

Letter to the Editor

A simple translation in cortical log-coordinates may account for the pattern of saccadic localization errors

Rufin VanRullen

CNRS, UPS, Centre de Recherche Cerveau et Cognition, 133 Rte de Narbonne, 31062 Toulouse Cedex, France

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Abstract. During saccadic eye movements, the visual world shifts rapidly across the retina. Perceptual continuity is thought to be maintained by active neural mechanisms that compensate for this displacement, bringing the presaccadic scene into a postsaccadic reference frame. Because of this active mechanism, objects appearing briefly around the time of the saccade are perceived at erroneous locations, a phenomenon called perisaccadic mislocalization. The position and direction of localization errors can inform us about the different reference frames involved. It has been found, for example, that errors are not simply made in the direction of the saccade but directed toward the saccade target, indicating that the compensatory mechanism involves spatial compression rather than translation. A recent study confirmed that localization errors also occur in the direction orthogonal to saccade direction, but only for eccentricities far from the fovea, beyond the saccade target. This spatially specific pattern of distortion cannot be explained by a simple compression of space around the saccade target. Here I show that a change of reference frames (i.e., translation) in cortical (logarithmic) coordinates, taking into account the cortical magnification factor, can accurately predict these spatial patterns of mislocalization. The flashed object projects onto the cortex in presaccadic (fovea-centered) coordinates but is perceived in postsaccadic (target-centered) coordinates.

1 Introduction

The contents of a visual neuron's receptive field are renewed after each eye movement, almost three times per second. It would be very costly to start processing afresh at each fixation. Rather, prior information about the content of other neurons' receptive fields, and about the direction and amplitude of the intended saccade, can help neurons predict what will enter their receptive fields even before the saccade occurs. For this, a drastic transformation in

the population representation of visual space must take place. Neural correlates of such a transformation have been observed experimentally, in the form of a neuronal receptive field shift prior to saccade onset in various brain areas (Walker et al. 1995; Duhamel et al. 1992; Tolia et al. 2001; Nakamura and Colby 2002; Kregelberg et al. 2003). One of the drawbacks of this remapping strategy is that unexpected events happening during the transformation will be wrongly assigned to spatial locations (Matin and Pearce 1965; Honda 1989, 1993; Schlag and Schlag-Rey 1995; Ross et al. 2001). This rarely happens in real life but can be used in the laboratory to investigate the underlying neural mechanisms.

It has been found that flashes appearing around saccade onset, whether closer to fixation or further away than the saccade target, are systematically mislocalized toward the saccade target (Ross et al. 1997; Morrone et al. 1997; Lappe et al. 2000; see, however, Miller 1996 for an opposing viewpoint). This compression of visual space around the saccade target is somehow unexpected: indeed, saccades result in a uniform translation of visual space across the retina, and thus it would seem that the optimal strategy to compensate for such changes should involve translation rather than compression. Even more surprising is a recent observation by Kaiser and Lappe (2004). Investigating perisaccadic localization errors not only along the saccade axis but also orthogonal to it, these authors found that the spatial pattern of distortion is highly anisotropic. Uniform translation of perceived locations (with little mislocalization in the direction orthogonal to the saccade) seems to occur for flashes close to fixation, while compression (with large mislocalization in the orthogonal direction) tends to occur for flashes appearing further away and beyond the saccade target (Fig. 1c). Current models of perisaccadic transformation involve a combination of two processes: a translation of the reference point and a compression of space around the fovea (Morrone et al. 1997; Ross et al. 1997). While the translation component can be easily explained in terms of a compensation of retinal displacement (e.g., using an efference copy of the eye movement), the compression component remains virtually unexplained (Ross et al. 1997). Further, the more

Correspondence to: R. VanRullen
(e-mail: rufin.vanrullen@cerco.ups-tlse.fr,
Tel.: +33-562-172839, Fax: +33-562-172809)

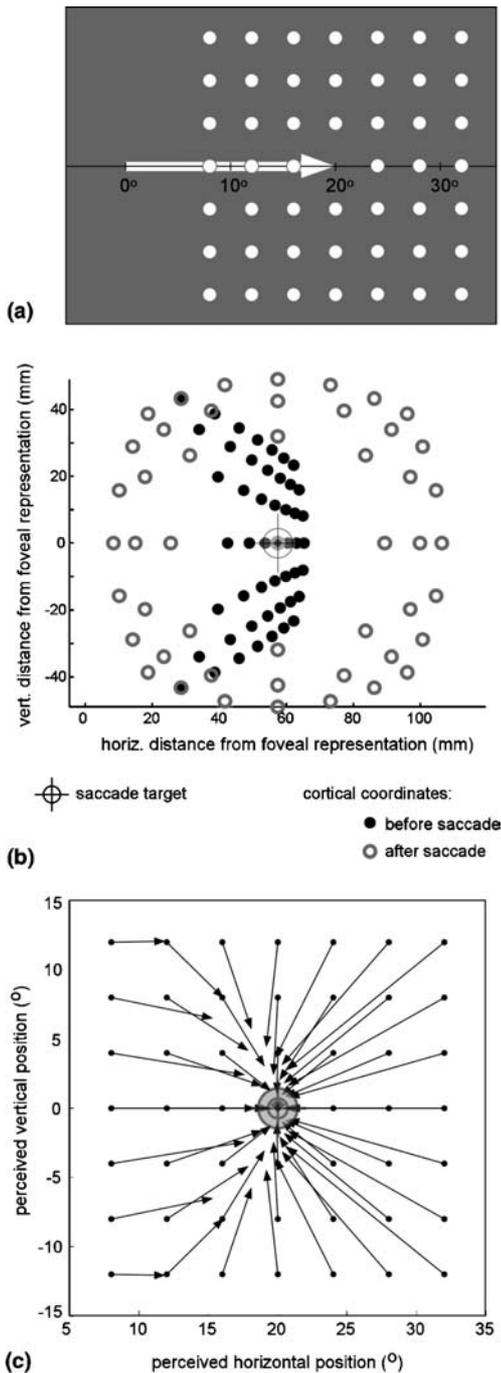


Fig. 1. Pattern of perisaccadic mislocalization for a 20° rightward saccade. In the simulations, 7 × 7 grid points (a) represent the possible locations of dots flashed at the time of saccade onset. Because of the cortical magnification factor, these grid points mapped onto cortex in a logarithmic fashion (filled circles in b). During the saccade, a simple uniform shift of reference frame was assumed to take place, making the saccade target the origin of the new logarithmic coordinate system. The open gray circles in b represent the cortical positions that would have been occupied by the grid points in this new reference frame. When the actual cortical locations of the points are mapped back onto the visual world according to the new logarithmic coordinate system, spatial distortion occurs (c). As in Kaiser and Lappe (2004), this distortion resembles translation for points closer to the fovea and compression for points at higher eccentricity. Each arrow indicates the amount and direction of spatial mislocalization of one of the 49 grid points (marked by the circle at the base of the arrow)

recent results of Kaiser and Lappe (2004) could only be explained in this framework by assuming that the two components act with different strengths in different parts of the visual field (and/or at different times; Kaiser and Lappe 2004).

Here I show that the two distortion phenomena (translation, compression) as well as their spatially specific behavior, can be predicted by a simple model assuming a uniform translation of spatial representations during saccades. The translation, however, occurs in cortical space rather than in visual space. Spatial locations, due to the cortical magnification factor, are mapped onto cortex in logarithmic coordinates. Locations farther from fixation are thus more strongly affected by the magnification factor than others: they tend to map closer together, around the representation of the saccade target. When the saccade occurs, making the saccade target the reference point of the new logarithmic coordinate system, these points will thus be misperceived as located much closer to the saccade target than they actually are – hence the compression phenomenon. The model is fully constrained by experimental observations and has no free parameter. Yet it produces both apparent translation (for small eccentricities) and compression (for larger eccentricities) and is able to replicate in a qualitative manner the findings of Kaiser and Lappe (2004).

2 Cortical magnification and logarithmic mapping

Most of today's digital cameras achieve a resolution of several mega pixels, allowing their owners to take crisp pictures of large scenes and panoramas. Our retinas, however, must sample the world with only about a million ganglion cells (Curcio and Allen 1990). The visual system counteracts this limitation by concentrating higher resolution on the fovea and gradually lower resolution toward the periphery. For the price of an eye movement, we can thus sample any object in the world with sharp resolution. This is why we find ourselves moving our eyes around many times per second.

The topographic organization of neurons on the cortical surface reflects this strategy. A given object in the world activates a much larger cortical surface (i.e., many more neurons) when it is in the fovea than in the periphery. Distances between objects in the world are not simply (or bijectively) related to distances between their representations on the cortical surface because their retinal eccentricity must also be taken into account. The magnification factor M at eccentricity e is the cortical distance (in millimeters) corresponding to 1° of visual angle. It is defined as:

$$M(e) = \frac{A}{e + e_2}.$$

Here, A and e_2 are constants such that A/e_2 is the cortical magnification at the center of the fovea (when $e=0$), and e_2 represents the retinal eccentricity at which cortical magnification will be half that of the fovea. Although the above form is most frequently used when referring to the cortical magnification factor, one can also estimate the cortical

eccentricity E_c (measured in millimeters from foveal representation) of a point at visual eccentricity e (in degrees), as the integral:

$$E_c(e) = \int_0^e M(x) dx = A \ln\left(1 + \frac{e}{e_2}\right). \quad (1)$$

This equation indicates that the visual world is mapped onto cortex into logarithmic coordinates. A flying insect moving at a constant speed across our retina and out of sight would be represented on the cortical surface by a wave of neuronal activation progressing more and more slowly. In contrast, constant movement on the cortical surface (away from the foveal representation) could only signify that the corresponding object in the visual world is picking up speed exponentially.

3 Methods

For the following simulations the values $A = 17.3$ mm and $e_2 = 0.75^\circ$ were used for (1), as estimated by Horton and Hoyt (1991) for primary visual cortex (V1). Hence the model was fully constrained and had no free parameter. Grid points, representing the possible locations of a perisaccadic flash (Fig. 1a), were projected onto cortex according to (1), with their eccentricity expressed with respect to the fovea (i.e., in presaccadic coordinates). The perisaccadic transformation (a uniform translation of foveal representation toward saccade target or, equivalently, a uniform translation of neuronal activities from the saccade target toward foveal representation) was then assumed to take place, with the result that the obtained grid point projections were now expressed with respect to the saccade target. In other words, the cortical coordinates of the saccade target were subtracted from all grid point cortical coordinates (i.e., a simple translation). This yielded the postsaccadic coordinates (Fig. 1b). The perceived location of the points was then calculated by inverting (1):

$$E(d) = e_2(\exp(d/A) - 1), \quad (2)$$

where d represents cortical distance from fovea/saccade target representation (millimeters, now in postsaccadic coordinates) and $E(d)$ expresses the perceived eccentricity (in degrees of visual angle). The distortion of perceived position, induced by the mismatch between the pre- and postsaccadic logarithmic coordinate systems, was then measured for all grid points.

Note that all points, whether in cortical or external coordinates, are always defined not only by an eccentricity but also by an angle from horizontal. However, the angle is unaffected by the cortical magnification factor and thus did not enter into the previous calculations.

Experimental data suggest that perisaccadic mislocalization follows a particular time course over approximately 100 ms, being maximal immediately before the saccade. Here the transform is assumed to be instantaneous and thus would only correspond to the maximum

mislocalization observed experimentally. Further mechanisms, such as a temporal “envelope”, would be needed to account for the temporal aspects of perisaccadic transformation.

4 Results

As in Kaiser and Lappe (2004), the effects of perisaccadic mislocalization were investigated for different saccade amplitudes and at different locations in the visual field (including locations with a component orthogonal to saccade direction, i.e., not along the saccade axis). The possible flash positions formed a 7×7 grid of unit size 4° , spanning horizontal retinal locations of 8° to 32° and vertical locations of -12° to 12° (Fig. 1a). Horizontal saccades of amplitudes 12° , 16° , 20° , 24° , and 28° were simulated. Note that other saccade directions (e.g., vertical) would yield comparable results, due to the radially uniform nature of the model, and thus were not simulated. Immediately before the saccade, the flashed grid points were assumed to project onto cortex in a fovea-centered (pre-saccadic) logarithmic reference frame. This is illustrated in Fig. 1b. During the saccade, a translation of cortical coordinates was assumed to take place from fixation to saccade target. The new logarithmic reference frame was thus centered on the saccade target, with highest resolution at that location (Fig. 1b). The flashed grid points were assigned to locations in the visual field according to this new coordinate system. Mislocalization thus occurred because of the mismatch between the pre- and postsaccadic logarithmic coordinate systems.

The spatial pattern of mislocalization for a 20° saccade is illustrated in Fig. 1c. As observed by Kaiser and Lappe (2004), it is obvious that distortion is not symmetrical around the saccade target. At smaller eccentricities, translation of perceived position in the saccade direction is prominent. This pattern changes gradually with eccentricity, compression becoming more and more apparent and almost exclusive for positions beyond the saccade target.

A comparable pattern was obtained for varying saccade amplitudes between 12° and 28° (Fig. 2). It appears as if mislocalization mostly reflects translation for eccentricities smaller than that of the saccade target, and compression for larger eccentricities. In fact, as noted by Kaiser and Lappe (2004), the saccade target does not act as an absolute landmark in that respect. The amount of vertical mislocalization toward saccade target (an indicator of the strength of compression toward saccade target) actually increases with eccentricity, almost independently of saccade amplitude (Fig. 3). Note that the absolute amounts of vertical mislocalization in Fig. 3 are roughly twice as high as those observed by Kaiser and Lappe (2004). In other words, in this case the model only reproduces their observation in a qualitative manner. This might be due to the fact that cortical translation is implemented as an instantaneous and noise-free process in the current model, whereas it is most certainly gradual, and possibly noisy, in the visual system. This and other shortcomings of the model are discussed in the next section.

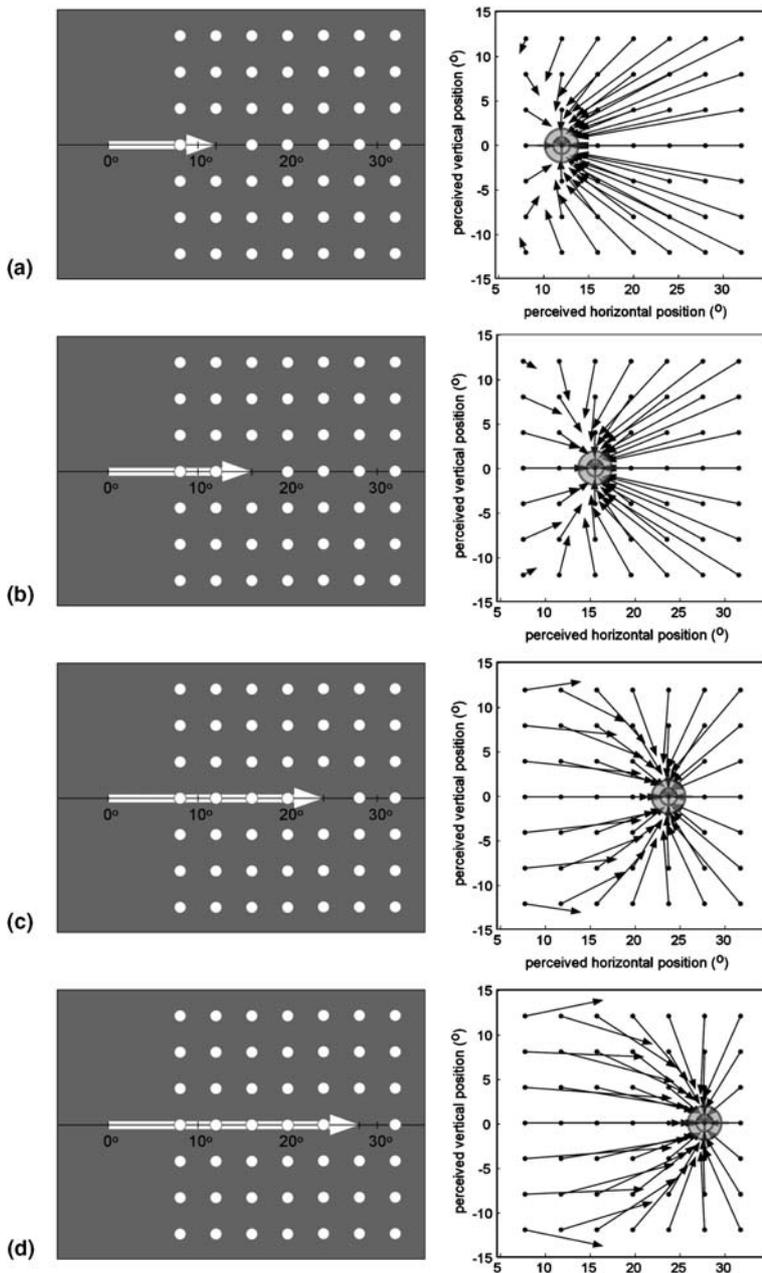


Fig. 2a–d. Spatial patterns of mislocalization for horizontal saccades of amplitude 12° (a), 16° (b), 24° (c), and 28° (d). In all cases, compression appears to be highest for locations beyond the saccade target, while uniform translation in the direction of the saccade is observed for smaller eccentricities

5 Discussion

It has long been presumed that the effects of eye movements on perceived position should be compensated in the visual system by a uniform translation of spatial representations using an “efference copy” of the saccade (von Helmholtz 1866; von Holst and Mittelstaedt 1950; Sperry 1950). Recent experimental evidence has challenged these simple views by showing complex, nonuniform distortions of visual space at the time of saccade, which depended on retinal eccentricity and position with respect to the saccade target. Many of these seemingly contradictory observations, however, can in fact be accounted for by a simple uniform translation, if it is assumed to occur in cortical rather than physical space. The present idea is not entirely incompatible with previous models of perisaccadic dis-

ortion (e.g., Ross et al. 1997). These authors noted that the compression term in their model was reminiscent of (and potentially related to) the inverse function of the cortical magnification factor. The present model, however, presents the first functional explanation of the relation between perisaccadic distortions and the cortical magnification factor.

Even though the model can qualitatively explain numerous experimental results, it is very important to note that its operation is restricted to an area spanning the fovea, the saccade target, and beyond (in the saccade direction). Indeed, expansion of perceived space is predicted by the model for locations whose cortical eccentricity in the postsaccadic reference frame is larger than that of the presaccadic fixation point. In the case of a horizontal saccade, this would thus happen for all points in

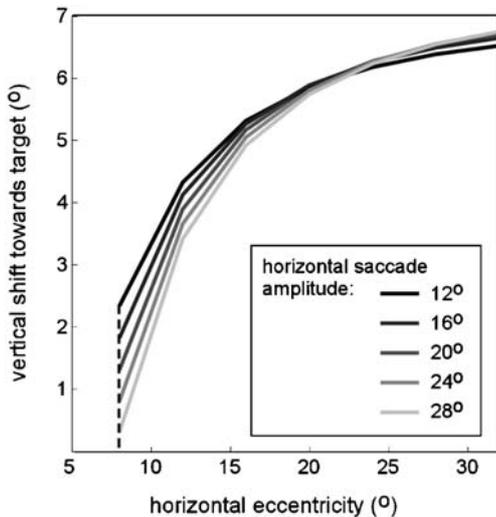


Fig. 3. Amplitude of vertical shift toward target as a function of horizontal eccentricity of the flashed point, for various saccade amplitudes. The vertical shift reflects the strength of compression toward the saccade target. Indeed, in the case of pure translation in the saccade direction (i.e., horizontal), vertical mislocalization should be zero. Here, as in Kaiser and Lappe (2004), vertical mislocalization increases with eccentricity, almost independently of saccade amplitude. In other words, the ratio between apparent translation and apparent compression depends mostly on the eccentricity of the flashed point and very little on its position with respect to the saccade target

the hemifield opposite the saccade target. One possible, cheap-to-implement solution to this problem would be to limit the proposed active saccade compensation mechanism to the cortical hemisphere containing the saccade target. This would not be sufficient, however, to explain a range of psychophysical observations. For example, Ross et al. (1997) and Morrone et al. (1997) found that points on the left of fixation (i.e., in the hemifield opposite saccade direction) were also displaced to the right when rightward saccades were made. Further mechanisms would be needed to account for these effects, and some solutions (e.g., localized gain control) are alluded to later.

What could be the neural substrate of the present model? The most obvious mechanism to underlie this saccade compensation would be an actual migration of neuronal responses from the saccade target to the fovea, thereby anticipating the postsaccadic reference frame. This “Cartesian” migration in a logarithmic space would easily account for the distortion results presented here. This translation could be driven, for example, by an efference copy of the planned eye movement. In practice, this transformation could be implemented by a transient spatial remapping of receptive fields (Duhamel et al. 1992). Dynamic changes in the shape of neuronal receptive fields, compatible with this idea, have been observed in numerous brain areas before saccade onset (Walker et al. 1995; Duhamel et al. 1992; Tolia et al. 2001; Krekelberg et al. 2003): receptive fields generally appear shifted toward the retinal location that they will occupy after the saccade. This qualitative observation alone is not enough, however, to validate the present model. What the model predicts is

that the spatial distribution of these receptive field changes will resemble a uniform translation in the direction of the saccade, when it is measured *in cortical space* but not in visual space. The pattern of receptive field shifts in visual (i.e., external) space should in fact be heavily distorted compared to a simple translation – this distortion being the inverse of that illustrated in Figs. 1c and 2. Single-unit electrophysiology or, more directly, optical imaging of the cortical surface may allow these predictions to be tested in the near future.

Because the circuitry underlying receptive field remapping is unknown, it is not easy to predict how the visual system could be implementing this transformation at the network level. It is important to point out, in any case, that this is by no means a simple operation.

Further neural mechanisms would also need to be added to this model, to accommodate for distortions in other parts of the visual field (in particular, to compensate for the predicted expansion of distal locations, as described above). Among them, a spatially specific gain control (or, as detailed later, a form of attentional modulation) could effectively limit the perisaccadic transformation to locations immediately surrounding the fovea and saccade target. Such an additional mechanism could also explain the quantitative differences between vertical localization errors obtained in the present model (Fig. 3) and those observed by Kaiser and Lappe (2004).

Although for simplicity the parameters used here were derived for primary visual cortex (Horton and Hoyt 1991), the same model could easily apply to other cortical areas, using different sets of parameters for (1) and (2) (Dougherty et al. 2003). The only requirement is that the areas involved should be retinotopically organized. No assumption is made here as to what actual cortical site(s) could be responsible for the phenomenon. In fact, electrophysiological investigations have failed to reveal neural correlates of a perisaccadic transformation in V1 neurons (Nakamura and Colby 2002). Strong remapping is observed, however, in various areas such as V3A, MST, or LIP (Nakamura and Colby 2002; Krekelberg et al. 2003; Duhamel et al. 1992). Because the exact circuitry underlying these remappings is not known, it is difficult to decide whether the shift in receptive fields observed in hierarchically higher areas arises as a simple consequence of the corresponding transformation taking place in lower areas or whether higher areas are the true site of this remapping, which is then fed back to lower areas.

One could wonder why the visual system would care to implement a compensation mechanism that is far from optimal, yielding gross spatial distortions in many cases. To answer this, one has to consider the alternative: in order to take into account the change in logarithmic coordinates that is induced by the cortical magnification factor, the visual system would need to implement a different compensation mechanism for each possible saccade target location in the visual field. The circuitry involved in such a strategy would probably turn out to be too costly compared to what can be gained by the compensation mechanism itself. On the other hand, an “approximate” compensation such as the one described

here, shifting information across the cortical surface in a manner independent of the actual location of the saccade target, could still provide important gains (i.e., information about the presaccadic world) while remaining fairly cheap in its implementation. It is also important to remember that distortions of visual space only occur for objects that appear briefly around the time of saccade onset. Under normal viewing conditions, when the world remains stable throughout the saccade, perisaccadic spatial remapping is undoubtedly a very successful process. It is possible that in the general case, presaccadic reference points in the environment are used to correctly reassign postsaccadic locations. The effects of the shift in logarithmic coordinates that I described here would thus go unnoticed. Such a presaccadic reference would be missing in the case of a flashed object, and only its postsaccadic cortical projection could be used to (wrongly) estimate its position.

Another possibility to keep in mind is that saccadic distortions might occur, not because of an active remapping process compensating for receptive fields displacements (as is most classically assumed), but as a simple consequence of an altogether different process. For example, a local and transient change in gain control around the saccade target in a retinotopically organized “position” map (e.g., where position would be represented by a hill of neural activity, which would be “smeared” by the gain control change; Kaiser and Lappe 2004) could induce a perceived shift in position. If the spatial arrangement of the map followed a logarithmic scaling (as observed in cortical magnification), the pattern of perceived distortion would be comparable to the one proposed here. Whether the generally assumed saccadic remapping is in fact the cause or the consequence of saccadic distortions cannot be easily decided. In both cases, however, the explanation of the phenomenon would involve logarithmic coordinate changes and cortical magnification, as explained here.

In addition to localized gain control, other possible (nonexclusive) mechanisms exist that could be compatible with the present model. In particular, a local and transient increase of spatial resolution around the saccade target, due to a shift of spatial attention (Yeshurun and Carrasco 1998), would act similarly to a shift of the foveal representation and could explain at least part of the results. This would be particularly true if the resolution increase was logarithmic, in which case (2) could be made to apply for decoding the perceived position (potentially with a different set of parameters). This hypothesized attentional mechanism, being necessarily local, would have the added advantage of being immune to the abovementioned problems of predicted expansion for distal locations. Among other possibilities, the necessary attentional signals providing information about the intended saccade could arise in FEF and feed back into occipital visual areas (Hamker 2003). Such an attentional model of perisaccadic integration has been recently described by Hamker et al. (2004).

Simple explanations should not be too hastily overlooked. Although the pattern of spatial distortions occurring at the time of saccades appears fairly complex, with translation and compression dominating at differ-

ent eccentricities, its key features can in fact be accounted for by a simple model assuming a uniform shift of spatial representations in cortical log-coordinates.

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