr, 1997; Park, Schlag-Rey, & Schlag, 2003; Pola, 2007; Zhang et al., 2004), and an even stronger reduction of the mislocalization occurs for a flash preceded by a prolonged, continuous or flickering stimulus (Sogo & Osaka, 2001; Watanabe et al., 2005). These findings suggest that the visual system has a mechanism for maintaining position constancy of objects across saccades. On the other hand, relative independence was found between clearly distinct stimuli, such as different shapes presented for different temporal intervals (Hamker, Zirnsak, & Lappe, 2008) or displaced orthogonally to the saccade (Morrone, Ross, & Burr, 1997; Sogo & Osaka, 2002).

Recently, we measured the interaction between stimuli pairs flashed asynchronously in the proximity of a saccade (Cicchini et al., in press). We found that the localization of a perisaccadic stimulus can be strongly affected by a similar pre- or postsaccadic stimulus, with the two perceived at similar positions even when actually separated by several degrees of visual angle. Figures 66.2B and D show the perceptual localization of probe bars presented at the same physical locations as in figure 66.2C, but followed (after 80 ms) by a similar "reference" bar flashed at screen center (0°): The pattern of probe mislocalization changed dramatically. Under these conditions probe positions closer to the reference bar were attracted by the reference; those closer to the saccadic target and those equidistant (like position 5) had a mixed attraction between the two visual signals. For probe bars presented around 0° (near the screen location of the reference bar) this resulted in virtually veridical localization across the time course. We tested multiple combinations of probe and reference locations and presentation times, and we found that the two bars interacted with each other and were seen together at the same position when they were displayed within a very broad acceptance range, spanning some 20° of horizontal space (the amplitude of the saccade) and some 200–300 ms of time. However, not all stimuli were found to interact: Bars of distinctly different orientation, or displaced orthogonally to saccade direction, have little or no effect on the probe, indicating that the phenomenon takes place late in the visual analysis after basic features have been processed. These two instances of spatial compression, usually toward saccadic target and here toward a postsaccadic reference, may have a common origin: The stronger visual reference (the saccadic target or the additional bar) attracts

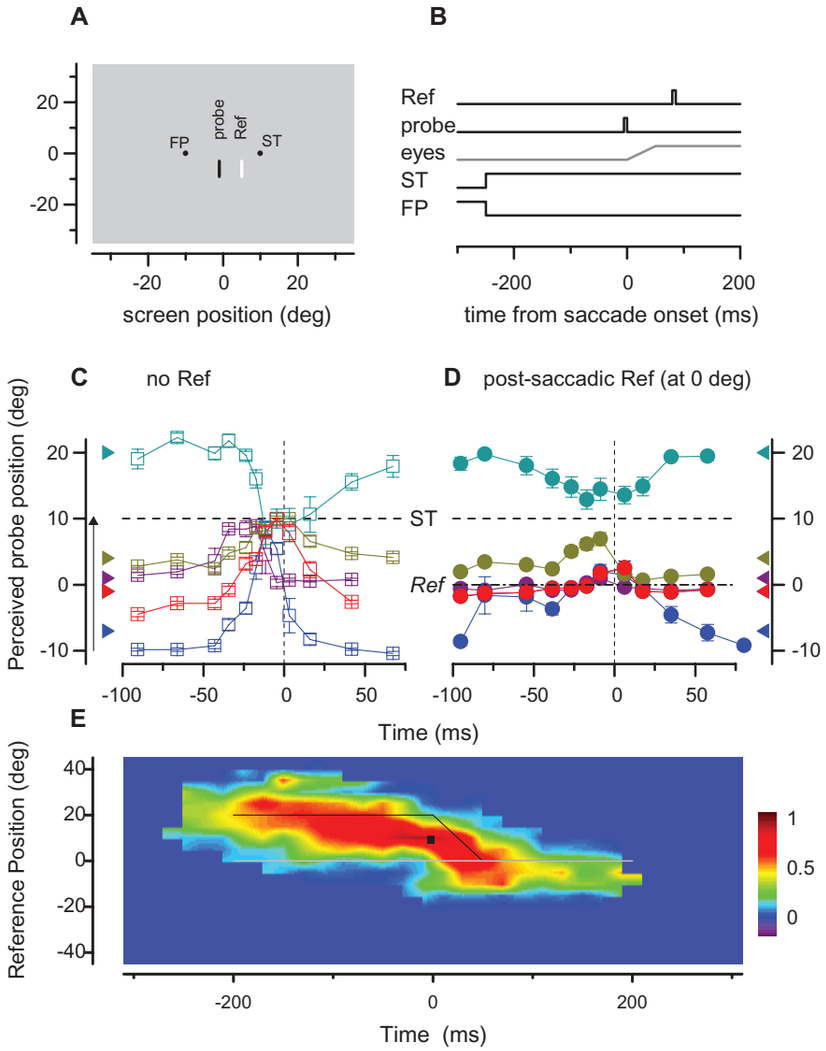

FIGURE 66.2 Effect of saccades on apparent bar position and number (reproduced from Cicchini et al., in press). (A) Perceived position of narrow light or black bars, briefly flashed on a red background at various times relative to the onset of a saccade from –10 to +10°. Stimulus display, with the fixation point (FP), the saccade target (ST), and the two flashed bars: reference (Ref) and probe. (B) time course of presentations. (C–D) Perisaccadic compression for a probe bar either flashed alone (C) or followed (80 ms) by another briefly flashed bar at screen center (D). The dashed-dot horizontal line shows the location of the reference stimulus. The black horizontal dashed line marks the location of the saccade target. The different curves refer to different screen positions of the probe (indicated by the color-matched triangles attached to the y-axes). The effect of the saccades (maximal at saccadic onset) is to shift the apparent position of the bar towards the saccadic target, where the eyes land in C (absence of reference bar) and for many bar positions towards the reference position in D. (E) Spatiotemporal map of interactions between a perisaccadic probe bar presented between –20 and 0 ms at screen position 0° (black symbol). The abscissa shows the time of the reference bars and the ordinate the horizontal position of the reference bar in retinal coordinates. The black square shows the location and timing of the probe. The gray and black lines show, respectively, the position of the fovea and of the saccade target. The interaction field extends over 20° of visual angle and over 300 ms across the saccade, but more importantly is elongated in space-time along the trajectory of spurious retinal motion.

the other brief stimuli presented perisaccadically and determines its localization.

Interestingly the spatiotemporal interaction field is quite extensive and, in retinal coordinates, slanted along the trajectory of spurious retinal motion. Figure 66.2E shows a sketch of this perisaccadic interaction that we suggest reflects the action of transiently altered neuronal receptive fields. Visual detectors with spatiotemporally oriented receptive fields are very common in the primate brain. They are fundamental for the computation of motion trajectories, particularly for perceiving the form of the moving object, which would

otherwise be subject to heavy motion smear (Burr, 1980). All models of motion perception, from Reichardt's (1957) classic proposal, involve nonlinear combination of systematically delayed signals, which generates a spatiotemporal orientation of receptive fields. A similar strategy may be used to stabilize visual images during saccades, as transiently oriented receptive fields could serve to effectively eliminate the spurious motion signals caused by the movement of the eyes. This profound alteration of neural receptive fields may be crucial to achieve perceptual stability. The perisaccadic extension in space and in time is so large that pre- and postsaccadic information can both activate the same detector, allowing for the integration of images from the two successive fixations. Importantly, only congruent information, concerning similar features, will take place.

But where in the brain do these transiently elongated receptive fields reside? Is there any evidence that a transient craniotopicity is actually implemented physiologically? Electrophysiological studies have reported several transient perisaccadic phenomena. In the LIP, receptive fields change spatial selectivity (Duhamel, Colby, & Goldberg, 1992) just before a monkey makes a saccadic eye movement, anticipating the change in gaze. This is illustrated in figure 66.3A, showing the response of an LIP cell to stimuli flashed within its usual receptive field position (hollow symbols) and within what will became the receptive field after the saccade has been made ("future receptive field," filled symbols). Note that the response in the current receptive field starts to decline, and that in the future receptive field to increase, long before the eye has actually moved to the new fixation. This is termed *predictive remapping*.

This phenomenon occurs not only in LIP but in many other visual areas, including superior colliculus (Walker, Fitzgibbon, & Goldberg, 1995) and area V3 (Nakamura & Colby, 2002), with area V4 showing somewhat different behavior (Tolias et al., 2001). It has even been suggested that a small fraction of neurons in primary visual cortex (V1) might have dynamic updating of receptive fields (Nakamura & Colby, 2002), and predictive remapping can be observed in human cortex using fMRI (Merriam, Genovese, & Colby, 2003, 2007). The origin of the phenomenon has been studied in the frontal eye fields (FEF), and firm evidence demonstrates that it is mediated by a corollary discharge signal, probably originating in the superior colliculus and mediodorsal nucleus of the thalamus (Sommer & Wurtz, 2002, 2006). Deactivation of this nucleus abolishes the predictive updating of the receptive field. The corollary discharge signal arrives nearly 100 ms before the updating starts in the FEF, indicating the complexity of the reorganization.

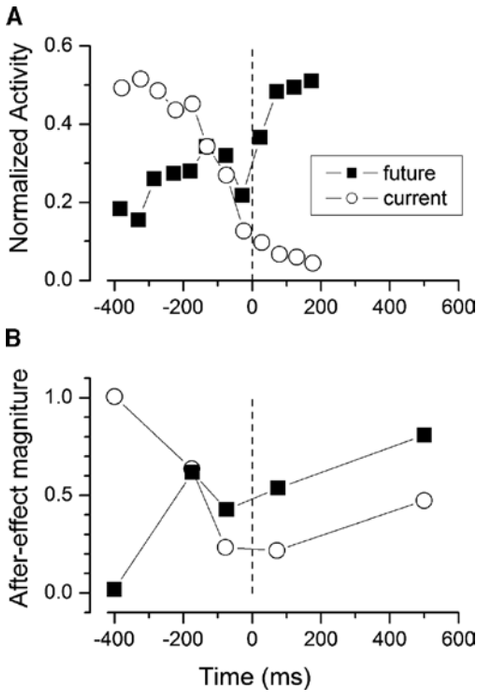

FIGURE 66.3 Predictive remapping in a lateral intraparietal (LIP) cell and human observers. (Reproduced with permission from Kusunoki & Goldberg, 2003; Melcher, 2007; Morrone & Burr, 2009.) (A) The response of a "remapping" cell of area LIP of the macaque around the time of the saccade, to brief stimuli displayed in the "current" (presaccadic) receptive field (open circles) and to stimuli flashed in what will become its receptive field after making the saccade. The response to stimuli in the current receptive field begins to decrease before the eyes actually move. Around the same time, the response in the "future" position begins to increase long before the eyes have actually displaced the receptive field. (B) An experiment showing analogous behavior in human psychophysics. Subjects adapted to a tilted grating, then measured the aftereffect to a grating presented in the same (retinal) position ("current": open circles) or to the position that will correspond to the retinal position of the adaptor after a saccade had been made ("future": filled squares). Long before the saccade, there is no adaptation in the future field and full adaptation in the current field (normalized to unity). Like the cell firing rate, adaptation effects in the current field begin to decrease, and those in the future field to increase, before the eyes have actually moved. Well after the saccade is terminated, the effects do not drop completely to zero, because this position corresponds to the spatiotopic position of the adaptor, and orientation adaptation has a spatiotopic component (Melcher, 2005).

In order to build a receptive field oriented along the saccadic direction, it is necessary to introduce delays in the visual processing that vary with position. Interestingly, there is evidence from many studies that early

responses at the "remapped" position are indeed delayed (Nakamura & Colby, 2002) and also possibly locked to the time of the saccade execution (Sommer & Wurtz, 2006; Wang, Zhang, & Goldberg, 2008).

Despite these recent efforts, there are several aspects of the remapping phenomenon that remain unclear. For example, between the time when the neuron starts to respond to stimuli in the remapped position and when, after the saccade, it regains its retinotopic specificity, are receptive fields anchored in a transiently craniotopic map as the psychophysical results of figure 66.2 suggest? Do the receptive fields undergo changes in size during the remapping?

The concept of a receptive field oriented in space-time, illustrated in figure 66.2E, provides a simple explanation of how predictive remapping can lead to perceptual stability. The initial presaccadic lobe of the elongated receptive field is consistent with the action of the remapping cells, being shifted in the direction of the saccade (*future receptive field*). However, this "predictive remapping" merely sets the stage for the receptive field to return to its rest position, and it is this return that is important for perceptual stability. The initial shift can be considered a "virtual saccade," before the actual eye movement, at a time when the regular retinotopic cells (which always coexist with predictive remapping cells: Duhamel, Colby, & Goldberg, 1992; Nakamura & Colby, 2002) still respond to a spatially stable visual region. The strategy is to anticipate the problem by shifting the receptive field of detectors in the direction of the saccade, thereby arming the receptive field to return to its resting position, as if loading a spring. The return in position that accompanies the eye movement causes the receptive field to become oriented in space-time, parallel to the spurious retinal motion induced by the saccade, and therefore effectively annulling it. We have no information yet on the dynamics of the return from the future to regular receptive field of visual neurons; no electrophysiological data are yet available. However, the simple concept of a transiently oriented space-time receptive field may not only elucidate the functional role of predicting remapping but also explain many perisaccadic perceptual phenomena, at first sight incongruent. The first is transsaccadic integration and transsaccadic masking.

Clever psychophysical studies also support the existence of transsaccadic integration in humans (Burr & Morrone, 2005; Melcher, 2005, 2007; Turi & Burr, 2012) by examining the spatial selectivity of visual aftereffects or input integration (Melcher & Morrone, 2003). Most aftereffects are spatially selective in retinotopic and/or in spatiotopic coordinates, and the effects can extend for as long as 3 s in time. The degree to which adaptation is spatiotopic varies with the complexity of the signals: Contrast sensitivities (thought to be mediated by primary visual cortex) are primarily retinotopic while more complex signals (like faces or motion coherence sensitivities) are primarily spatiotopic. Adaptation techniques with briefly presented probes (Melcher, 2007) revealed the dynamic of the predictive remapping consequence in perception. Long before the saccade, adaptation is maximal when test and adaptor are presented at the same position, at fixation, with very little adaptation at the position of saccadic target. However, when the test is presented perisaccadically, before and during the eye movement, the maximum adaptation occurs for tests near saccadic target, the position that will correspond to the adapted retina after the eyes have moved (see figure 66.3B). The similarity of the time courses of the adaptation and the response of the LIP neuron (see figure 66.3A) strongly suggests that the brain utilizes the period in which the receptive field regains postsaccadic retinotopic specificity to bridge the perception between the two fixations. That the integration receptive field extends over a large area (see figure 66.2E) is also supported by the recent finding by Zirnsak et al. (2011), who repeated the experiment of Melcher for stimuli positioned in the future receptive field. Interestingly, TMS on human FEF (whose neurons show prominent predictive remapping) reduces transsaccadic memory (Prime, Vesia, & Crawford, 2011) when delivered close to saccadic onset, probably interfering with the organization of the transsaccadic receptive fields.

Recent studies (De Pisapia, Kaunitz, & Melcher, 2010; Hunt & Cavanagh, 2011) show that making saccadic eye movements can actually enhance (rather than degrade) the visibility of a brief perisaccadic stimulus. They presented a brief visual target, followed at various intervals by a "mask," which impedes recognition of the test by "backward masking." The most interesting condition was when test and mask were separated by a brief (12-ms) interval, both presented to stationary eyes, at the same retinal position. When presented 20–30 ms before saccadic onset, visibility of the test improved considerably, particularly for trials where it was perceived as displaced. The results imply that the perisaccadic mislocalization of the test shifts it away from the mask, effectively *demasking* it. In another condition, they used a long test–mask separation with the test and mask straddling the saccade, therefore stimulating distinctly different retinal positions: Yet the masking was strong, suggesting that the representation had been transferred to a spatiotopically corresponding position. Both effects can be simulated by applying filters of the kind illustrated in figure 66.2E.

A well-known perisaccadic phenomenon is that if the saccadic target is displaced after the saccade has been initiated, the displacement (of up to 30% saccade size) is not noted (Bridgeman, Hendry, & Stark, 1975). However, if there is a brief gap in the reappearance of the target in the displaced position, the displacement is immediately apparent (Deubel, Schneider, & Bridgeman, 1996). This observation led to the idea that the system assumes object stability in the absence of contrary information, probably by comparing pre- and postsaccadic positions with some form of short-term memory buffer. These results suggest that the visual system takes advantage of static visual references to help maintain stability across saccades, but the details of how these are selected and stored in some form of memory buffer of limited capacity has been elusive. It is possible that the effect may be mediated by predictive remapping, given that the displacement suppression thresholds are altered by TMS delivered in the perisaccadic epoch over the posterior parietal cortex (Chang & Ro, 2007).

It has recently been argued that the insensitivity to saccadic target displacement (Bridgeman, Hendry, & Stark, 1975) may be explained by optimal sensorimotor integration between the retinal signal and extraretinal corollary discharge signals (Niemeier, Crawford, & Tweed, 2003). At the time of saccades, spatial information about eye position, which is necessary to localize objects in external space, is unreliable. Therefore spatial information during this period is given less weight than information before and after the saccade. The transient distortions of the kind shown in figure 66.2C may also be consistent with statistically optimal, or "Bayesian," integration of information. A recent study has shown how this may be the case by examining audiovisual integration during saccades. Auditory stimuli are usually far more difficult to localize in space than visual stimuli: When vision and sound are in conflict, vision dominates (the "ventriloquist effect") as predicted by optimal integration. However, when visual stimuli are artificially degraded by blurring, audition can dominate (Alais & Burr, 2004), again consistent with optimal integration. As saccades have little effect on auditory space perception (Harris & Lieberman, 1996), they are a useful tool to study saccadic mislocalization. Indeed audiovisual stimuli (bars and beeps presented together in the same spatial position) are mislocalized much less than visual stimuli presented alone, suggesting that visual information is given a low weight during saccades, and this can lead to

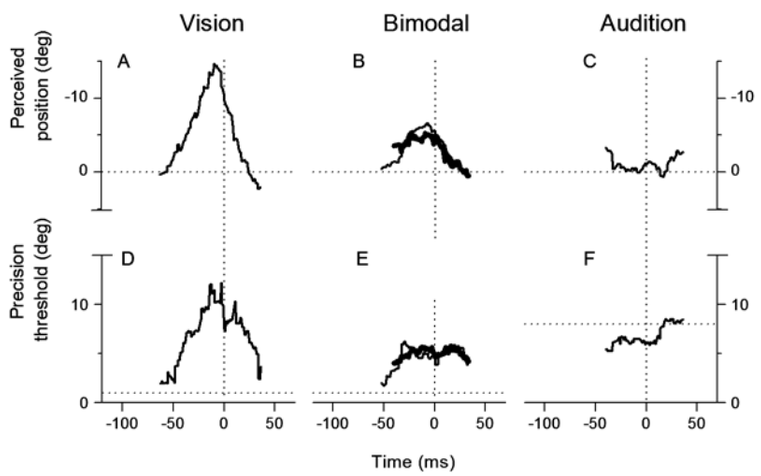

FIGURE 66.4 Illustration of how saccadic mislocalization may result from optimal "Bayesian" fusion. (Reproduced with permission from Binda et al., 2007.) In a two-alternative forced-choice procedure, subjects were asked to report whether a perisaccadic test bar displayed midway between fixation and saccadic target seemed to be located right or left of a presaccadic probe bar (for full details see Binda et al., 2007). Psychometric functions were fitted to these data, to give an estimate of perceived position and also of precision of localization. The upper curves show how perceived position varied with time (relative to saccadic onset). Visual stimuli presented on their own (A) showed the characteristic mislocalization, like that of figure 66.1A. Auditory stimuli, however, were not at all affected by the saccade (C). However, when the sound was played contemporaneously with the bar display, the mislocalization of the bar was reduced (B). The lower curves show the localization thresholds. Again, sound was unaffected by saccades, but the precision of visual localization reduced drastically near saccadic onset. During the bimodal audiovisual presentation, precision improved and was better than either the visual or auditory unimodal localization precision. Indeed this performance, both for perceived position and for precision thresholds, was very close to the Bayesian prediction, indicated by the thick gray line. The dotted horizontal lines indicate performance during fixation.

K2

mislocalization of transient stimuli (Binda et al., 2007). Not only does the idea explain qualitatively the mislocalization, it explains quantitatively the mislocalization of bimodal audiovisual stimuli over the whole time course relative to saccade onset (see figure 66.4).

Binda et al. (2007) go on to develop a Bayesian model of saccadic mislocalization, simply assuming, like Niemeier, Crawford, and Tweed (2003), an increase in noisiness of the eye-position signal at the time of saccades. However this kind of model accounts for only the shift in the direction of the saccade, not the accompanying compression. This would require a further assumption, such as a "prior" or "default rule" for objects to be seen at the fovea. Cicchini (in press) advanced the idea of the transient receptive field of the kind illustrated in figure 66.2E, which transiently merges stimuli over an oriented spatiotemporal profile, effectively *implementing* a prior for spatial constancy across saccades.

It is interesting that saccadic compression is positively correlated with peak saccadic velocity (Ostendorf et al., 2007): Individuals with high saccadic velocity show large compression while subjects with slow saccadic velocity show mainly a shift in the saccadic direction (but the effect is not related to the spurious visual motion). This suggests a strong link between perception at the time of saccades and the motor system, probably mediated by the corollary discharge signal (see chapter 65). The receptive field of figure 66.2E is aligned with the saccadic trajectory and may well vary in extension and orientation depending on peak saccadic velocities, explaining this surprising correlation.

Although saccades cause dramatic perceptual localization illusions, when subjects are required to indicate their response by a motor action—secondary saccades or blind hammering—their responses are near veridical (Hallett & Lightstone, 1976a, 1976b; Hansen & Skavenski, 1977, 1985). Other studies (e.g., Bridgeman et al., 1979) 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. However, a few experiments have failed to replicate the original dissociation between motor accuracy and perceptual error during saccades, reporting localization errors for both tasks (Bockisch & Miller, 1999; Dassonville, Schlag, & Schlag-Rey, 1992, 1995; Honda, 1991; Miller, 1996; Schlag & Schlag-Rey, 1995). Burr, Morrone, and Ross (2001) and Morrone, Ma-Wyatt, and Ross (2005) reported a clear dissociation between verbal reports and blind pointing for saccadic compression. The plot of figure 66.5 shows that briefly flashed stimuli were perceived clearly in false positions, causing the characteristic compression (filled symbols); but when asked to point blindly at the stimuli, with the screen temporally obscured by liquid crystal shutter, observers did so veridically (open symbols).

Interestingly, analogous effects have been reported in audition. Although saccadic eye movements do not affect the localization of tones, saccadic head movements do (Leung, Alais, & Carlile, 2008). Sounds are

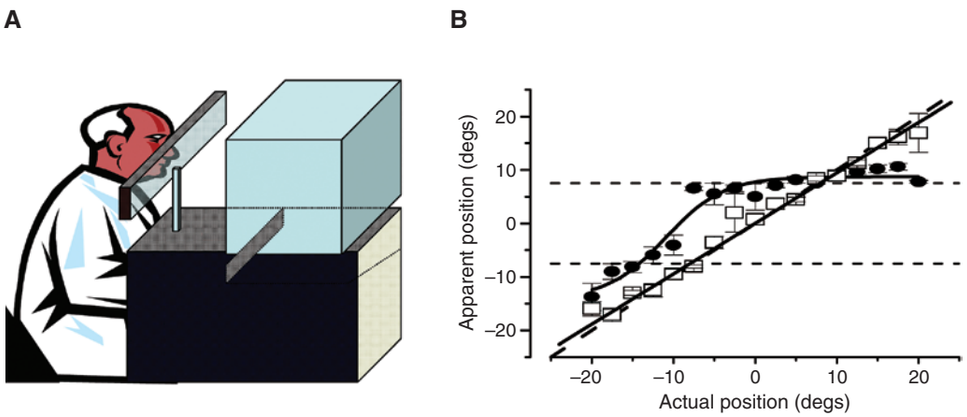

FIGURE 66.5 No spatial compression occurs for rapid motor responses. (Reproduced with permission from Burr, Morrone, & Ross, 2001.) (A) Subjects viewed a CRT monitor through a liquid crystal shutter. On command they made a 15° saccade from –7.5 to +7.5° (dashed lines in B), and a bar was briefly displayed just prior to saccadic onset. Shortly after the saccade was completed, the shutter closed and subjects responded by jabbing at the touch screen with a brisk ballistic movement, the hand hidden from view. (B) The open squares show the results for the jabbing response, for stimuli presented just prior to saccadic onset ($-30 < t < 0$ ms). The responses are near veridical. The filled circles show results for verbal reports under identical conditions. As shown in figure 66.2, there is a very strong compression, with all stimuli within 10° (degs, degrees) of the saccadic target seen at saccadic target.

compressed toward the end point of the head turn. However, if subjects are ask to point to the apparent sound source (by head turn), the compression disappears, as it does for vision (Burr, Morrone, & Ross, 2001).

However, for visual judgments, introducing clearly visible postsaccadic references under normal lighting conditions causes both verbal report and pointing to show compression. This suggests that vision has access to two maps, one subject to distortion where the receptive fields transiently change form and the other not: The motor map shows no compression except when visual references remain in view for a substantial time after saccade, indicating that these maps are updated postsaccadically, while for perceptual judgments the updating occurs before and during the actual saccade. Both maps contribute to determining the weight given to each map. Perhaps the popular distinction between conscious perception and action (Goodale & Milner, 1992; Trevarthen, 1968) is at best an oversimplification.

Space and time are generally studied separately and thought of as separate and independent dimensions. However, we have observed that not only space but also time undergoes severe transient distortions at the time of saccades: Objects become compressed toward the saccadic target (Ross, Morrone, & Burr, 1997), and perceived temporal durations are severely shrunk (Morrone, Ross, & Burr, 2005). When asked to compare the perceived duration of a temporal interval presented around the time of a saccade with one presented 2 s afterwards, subjects judged it much shorter, about half the duration (see figure 66.6B). Interestingly, the precision of the judgment is higher perisaccadically than at fixation, obeying the general rule of constant Weber fraction (Morrone, Ross, & Burr, 2005). Again the time course of the temporal distortion is quite tight and, after taking into account the duration of the stimuli and the effect of contraction, similar to that of the spatial compression. As the transient changes both in space and in time follow very similar dynamics (compare figure 66.2C with figure 66.6B—continuous curve), they may well be manifestations of a common neural cause, possibly a distortion in the space-time metric induced by the transient orientation of the neuronal receptive field as shown in figure 66.2E.

Alteration of the sense of time can also be more dramatic: Saccades can even cause an inversion of the perceived temporal sequence (Morrone, Ross, & Burr, 2005), anticipating or delaying temporal process.

Binda et al. (2009) showed that the perceived time at saccadic onset (measured by matching an auditory tone) is delayed by about 100 ms, while 50 ms before saccade the latency is reduced by only 20 ms, a small

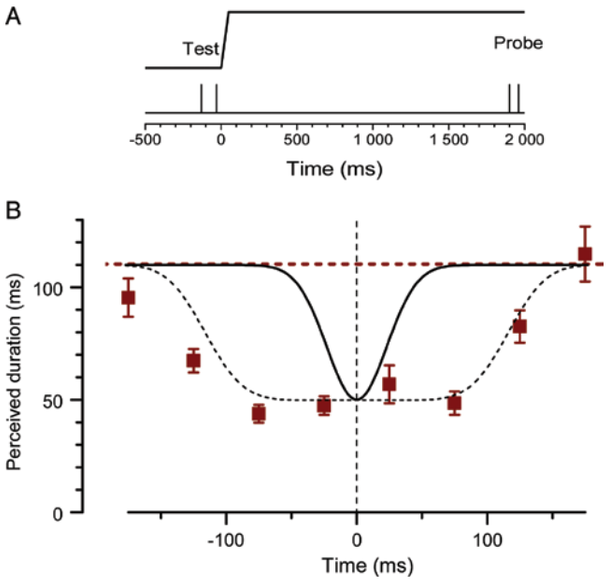

FIGURE 66.6 Time is also compressed during saccades. (Reproduced with permission from Burr et al., 2010.) (A) The subject was asked to compare the duration of the interval of two test flashes (separated by 100 ms) with a postsaccadic probe of variable duration that appeared 2 s later. (B) The apparent duration was then calculated from psychometric functions. Around the time of saccadic onset, apparent duration was about half the physical duration. The dashed line shows the duration match during fixation. Note that the predicted time course is much more tightly tuned than the data (continuous curve), because the data were collected with a broad temporal stimulus (100 ms long) that necessarily blurs the effects over time. A good approximation is obtained by deconvolving the dashed curves with a temporal interval given by the physical stimulus separation and a broad temporal impulse response such as that illustrated in figure 66.2E.

effect, but one sufficient to produce an inversion (Morrone, Ross, & Burr, 2005). This result is consistent with the fact that during remapping the latencies of neurons in areas MT and MST are shorter in response to real than simulated saccades (Price et al., 2005). On the other hand, the postsaccadic delay of the visual stimulus is consistent with the delay of the response of the future receptive field observed by Nakamura and Colby (2002) and Wang, Zhang, and Goldberg (2008). In addition, the delay also indicates that stimuli presented at saccadic onset are coincident with stimuli presented soon after saccades, facilitating the interpretation of their position in the postsaccadic coordinate system and decoding after that the saccade is complete, in a form of *postdiction* (Eagleman & Sejnowski, 2000). Again this is consistent with the long temporal extension of the perisaccadic receptive field (figure 66.2C), which extends well after the saccade is completed.

CONCLUSION

Seeing is usually believing: For about two thirds of our waking lives, we perceive objects where vision tells us they are—which, more often than not, coincides with their actual position. In the remaining time, the visual system sends us erroneous spatial information, presumably because it is engaged in correcting the troublesome consequences of eye movements on retinal afferences. When this happens, we disbelieve visual information; if available, spatial cues from other senses become dominant; if we have to act, we use the robust postsaccadic representation without attempting to update it transsaccadically. If vision is the only signal available, we deform our concept of space, of time and numerosity to make sense of it and do not miss visual information for more than one third of our waking time.

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