

Modeling Motion Induction to Analyze Connectivity in the Early Visual System [★]

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Abstract

Early cognitive bottom-up induced effects are stable and easier to observe experimentally than cognitive top-down effects. Motion induction (MI) is an example of such an effect. It is an illusionary visual motion perception based on the successive presentation of a small light cue followed by a light bar. By analyzing and modeling the underlying neurophysiological mechanisms insight into the connection structure of the early visual system is gained. For the MI-effect a pre-depolarization around the cue plays a dominant role. It is mediated by feedback and intracortical connections for delays between cue and bar <100ms and by top-down innervation for longer delays.

Key words: computational model, psychophysics, attention

1 Introduction

If the presentation of a cue in form of a small light spot is followed by the presentation of a light bar, subjects perceive a motion with direction away from the cue (Fig. 1). This is called the ‘motion induction’ (MI) effect [1]. Psychophysical studies showed that this effect is fast, dominant and extremely stable, indicating that it is pre-attentive (for a review see [2]).

It has been shown that the percentage of trials I in which a MI-effect is reported depends on the delay time between cue and bar, called ‘stimulus onset asynchrony’ (SOA) [1] (cf. Fig. 2 top). Two dynamic components of I can

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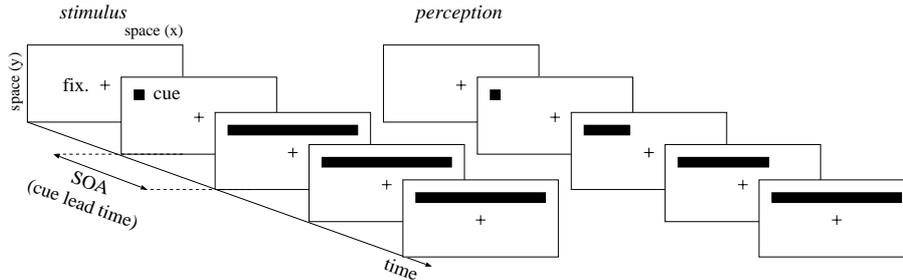


Figure 1: Experimental setup of the psychophysical MI-experiment (left) and the perceptual result (right). The delay between cue and bar is called *stimulus onset asynchrony* (*SOA*). Although the whole bar is presented at once, a motion away from the cue is perceived. Fixation is constant during the whole time at a central fixation point (fix. +).

be distinguished: A transient component that builds up after approximately 20 ms and peaks with $I=100\%$ between 50 and 120 ms, and a sustained one that saturates after 100 to 150 ms and which can last up to several seconds. The neurophysiological mechanisms underlying the MI-effect are not clear, it has been proposed, though, that the substrate of the MI-illusion is located in early stages of the visual system, perhaps as early as in V1 [1,3].

2 Analyzing the Underlying Neurophysiological Mechanisms

Based on the experimental facts an hypothesis about the underlying neurophysiological mechanisms can be formulated: The cue leads to a facilitation in form of a depolarization (priming), which will be maximal around the location of the cue and decay with distance due to decreasing connection strengths. Thus, if the bar is presented, neurons closer to the cue will fire earlier than neurons farther away. This will lead to detectable differences in firing times which will in turn cause a motion perception.

A remaining question is whether this depolarizing facilitation is caused by a feedforward and/or by an intracortical spread of activity. We suggest that the MI-effect cannot be explained by a feedforward effect alone. This hypothesis is based on experiments indicating that the priming around the cue can have a range of up to 7° [2]. It is extremely unlikely that thalamocortical point-spread connections can reach so far, but possible that intracortical ones do [4,5].

In a simulation of a biologically realistic model this hypothesis of the neurophysiological mechanisms causing the motion illusion is tested. The model describes the afferent signal flow of the primary visual pathway (retina, LGN, V1) using integrate-and-fire neurons. The input stage is the retinal layer, which is not modeled explicitly, but represents a deterministic spatio-temporal firing pattern. This serves as the input to the LGN. The projection is topographic

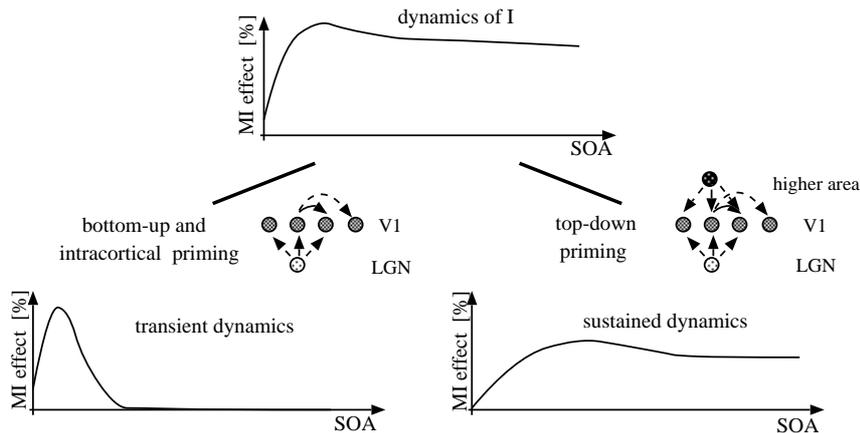


Figure 2: A superposition of two different mechanisms with different time course can explain the dependence of the dynamics of the percentage of trials I —in which a motion perception is reported—on the delay between cue and bar (s.c. SOA, top). Thalamocortical bottom-up and intracortical priming around the location of the cue are strong and fast but only transient (left), whereas a top-down induced priming is sustained but weaker and slower (right).

with adjustable convergence (usually 1:1). The connections are excitatory and very strong. LGN cells are reciprocally excitatorily connected with cortical cells. The connections are also topographically arranged with adjustable convergence and divergence parameters (usually 10:1). The connection strengths decay with distance. Intracortical connections are also excitatory. V1 neurons additionally get an excitatory input from a higher cortical area (e.g. V2). This input is not explicitly modeled but switched on or off by a black-box model.

In a first simulation we tested the basic hypothesis, i.e. that a pre-depolarization with decreasing strength can cause firing time differences, which can, in turn, be responsible for the motion illusion (Fig. 3).

The stimulus elicits a firing activity in the retina, which is transferred to the LGN. The LGN, in turn, is the main input to the cortical layer, where the size of the activated region slightly increases due to the feedforward divergence of the thalamocortical connection, and also due to the lateral intracortical spread of activity (not shown). Connection weights are adjusted in a way that the intracortical spread will elicit a subthreshold response in form of a depolarization in the region where no stimulus is given [4]. The strength of the depolarization decays with distance from the cued region. If the lateral spread of activity elicited a suprathreshold firing response the system would react with a state of extremely high firing which is not physiologically realistic.

When the bar is presented the (pre-)depolarization around the cued region leads to differences in firing times of the neurons inside the cortical layer. Neurons farther away from the cued region fire later (Fig. 3 B, C). The differences in firing time can lead to a motion perception away from the cue just

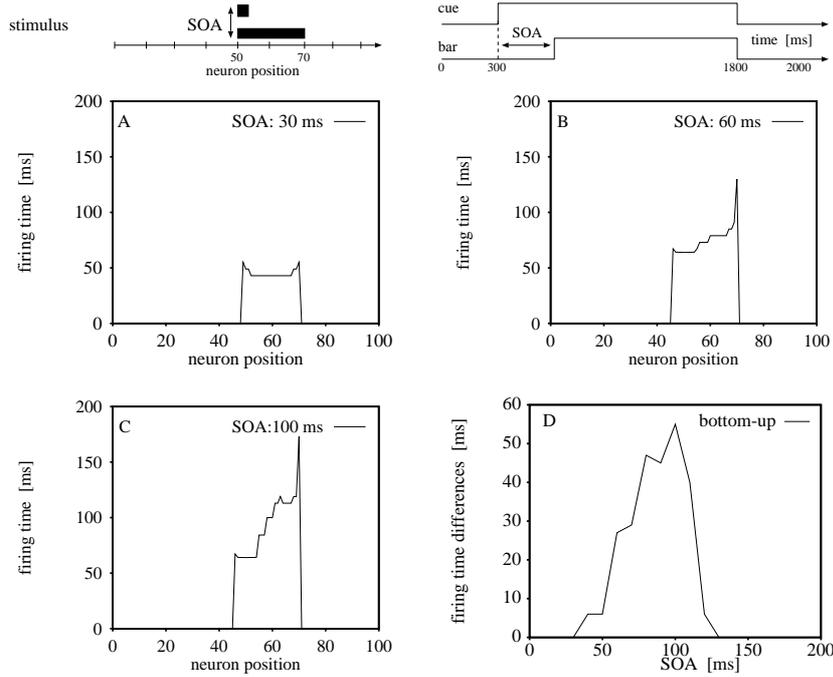


Figure 3: Simulation of bottom-up induced MI. Spatial and temporal setup of the stimulus are shown at the top. A–C: Initial firing times for the cortical neurons as a function of neuron position. The delay between dot and bar, the SOA, is varied (A: 30 ms, B: 60 ms, C: 100 ms). D: Firing time differences between neurons at position 50 (cue) and 69 (right end of bar) as a function of the SOA.

as reported in the experiments. A perception mechanism is not implemented in the model because it is not necessary for the basic mechanism causing the differences in firing times. In a simulation without intracortical connections the spread of activity is not sufficient (not shown).

In accordance with the experiments, our simulations also show a dependence of the magnitude of the differences in the firing times on the SOA (Fig. 3 D). If the bar follows the dot immediately, firing time differences are small or not existent at all, because there is not enough time for the activity to spread out in the cortical layer (Fig. 3 a, SOA: 30 ms). Even more important is the fact that the activity fed into the network is not strong enough to depolarize a wide area in V1, if the cue is only presented for a small time.

If the delay is too long (>100 ms), the pre-depolarization will have decayed before the bar is presented and thus the firing time difference will also decay. The decay is mainly caused by adaptation processes, which reduce the activity on all levels of the system. Thus, especially the weak subthreshold activity decays and there is no priming around the cue anymore. Thus, all neurons will fire at the same time when the bar is presented and no motion is perceived.

In between the described small and large values of the SOA, the firing time differences reach a maximal value (around 100 ms SOA, Fig. 3 D).

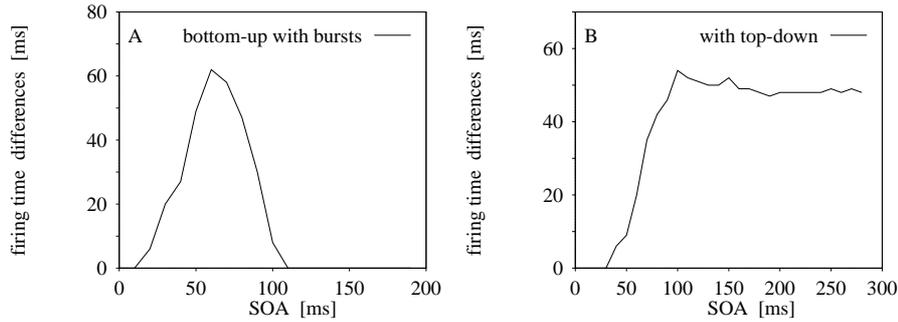


Figure 4: A: Simulation of bottom-up induced MI including a strong phasic burst component in the LGN. B: Simulation of MI including a top-down component. In (A) and (B) firing time differences are plotted as a function of the SOA. The simulations are similar to the one carried out for Fig. 3 (F) with the exception that in (A) LGN neurons respond with a burst at stimulus onset, which leads to a shift of the curve to the left and that in (B) a subthreshold top-down induced component is added to the network, which leads to persistent firing time differences.

In all, the transient (bottom-up) component of the MI-effect (left part of Fig. 2) can be simulated by the model in a satisfying way. Still, there is a difference to the experimental data as most subjects already report a motion perception for a delay of 0–50 ms. In this regime the model exhibits no significant time differences (Fig. 3 E). Thus, the model has to be extended: In the simulations described so far, thalamic cells exclusively fired with a tonic mode, while the strong initial phasic mode, which is well known from experiments [6], was neglected. If this is changed and the onset of the cue elicits a strong high-frequency (burst) component, activity will spread out farther in the cortical layer due to temporal summation. The increase of the area of suprathreshold cells results—via the intracortical connections—in an increase of the area of subthreshold activity. Therefore, firing thresholds will be reached earlier and firing time differences already exist for SOAs around 50 ms. This results in a shift of the dynamics to shorter SOAs (Fig. 4 A). Now, a better agreement between experiment and model is achieved. The effect of bursts can be psychophysically tested by using non-flashed stimuli with a smaller luminance difference to the background which should hinder bursts. Neurophysiologically, bursts could be eliminated by blocking the calcium channels responsible for the bursts.

Although experiment and model are in better agreement with bursting LGN-model-neurons, still, the model does not show a motion-induction effect for longer SOAs. On the basis of the known physiology this sustained MI-component can only be explained by an additional top-down component [5] which is responsible for maintaining the depolarization around the cue for longer SOAs. As the exact neurophysiological mechanism of attentional facilitation is not known, this top-down component is implemented as a black box mechanism in the model. This depolarizing activity is given as an additional input to V1- neurons around the cue (2). It reflects an attentional beam being

send down from higher areas. The resulting MI-curve is shown in Fig. 4 B and is in good agreement with the experimental data.

3 Conclusions

With the help of a biologically realistic model we showed that MI can be explained on the basis of firing time differences caused by a pre-depolarization around the cued region. In addition, analyzing the MI-dynamics different ways of how activity can spread in the cortical network are revealed (Fig. 2). To this end, thalamocortical feedforward connections are important to provide the necessary input for a pre-depolarization. As the MI-effect extends over several degrees in space and as it acts on a time scale of up to 100 ms a feedforward mechanism alone is not sufficient to model the observed effect. The necessary far-reaching, relatively slow (~ 100 ms) subthreshold depolarization is provided by excitatory intracortical connections. To fully explain the onset of the MI-effect (SOAs <50 ms) a strong phasic component of the LGN cells and for the sustained MI-component (SOAs >100 ms) a top-down component to V1 cells is necessary.

In all, the motion induction paradigm is well suited to analyze the connection structure in the early visual system and allows to combine modeling with neurophysiological and psychophysical experiments.

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