

A model of repetitive microsaccades, coupled with pre-microsaccadic changes in vision, is sufficient to account for both attentional capture and inhibition of return in Posner cueing

Ziad M. Hafed and Xiaoguang Tian, Werner Reichardt Centre for Integrative Neuroscience, Tuebingen

When a cue is presented at a location¹ (Fig. 1a), orienting efficacy towards that location is improved relative to other locations (“attentional capture”), but only briefly; a mere few hundred milliseconds later, orienting incurs large costs. These costs have been classically termed “inhibition of return” (IOR)², alluding to voluntary, cognitive strategies avoiding perseverance at one location. However, despite this popular hypothesis, the origins of both attentional capture and IOR remain to be elusive.

To understand Posner cueing mechanisms, we were motivated by two observations. First, tiny fixational microsaccades occur in a machine-like manner during cueing, with their temporal and spatial patterns likely being dictated by low-level, sub-cortical oculomotor reflexes³. Second, prior to each microsaccade, there are significant changes in visual processing^{4,5} that take place (also see Chen, Ignashchenkova, and Hafed, VSS, 2015), which probably help establish perceptual stability in the face of eye movements. We developed a model testing a simple hypothesis: that the repetitive occurrence of tiny microsaccades, coupled with pre-microsaccadic changes in vision, is entirely **sufficient** to account for both attentional capture and IOR in Posner cueing.

The first component of the model (Fig. 1b) accounts for the highly systematic temporal structure of microsaccades during cueing, and it is based on our earlier model³. Briefly, we simulated a repetitive microsaccade process using simple rise-to-threshold; a microsaccadic accumulator rose linearly with constant slope towards a threshold:

$$\frac{dM_{\text{microsaccade}}}{dt} = r_B \quad (\text{equation 1})$$

Once the threshold was reached, the accumulator was reset, and the process repeated³. If a stimulus appeared (e.g. cue onset), it reset the phase of microsaccades through countermanding: after a short afferent processing delay (ΔS), the slope of the accumulator process became time-varying (Fig. 1b, “canceled” or “escape”). It was dictated by:

$$\frac{dr_B}{dt} = \frac{r_{DN} - r_{B0}}{\tau} \quad (\text{equation 2})$$

We recently showed that this simple model captures well-known cue-induced microsaccadic modulations³. In Posner cueing, a second stimulus occurs after the cue (Fig. 1a). In our present model, this acted exactly like the cue, but it also released a response accumulator (which was identical to equation 1) to model either button or saccade reaction time (RT) to the target. The slope of the response accumulator was dictated by the instantaneous direction of microsaccade accumulation at target onset, and completely independently of the prior cue (Fig. 1c): if the microsaccade accumulator at target onset was rising for a direction congruent with the target location, the response accumulator was faster to rise than if the microsaccade was opposite. This aspect of the model simulates pre-microsaccadic modulations of visual bursts, which we robustly see neurophysiologically (Chen et al., VSS, 2015) (Fig. 2), and which influence RT⁶. Critically, this means that final performance in the model (Fig. 3) is dictated by pre-microsaccadic modulation of the response accumulator, and completely independently of prior cueing.

To summarize, our model posits that cues reset the phase of ongoing 1.5-3 Hz microsaccadic temporal frequency rhythms; attentional capture and IOR simply depend on the post-cue phase of the reset rhythms at which subsequent targets appear. We conclude that “attentional capture” and “IOR” may surprisingly be simple emergent properties of motor rhythmicity. More broadly, the strong explanatory power of phase modulation in our model suggests that attentional alterations may be manifestations of existing oscillatory brain fluctuations, which are merely uncovered when cues reset them.

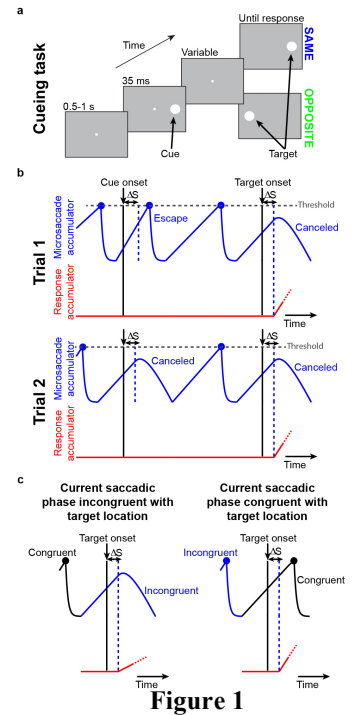


Figure 1

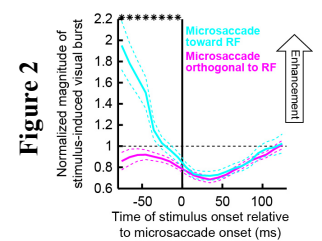


Figure 2

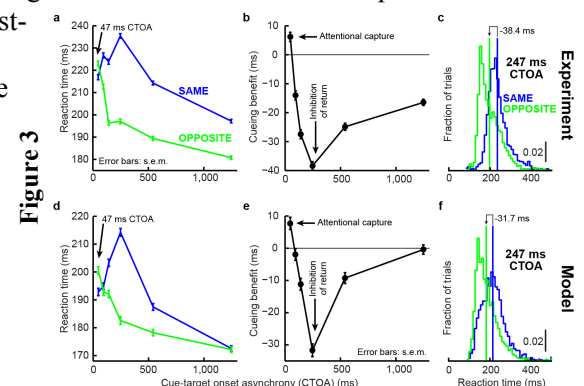


Figure 3

- 1 Posner, M. I. *Q J Exp Psychol* **32**, 3-25 (1980).
- 2 Klein, R. M. *Trends Cogn Sci* **4**, 138-147 (2000).
- 3 Hafed, Z. M. & Ignashchenkova, A. *J Neurosci* **33**, 16220-16235 (2013).
- 4 Hafed, Z. M. *Neuron* **77**, 775-786 (2013).
- 5 Hafed, Z. M. & Krauzlis, R. J. *J Neurosci* **30**, 9542-9547 (2010).
- 6 Boehnke, S. E. & Munoz, D. P. *Curr Opin Neurobiol* **18**, 544-551 (2008).