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Planned action determines perception: A computational model of saccadic mislocalization

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Abstract. Studies that investigated perception around saccadic eye movements reported a mislocalization of briefly flashed stimuli. We propose an anatomically and physiologically plausible model in which mislocalization originates early in the visual pathway through spatial reentry.

1 Introduction

The perception-action-cycle describes how actions can affect our perception by changing the sensory input. However, little is known about how planned actions influence our perception. In the domain of eye movements, models of vision have generally assumed that eye movements serve to select a scene for perception, such that action and perception are sequential processes. We suggest a less distinct separation. According to our model, occulomotor areas responsible for planning an eye movement, such as the frontal eye field, influence perception prior to the eye movement. The activity reflecting the planning of an eye movement reenters the visual areas and sensitizes all cells within the movement field such that the planned action determines perception [2].
In this paper we show that this spatial reentry theory, originally proposed to explain phenomena of attention, can account for the finding of a 'compressed' visual space. Recent studies that investigated perception around saccadic eye movements in the presence of spatial references reported a mislocalization of briefly flashed stimuli towards the saccade target [10], [7]. Such a 'compression' of the visual space occurs even before the eye starts to move. It has been suggested that the observed pattern can be explained by two effects. An extraretinal eye position signal results in a positive or negative shift of the perceived position along the direction of the saccade and another process implements a compression of visual space [8]. While a number of other studies further investigated those phenomena, the origin of 'compression' is still unknown. A recent study showed that the amount of mislocalization crucially depends on stimulus position. By presenting dots at various positions in space (Fig. 1A) it has been found that compression does also occur orthogonal to saccade direction. This finding seemed to further complicate a reasonable explanation. We here give a simple explanation for the observations.

2 Model

The outline of the model is as follows: The flashed stimulus (e.g., a bar or a dot) is processed through the hierarchy of visual areas. At present we focus on two areas and denote these areas with stage 1 (S1) and stage 2 (S2). The flashed stimulus exerts an activity hill on the cortical surface as determined by the receptive field size and cortical magnification (Fig. 1B). We follow the assumption that a saccade related reentry signal modulates visual processing in these areas [2]. We simplify the approach such that the reentry signal targets S2 to modulate the gain of its input. Thus, we ignore the gain modulation of the S1 input and effects in later stages after S2. The activity of the saccade related reentry signal peaks at the location of the planned eye movement and falls off with increasing distance. We further assume that the reentry signal increases prior to an eye movement, is maximal around the time of the eye movement and drops in strength after saccade onset. The width of the reentry signal on the cortical surface varies with saccade amplitude. An additional, but less crucial assumption is that the width of the reentry signal also varies in time relative to saccade onset. Initially, it is broadly tuned in space but it shrinks towards a minimal size at the time of the eye movement.

The reentry signal $v^{re}_{S2,i}$ modulates the gain of the input of each cell i in S2 according to [3]

$$v^{in}_{S2,i} = v^{out}_{S1,i}(1 + w_{re} v^{re}_{S2,i}), \tag{1}$$

Thus, the input of S2 is a function of the S1 output $v^{out}_{S1,i}$ and the gain factor $1 + w_{re} v^{re}_{S2,i}$. Due to this gain modulation the neural population shifts towards the saccade target (Fig. 1B). For simplicity we assume that the maximum of the resulting population indicates the perceived location of the flashed stimulus. Since we want to observe the pattern of mislocalization produced by this gain modulation we assume that the location of the flashed stimulus can be transferred into world centered coordinates and memorized for report without any additional error.

The anatomy and physiology that support this view of visual perception is outlined in detail elsewhere [4]. In brief, consistent with the proposed approach Tolias et al. [12] report that prior to the eye movement the receptive field of cells in V4 shrinks and shifts towards the saccade target. A potential source of the reentry signal is the frontal eye field (FEF) [2]. A comparison of the neural response in MT and MST sufficiently before the eye movement with the response around the eye movement is also consistent with the idea that the encoded location is shifted towards the saccade target [6]. Since the FEF also projects to MT and MST [11], a reentry signal could explain this finding. Although the behavioral data of saccadic mislocalization is obtained from humans, we used monkey data to constrain the model with a plausible set of parameters to allow a simple fitting of the remaining parameters. We determine S1 from monkey data in area MT. However, we consider this as an initial hypothesis, since present electrophysiological data does not allow to determine the area that determines

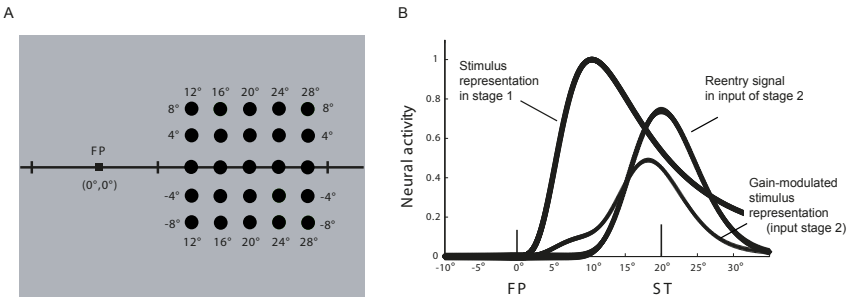

Fig. 1. A) Experimental approach of Kaiser and Lappe [5]. Subjects made horizontal rightward saccades of different amplitudes ($12°, 16°, 20°, 24°$). Around saccade onset, a green dot was flashed randomly on a red background taken from a grid of dots at positions ranging from $12°$ to $24°$ in x-direction and from $-8°$ to $8°$ in y-direction. Flash duration was 12 milliseconds. B) Computational model (1D-view) of the saccade related modulation of visual processing. Using a function of receptive field size, cortical magnification and gaze position we determine the cortical population response in stage 1 evoked by the flash of a dot. The activity in stage 1 provides the input to stage 2. The time of the flash relative to saccade onset determines the width and strength of the reentry signal. The center of the reentry signal is directed to saccade target on the cortical surface. The normalized gain modulated population response of stage 1 is clearly shifted towards the saccade target.

mislocalization. For the receptive field size over eccentricity we use [1]

$$s_{rf}^{S1}(\epsilon) = 1.04° + 0.61\epsilon$$

where ϵ denotes the eccentricity and $s_{rf}(\epsilon)$ the square root of the manually mapped receptive field area. The magnification is determined as

$$M^{S1}(\epsilon) = 1.14(0.2 + \epsilon)^{-0.76} \tag{2}$$

using a correction of the magnification in the range of the fovea as compared to the originally fitted curve [1]. The parameters of S2 are not crucial for the fitting process.

3 Results

We now demonstrate how our model quantitatively accounts for the mislocalization of briefly flashed dots (Fig. 1A). We constructed three models (M1-M3) with different cortical magnification using a mapping of the visual field V onto cortical surface C [9] (Fig. 2A). The visual field is transformed according to two cortical magnification functions $M_p(\epsilon)$ and $M_e(\epsilon)$, where ϵ denotes eccentricity. $M_p(\epsilon)$ describes the changes in cortical magnification along the meridians of the visual field and $M_e(\epsilon)$ the changes in cortical magnification along the circles

with constant eccentricity. If both functions are equal, the cortical magnification is isotropic, i.e. magnification along a circle of constant eccentricity is the same as magnification along a meridian. For M1 we used $M_p(\epsilon) > M_e(\epsilon)$, for M2 $M_p(\epsilon) = M_e(\epsilon)$ and for M3 $M_p(\epsilon) < M_e(\epsilon)$. Even an isotropic magnification results in an asymmetric mislocalization pattern. We found that a slight unisotropic magnification $M_p(\epsilon) > M_e(\epsilon)$ is more consistent with the data (Fig. 2B). Model M3 produces an almost symetric pattern (not shown).

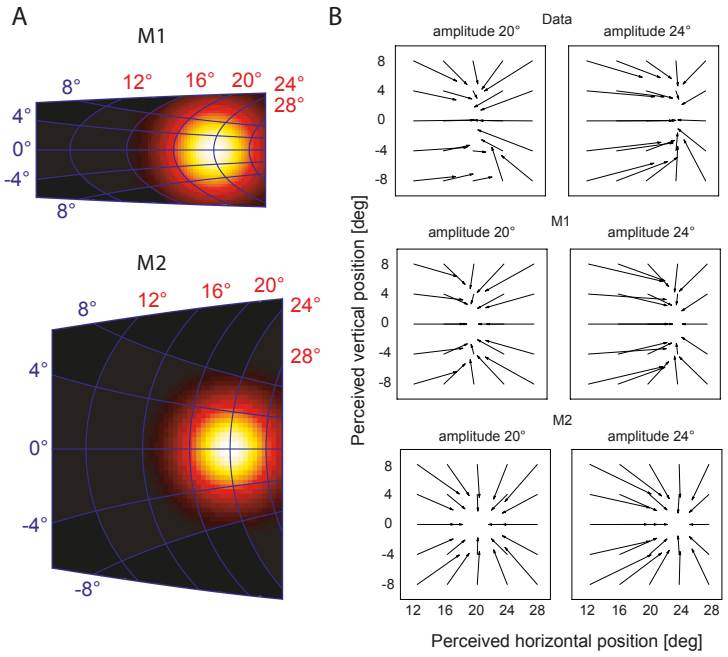

Fig. 2. A) Two mappings M1 and M2. The crossings of the grid represent the cortical positions of the flashed dots. The reentry signal is projected onto the surface and centered on 24°, i.e. this example shows a simulation of a 24° saccade. B) Spatial pattern of mislocalization for two saccade amplitudes obtained experimentally and theoretically. Vector origins indicate the veridical flash position. Vector endpoints indicate the perceived positions of the perisaccadically flashed dots. Please note that in our earlier analysis of the data [5] we computed the relative mislocalization with reference to a baseline whereas here we show the absolute mislocalization with reference to the flashed position. Although the absolute mislocalization slightly differs from the relative mislocalization pattern, the trend is similar.

The time course of mislocalization also qualitatively fits the data of dots flashed along the horizontal median (Fig. 3). The mislocalization already occurs prior to saccade onset. The exact temporal pattern is not symetric around saccade tar-

get and depends on the exact shape of the population and the timing of the eye movement relative to the decay of the reentry signal. As soon as the eye starts to move the reentry signal remains centered at the position of the planned eye movement, while the stimulus is projected to a different cortical position. Thus, stimuli flashed after the eye starts to move are mislocalized stronger into the direction of the saccade until the effect of the reentry signal considerably weakens. From a quantitative perspective we observe that the duration of compression in the experimental data is longer than predicted by the model.

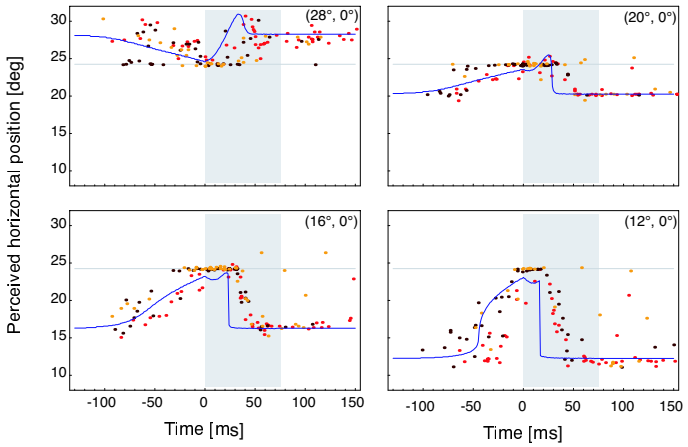

Fig. 3. Time course of compression for the 24 degree saccade. Four stimuli were flashed along the horizontal meridian $((12°, 0°), (16°, 0°), (20°, 0°), (28°, 0°))$. The gray shaded area denotes the time of the simulated saccade. The fixation point was at $(0°, 0°)$. The dots represent individual trials and the line is the fit of the model. The horizontal line shows the eccentricity of the saccade target.

4 Discussion

Our model explains 'compression' by the gain modulation due to a spatial reentry signal, which is directed to the saccade target (Fig. 2A). The reentry signal shifts the population response towards the saccade target. At the first glance, this theory would predict a symmetric compression in space. However, we have been able to show that when projecting visual space into cortical space the observed asymmetric pattern [5] is not surprising. Our model predicts that in the representative area, cortical magnification along the meridians of the visual field $M_p(\epsilon)$ is sightly larger than magnification along the circles with constant eccentricity $M_e(\epsilon)$ as least in the range where the flashes have been presented.
The model can also account for the time course of mislocalization. We have modeled the strength of the reentry signal with two exponential functions for increase and decrease. This signal is consistent with the firing rate of movement related cells, such as in the FEF [2]. Future simulations have to investigate in

how far the experimental data allows to constrain the time course of the reentry signal. Possible limitations of the present model might be that we do not simulate the time of stimulus presentation and we do not account for a compression over many areas, specifically in those which have cells with larger receptive fields. These simplifications might explain the deviations from the data after the eye starts to move (Fig. 3).

In extension to a more abstract mathematical model of mislocalization [8], this is the first comprehensive account for explaining the phenomena of perisaccadic mislocalization on a cortical and physiological basis. Previous approaches have emphasized the role of an extraretinal eye position signal. Although we postulate as well an extraretinal effect, we predict a modulatory signal that is directed to the saccade target. The level of implementation details allows to test the model with different data to investigate specific influences such as the influence of contrast and illumination, presentation time of the flash, saccade length, role of landmarks, relation between center of compression and landing position of the eye, etc.. Our computational analysis provides not only a major step towards the understanding of perisaccadic mislocalization, it further suggests that phenomena of spatial attention and saccadic compression can be unified by a spatial reentry theory.

References

1. Albright, T.D.; Desimone, R. (1987) Local precision of visuotopic organization in the middle temporal area (MT) of the macaque. Exp.Brain Res. 65:582-592.
2. Hamker, F.H. (2003) The reentry hypothesis: linking eye movements to visual perception. Journal of Vision, 11:808-816.
3. Hamker FH (2004) Predictions of a model of spatial attention using sum- and max-pooling functions. Neurocomputing. 56C:329-343.
4. Hamker, F. H. (2004) The Reentry Hypothesis: The Putative Interaction of the Frontal Eye Field, Ventrolateral Prefrontal Cortex, and Areas V4, IT for Attention and Eye Movement. Cerebral Cortex, in press.
5. Kaiser M, Lappe M. (2004) Perisaccadic mislocalization orthogonal to saccade direction. Neuron, 41:293-300.
6. Krekelberg, B., Kubischik, M., Hoffmann, K. P., Bremmer, F. (2003) Neural correlates of visual localization and perisaccadic mislocalization. Neuron. 37:537-545.
7. Lappe M, Awater H, Krekelberg B. (2000) Postsaccadic visual references generate presaccadic compression of space. Nature, 403:892-895.
8. Morrone MC, Ross J, Burr DC. (1997) Apparent position of visual targets during real and simulated saccadic eye movements. J Neurosci. 17:7941-7953.
9. Rovamo, J. and Virsu, V. (1983) Isotropy of cortical magnification and topography of striate cortex. Vision Res., 24:283-286.
10. Ross J, Morrone MC, Burr DC. (1997) Compression of visual space before saccades. Nature, 386:598-601.
11. Schall, J.D., Morel, A., King, D.J., Bullier, J. (1995) Topography of visual cortex connections with frontal eye field in macaque: Convergence and segregation of processing streams. J. Neurosci. 15:4464-4487.
12. Tolias AS, Moore T, Smirnakis SM, Tehovnik EJ, Siapas AG, Schiller PH (2001) Eye movements modulate visual receptive fields of V4 neurons. Neuron 29:757-767.