# Facilitatory Neural Activity Compensating for Neural Delays as a Potential Cause of the Flash-Lag Effect

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***Abstract*— In flash-lag effect (FLE), the position of a moving object is perceived to be ahead of a brief flash when they are actually co-localized. This phenomenon may be due to motion extrapolation: The nervous system has internal conduction delay, thus signals received with a delay in central areas have to be extrapolated for the internal state to be temporally aligned with that of the environment. The precise neural mechanism of such a process has not been fully investigated. Here, we propose that facilitating synapses can be a potential candidate. We tested this idea in FLE and showed that our model behavior is consistent with experimental data. In sum, facilitatory neural dynamics may underlie delay compensation, thus giving rise to FLE.**

## I. Introduction

Flash-lag effect refers to a visual phenomenon in which the position of a moving object is perceived to be ahead of a briefly flashed object when they are actually co-localized [1], [2]. Interestingly, flash-lag effect has been found in various sensorimotor modalities such as motor performance [3], auditory perception [4]; and in various visual modalities such as color, pattern entropy, and luminance [5].

A potential explanation for flash-lag effect (FLE) is motion extrapolation: Flash-lag effect may be caused by a delay compensation mechanism embedded in our nervous system (see, e.g., [2], [6]). Fig. 1 illustrates visual motion flash-lag effect in view of the motion extrapolation hypothesis. In Fig. 1$a$, the state $S(t)$ of a moving object (black rectangle) which is physically aligned with a flashed object (white solid rectangle) in the environment is received at a peripheral sensor (such as the retina) at time $t$. The state information $S(t)$ takes time ($= \Delta t$) to travel from the sensor to the central nervous system in the organism. If the delay is not taken into consideration, the perceived location of the moving object based on state $S(t)$ will be outdated by time $t + \Delta t$ (Fig. 1$b$). In that case, there will be no flash-lag effect because the location information of moving stimulus and flashing stimulus are the same, albeit delayed (Fig. 1$b$). On the other hand, if the received object location is corrected based on a predicted (or extrapolated) state of the moving object for $t + \Delta t$, i.e., $S(t + \Delta t)$, then the extrapolated object location will be closer to the actual environmental state at the time of the perception (Fig. 1$c$). The flashed object, on the contrary, is perceived without extrapolation because the abrupt flashing has no previous history to be extrapolated from. Thus, visual displacement occurs between the moving bar and the flashed bar due to such a discrepancy in extrapolation.

However, the motion extrapolation model has some limitations. For example, humans do not perceive displacement between a flashed object and a moving object when the moving object stops or reverses its direction of motion at the time of the flash [7] (see Fig. 2$a$ for an illustration of motion reversal). In other words, to make human subjects perceive a moving bar and a flashed bar to be aligned at the instant of flash, the flashed bar should be presented ahead of the moving bar. However, as the position of the moving bar approaches the motion reversal point, the gap between the perceived location of the two objects decreases (i.e., diminished flash-lag effect) and increases again after motion reversal. As illustrated in Fig. 2$a$, the perceived location of the moving bar leads (solid line), while that of the flashed object lags behind (points marked $*$).

To resolve this issue, "postdiction" [8] has been suggested as an alternative, which explains that the visual system uses motion information occurring *after* time $t$ to compute the perceived location at time $t$. It distinguishes perception time from neural-activity time assuming that perception is established by integrating information from the past, the present, and the near future that fall within a fixed temporal window. With the postdiction model, the lack of flash lag effect around the point of motion reversal can be explained. However, with this scheme, the nervous system would face perceptual delay (from 80 to 100 ms) in addition to neural transmission delay (tens to hundreds of milliseconds), which can become a serious problem for real time computation.

Despite its limitation in explaining motion reversal (or termination) condition, the extrapolation model has many desirable properties [9], [10], [11], [12]. In this paper, we suggest that the limitation can be overcome by an extended extrapolation model. An interesting question arises at this point: "What is the neural mechanism embedded in central nervous system to implement such an extrapolation process?" This is an important question that has not been fully investigated. One potential mechanism is the facilitatory dynamics found in neurons. Previously, facilitating (or depressing) synapses have been studied in the context of memory (e.g., sensitization and habituation, [13], [14]) or temporal information processing [15], [16], [17], [18]. Here, we suggest that facilitating synapses may also play an important role in compensating for neural delays. Facilitatory neural activity may effectively compensate for neural delays, and as a result it may cause

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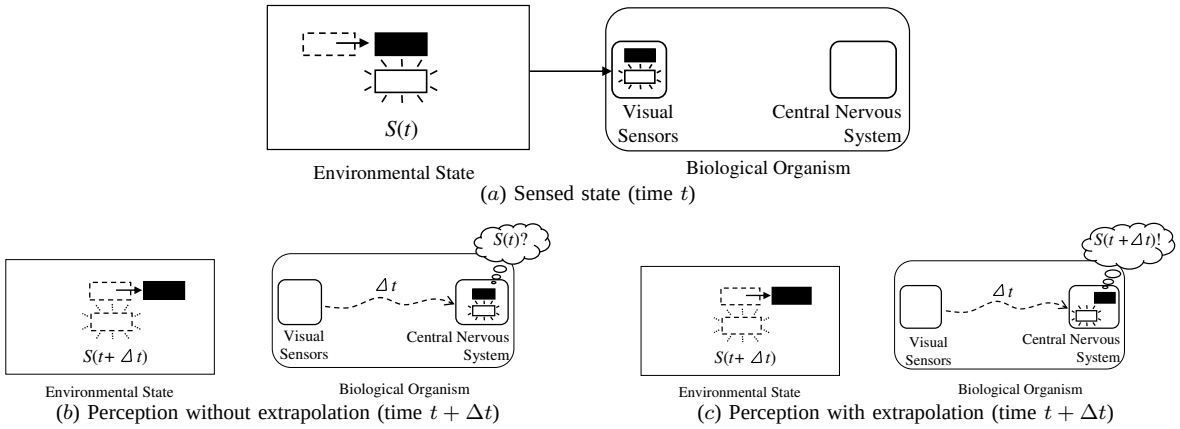



($a$) Sensed state (time $t$)

($b$) Perception without extrapolation (time $t + \Delta t$)

($c$) Perception with extrapolation (time $t + \Delta t$)

Fig. 1. **Flash-lag effect in the view of compensation mechanism for neural delay.**

extrapolation in perception (e.g., as expressed in the visual flash-lag effect).

To test our idea, we formalized the extrapolation mechanism as facilitatory neural activity (we call it the *facilitation model*) and revisited the motion reversal FLE previously modeled by postdiction [19]. The results indicate that our facilitation model can successfully account for the data from human subjects for both FLE in continuous motion and abolished FLE in motion reversal point, while minimizing the effect of neural conduction delay.

Our initial facilitation model is based on a firing-rate model, where continuous-valued neural activity was represented as a single real number. However, biological neurons communicate via spikes, thus the biological plausibility of the facilitation model may be questioned. To address this issue, we derived a spike-based model based on known neurophysiological mechanisms that can express facilitatory behavior. We extended an existing facilitating synapse model [15], [16] to account for both increasing and decreasing firing rates, thus firmly grounding the facilitation model on a biological foundation.

In the following, first, the model and experiment with motion reversal FLE will be presented to test whether the facilitation model can explain the FLE in terms of delay compensation (Sec. II). Next, a spike-based single-neuron model for facilitating activity will be proposed, followed by tests with luminance FLE (Sec. III). Finally, we will conclude, with a brief discussion of possible extensions of the model presented in this paper (Sec. IV–V).

## II. Facilitation Model for Delay Compensation

To test our facilitation model as a delay compensation method, we conducted experiments in the motion reversal FLE and compared the results to that of postdictive optimal smoothing model. First, we will briefly review the postdiction model by Rao et al. [19] (Sec. II-A) and then present our facilitation model (Sec. II-B).

### A. Optimal smoothing: postdictive perception

Rao et al. [19] formalized postdiction using *optimal smoothing*, a commonly used method in engineering applications [20]. They tested the model in motion reversal FLE and showed that optimal smoothing can successfully account for the curve around the reversal point which is observed in human experiments (Fig. 2$a$, $t = t_1$). Using Kalman filtering [21], the best estimate of the location $\hat{X}(t)$ of a moving object at time $t$ is derived from its predicted location $\bar{X}(t)$ with error correction $G(t)(X(t)-\bar{X}(t))$ after observing the current value $X(t)$ [19]:

$$\hat{X}(t) = \bar{X}(t) + G(t)(X(t) - \bar{X}(t)), \quad (1)$$

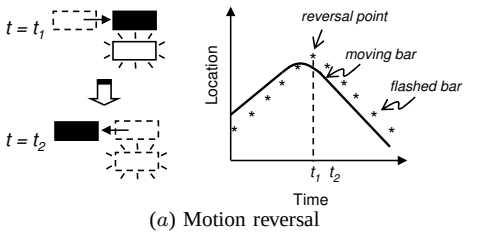
$$\bar{X}(t) = \hat{X}(t-1) + c(t-1)\hat{Y}(t-1), \quad (2)$$

where $G(t)$ is a gain term, $c(t-1)$ denotes motion direction at time $t-1$ (1 for forward and $-1$ for reverse trajectory), and $\hat{Y}(t-1) = \bar{Y}(0) = a$, which indicates the velocity of the object ($a = 1$). To estimate the final perceived location, the best estimate $\hat{X}$ is recursively smoothed using the estimation from future time steps:

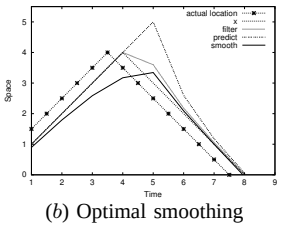
$$X_{sm}(t) = \hat{X}(t) + h(t)(X_{sm}(t+1) - \bar{X}(t+1)), \quad (3)$$

where $h(t)$ is a gain term and $X_{sm}(t)$ the final perceived location of the moving object at time $t$.

Fig. 2$b$ shows results from motion reversal experiments modeled by Eq. 1 through 3 (with $G(t) = 0.7$, $h(t) = 0.5$). The $x$ axis represents time (second) and the $y$ axis the location of the object. The velocity of the moving bar was 1 m/sec and the neural delay 500 ms. The actual trajectory of the two objects (solid line for the moving bar and $*$ for the flashed bar) resulted in delayed neural activity $X$. Based on the input $X$, the optimal smoothing method generates a predicted, filtered, and smoothed estimation for the perceived object location. Notice that even though the smoothed trajectory (solid dark line) faithfully reproduced the curve around reversal point (Fig. 2$a$, $t = t_1$), the estimated location undershot the actual locations unlike in the standard flash-lag effect for continuous motion without reversal. Another point to note here is that for the standard FLE, the smoothed trajectory should be shifted to the left based on the temporal integration of position signals (see [8], [19] for details). Certain problems arise here since the final perceptual location is determined by recursive smoothing (which is possible only *after* observing all the input

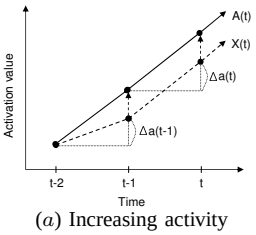



($a$) Motion reversal

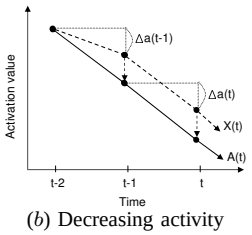



($b$) Optimal smoothing

Fig. 2. **Motion reversal experiment and optimal smoothing.**




($a$) Increasing activity




($b$) Decreasing activity

Fig. 3. **Facilitatory neural activity.**

data), and the temporal window needs visual integration mark (i.e., flash) to be set on, which contradicts with the finding that localization error exists when there is no accompanying flash [9], [11]. Also, the window size is hard to determined for a varying stimulus configuration. In sum, the smoothing model may be difficult to be applied within the nervous system, which has to work in almost real time.

### *B. Facilitation model: extrapolative perception*

Besides its limitation in explaining motion reversal in FLE, the extrapolation model has many desirable properties [9], [10], [11], [12]. In this section, we will derive a simple facilitation (extrapolation) model and its extension to overcome limitations regarding motion reversal or termination.

How can the motion extrapolation model be implemented in the nervous system? Our hypothesis is that for fast extrapolation, single neurons should be able to extrapolate. Let us assume that the activation value $X_i(t)$ of a neuron $i$ at time $t$ represent the the perceived location of a moving object at time $t$. In a network of neurons, the activation level can be defined as follows:

$$X_i(t) = g(\sum_{j \in N_i} w_{ij} X_j(t)), \tag{4}$$

where $g(\cdot)$ is a nonlinear activation function (such as a sigmoid function), $N_i$ the set of neurons sending activation to neuron $i$ (the connectivity graph should be free of cycles), and $w_{ij}$ the connection weight from neuron $j$ to neuron $i$. Based on Eq. 4, the activity of a neuron with facilitating mechanism can be defined as follows (we will drop the neuron index $i$ for notational simplicity):

$$A(t) = X(t) + (X(t) - A(t-1))r, \tag{5}$$

where $A(t)$ is the facilitated activation level at time $t$, $X(t)$ the instantaneous activation solely based on the present input at time $t$, and $r$ the facilitation rate ($0 \leq r \leq 1$). The basic idea is that the current instantaneous activation $X(t)$ is augmented with the rate of change $X(t) - A(t-1)$ modulated by the facilitation rate $r$. We will refer to the rate of change as $\Delta a(t)$ ($= X(t) - A(t-1)$).

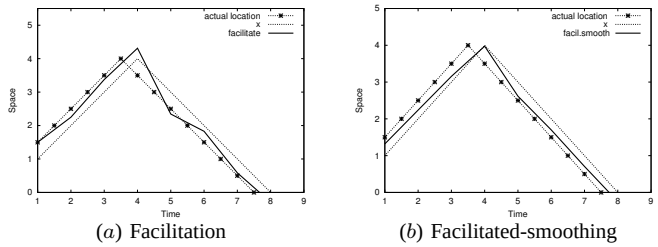
Note that Eq. 5 is similar to extrapolation using forward Euler's method where the continuous derivative $A'(\cdot)$ is replaced with its discrete approximation $\Delta a(\cdot)$ [22] (p. 710). Fig. 3 shows how facilitatory activity is derived from the current and past neural activity (for both increasing and decreasing activation levels). Basically, the activation level $A(t)$ at time $t$ (where $t$ coincides with the environmental time) is estimated using the input $X(t)$ that arrived with a delay of $\Delta t$. If the facilitatory rate $r$ is close to 0.0, $A(t)$ reduces to $X(t)$, thus it will lag behind in comparison to the environment. If $r$ is close to 1.0, maximum extrapolation will be achieved.

We will now apply our model to the motion reversal experiment under the same conditions as in Sec. II-A. Fig. 4$a$ shows the results where $X(t)$ corresponds to the delayed neural activity arrived in the visual cortex and $A(t)$ the facilitated perception for the location of the moving bar ($r = 0.5$). If there is no facilitating neural activity, the perceived location will be significantly behind the real positions (refer to Fig. 1$b$ for better understanding of this point). With facilitatory neural activity, however, the delayed neural signal is facilitated so that the perceived locations (solid line) become closer to the actual location (solid line with $*$) of the moving bar in the environment at the same instant. Notice that the visual FLE occurs due to the spatial gap between the facilitated activity for the moving bar (solid line) and the non-facilitated activity for the flashed bar (dotted line). However, as mentioned above as a shortcoming of the extrapolation model, the facilitated perception generates an overshoot around the reversal point, which is not found in human experiments.

What happens at the instant of the motion termination or reversal? A potential answer is that the misperception (i.e., overshoot at reversal) is corrected by backward masking with an immediate motion offset signal (see [23] for details). The motion offset signal which arrives at the time of extrapolation can cancel out the extrapolatory neural activity so that there is no overshooting perceived at the terminating (or reversal) point. This is consistent with the postdiction model to some extent such that information occurring after time $t$ can affect the judgment about the perceived location at time $t$. However, differentiating from optimal smoothing, our model uses extrapolated activity $A(t)$ instead of the filtered estimate $\hat{X}(t)$ for smoothing, and considers only one step future event to modify the estimate. (We assume that the time step is much smaller than the transmission delay, i.e., $1 \ll \Delta t$.) Thus, the perceived location can be redefined as follows:

$$A_{sm}(t) = A(t) + h(t)(A(t+1) - X(t+1)), \tag{6}$$

where $A_{sm}$ is called facilitated smoothing value. Compared to Eq. 3, Eq. 6 is much simpler and suitable for real-time perception. Fig. 4$b$ shows the result using Eq. 6 (with $h(t)$ set to $0.4$). Compared to Fig. 4$a$, facilitated smoothing ($A_{sm}$; dark solid line) successfully produces no-overshoot at the time of




(*a*) Facilitation (*b*) Facilitated-smoothing

Fig. 4. **Perceived trajectory in the facilitation model.**

reversal as well as closely approximating the overshoot events for continuous visual motion.

This computational result strongly suggests that our nervous system may use extrapolatory neural mechanisms for delay compensation as well as a small delay in perception to increase perceptual accuracy.

## III. Spike-based model for facilitating activity

The model in the previous section treated neural activity as a single real number. How could the facilitatory dynamics as described in Eq.5 and Eq.6 be implemented in a more biologically plausible manners? In this section, we will propose a spike-based model based on known neurophysiological mechanisms that can potentially express facilitatory behavior.

### *A. Single neuron model with facilitating synapses*

Dynamic synapses generate short-term plasticity which shows activity-dependent decrease (depression) or increase (facilitation) in synaptic transmission occurring within several hundred milliseconds from the onset of the activity (for reviews see [24], [17], [25]). Especially, facilitating synapses cause growth of postsynaptic response through increasing synaptic efficacy with successive presynaptic spikes. (Synaptic efficacy is the fraction of neural transmitter released when presynaptic action potentials arrive at the axon terminal.) Unlike depressing synapses, the rate of neural transmitter release is not constant but is a dynamic variable in facilitating synapses.

According to the dynamic synapse model by Markram and colleagues, based on neurophysiological data [16], [15], synaptic efficacy $U$ evolves over time as described in the following differential equation:

$$\frac{dU}{dt} = -\frac{U}{\tau_f} + C(1-U)\delta(t-t_s), \tag{7}$$

where $\tau_f$ is the time constant for the decay of $U$; $C$ a constant determining the increase in $U$ when a successive action potential (AP) arrives at time $t_s$ at the synaptic terminal; and $\delta(\cdot)$ the Dirac delta function. This equation is already suitable enough to replicate the facilitating dynamics in Eq. 5 when the activation level is increasing (as in Fig. 3*a*). However, it is not capable of handling cases where the activation level is decreasing (as in Fig. 3*b*). Ideally, extrapolation should work for both increasing and decreasing trends.

Here we modify the equation by redefining $C$ as a dynamic variable which is varied in proportion to the change of input firing rate:

$$C = \left(\frac{I(n-1)-I(n)}{|I(n-1)-I(n)|}\right)\left(\frac{I(n-1)}{I(n)}\right) r, \tag{8}$$

where $I(n)$ is the interspike interval between the $n$-th spike and the $(n-1)$-th spike which reflects whether a spike train consists of high-frequency APs or low-frequency APs. The first term in Eq. 8 determines the sign of $C$: "$+$" for increase or "$-$" for decrease in firing rate. The second term represents the ratio of the change in frequency, and $r$ is a gain parameter. As the input firing rate increases, $C$ becomes positive and increases proportional to the rate of change in frequency. On the contrary, as the firing rate decreases, $I(n)$ becomes larger which results in a negative $C$ and thus leads to the decrease in the synaptic efficacy $U$.

With this, we can now fully describe our membrane potential model, (cf. [16], [15]). The time course of postsynaptic current $P(t)$ at time $t$ triggered by incoming spikes is defined as follows:

$$P(t) = Ee^{-\frac{t}{\tau_p}}, \tag{9}$$

$$E = AU, \tag{10}$$

where $E$ is the excitatory postsynaptic potential (EPSP) amplitude; $\tau_p$ the time constant of decay in $P(t)$; $A$ a constant for maximum postsynaptic response amplitude; and $U$ the synaptic efficacy as defined above. Finally, the membrane potential $V_m(t)$ at time $t$ evolves as follows:

$$V_m(t) = V_m(t-1)e^{-\frac{t}{\tau_m}} + P(t)(1-e^{-\frac{t}{\tau_m}}). \tag{11}$$

The membrane potential is determined by the membrane current $P(t)$ at that moment and the previous membrane potential $V_m(t-1)$, both of which are regulated by a membrane time constant $\tau_m$. The last part of the spiking neuron model is the spike generation mechanism. Once $V_m$ exceeds the spike threshold $\theta$, a spike is generated, followed by an absolute refractory period of $\tau_{\mathrm{refrac}}$ during which spikes cannot be generated. With the model above, we simulated a single neuron under increasing or decreasing input firing rate.

### *B. Experiment with visual luminance flash-lag effect*

When a stationary disk continuously becomes brighter in luminance, it appears brighter than a neighboring flashed object of equal luminance (and analogously, darker for a disk becoming darker) [5]. Such perceptual phenomena expressing extrapolation (brighter than bright, and darker than dark) can be modeled at a single-neuron level using facilitating synapses as described in the previous section.

Through sensory transduction, sensory signals such as photons hitting the retina are converted into spikes (or action potentials). These spikes cause a chain reaction through the sensory pathway to reach the primary sensory area (the primary visual cortex, in case of vision). Our interest mostly lies in the last part of the journey of these spikes, where the input spike train releases neurotransmitters from presynaptic neurons

to a postsynaptic neuron through facilitating synapses. Further simplifying this, we assumed that there is only one synapse. By varying the spike firing rate in the presynaptic neuron, we are able to model the extrapolatory phenomenon described above.

We tested two types of input: (1) increasing firing rate (analogous to the visual stimulus becoming brighter) and (2) decreasing firing rate (modeling the visual stimulus becoming darker). The parameters used for the simulation below were as follows: Initial value for synaptic efficacy $U = 0.2$; $U$-recovery time constant $\tau_f = 150$ ms; postsynaptic potential time constant $\tau_p = 30$ ms; membrane current time constant $\tau_m = 200$ ms; spike threshold $\theta = 160$ mV; duration of absolute refractory period $\tau_{\text{refrac}} = 4$ ms; maximum postsynaptic response amplitude $A = 300$; and $C$-gain $r = 0.175$. The results are shown in Fig. 5. As the results show, the facilitating synapse model generated extrapolatory neural activity for both increasing and decreasing firing rate conditions. Dynamic change in the synaptic efficacy $U$ caused the postsynaptic neuron to generate more spikes than the input when the input firing rate was increasing (Fig. 5$a$). On the other hand, the postsynaptic neuron generated less spikes than what it received when the input firing rate was decreasing (Fig. 5$b$).

This kind of behavior is quite reasonable if we consider the following. Suppose the spikes in the presynaptic neuron (the top rows in Fig. 5) were originated earlier (about 100 ms) in peripheral sensors. Here is an example sequence of events: (1) Peripheral spiking at 300 ms would be replicated at 400 ms in the presynaptic neuron in the top row, due to the delay. (2) The postsynaptic neuron (bottom row) receiving input from the presynaptic neuron (top row) at 300 ms fires from information from 200 ms in the periphery. (3) However, the postsynaptic neuron's firing rate at 300 ms (bottom row) is nearly the same as that of the presynaptic neuron's rate at 400 ms (top row). This means that the postsynaptic neuron, at time 300 ms, is exactly firing at the same frequency as the peripheral neuron at time 300 ms (refer to (1) above), precisely reflecting the present environmental state. Note that the presynaptic (top row) and the postsynaptic (bottom row) neuron in Fig. 5 are both located in the central nervous system as shown in Fig. 1.

In contrast with the previously defined facilitating synapse equation (Eq. 7, [16], [15]), our modified equation (Eq. 7 with Eq. 8) was able to generate extrapolated neural activity under both increasing and decreasing firing rate. These experiments suggest that dynamic facilitating synapses can trigger a general extrapolatory neural activity, thus providing a neurophysiological basis for the model in Sec. II. In sum, the time-varying stimuli (brighter or darker in luminance) were extrapolated to be perceived close to the present intensity of light, while the firing rate of abrupt stimulus was the same as the presynaptic neuron (not shown). The different firing rates between two stimuli might cause FLE in visual luminance change.

The last question to be resolved is what neural mechanisms can implement backward masking so that it can cancel out potential overshooting occurring at a motion-reversal point. We suggest that inhibitory synaptic transmission conveying motion offset signal may prevent the postsynaptic neuron from generating surplus spikes. Fig. 6 shows the pre-postsynaptic neural activity where motion offset signal follows the input spike train (top row) consisting of increasing firing rate. The second row in Fig. 6 shows that spikes of motion offset stimulus are delivered by an inhibitory synapse immediately following the input spike train. The motion offset signal was generated immediately after the last signal of continuously changing stimuli (around 400 ms at the periphery), and delivered with neural delay (100 ms) at presynaptic neuron. Influenced by inhibitory postsynaptic potential (IPSP) the increased postsynaptic membrane current is pulled down (the third row), which makes the postsynaptic neuron to fire the same number of APs as that of the presynaptic neuron (cf. Fig. 5$a$) so that extrapolation of firing rate did not occur at the moment of stimulus termination.

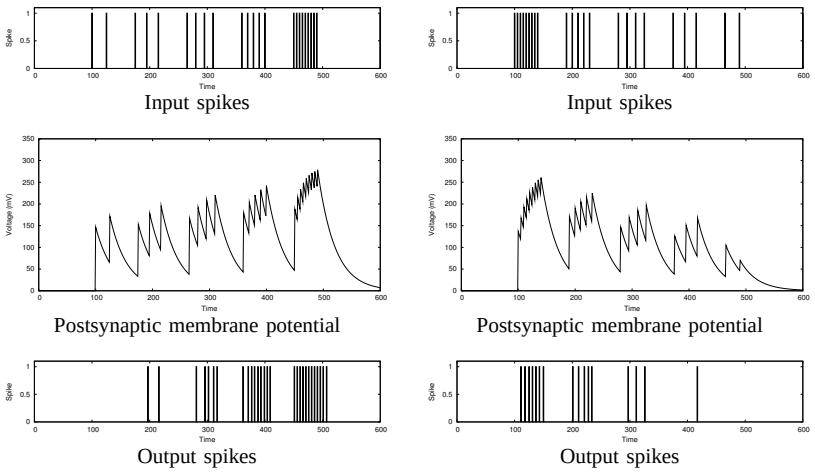
## IV. Discussion

The main contribution of this paper is that it proposed a biologically plausible neural mechanism on which motion extrapolation model can be grounded. We proposed and tested a neurophysiologically based facilitating synapse model, demonstrating that extrapolatory dynamics can compensate for neural transmission delay and various FLE may arise due to such a mechanism. We also showed that the lack of such an effect at motion reversal (or termination) can be interpreted as due to inhibitory synaptic dynamics. In fact, the motion reversal of moving bar and the visual luminance experiments showed that facilitatory neural activity helped align the internal state of the nervous system with the present rather than with the past environmental state.

We expect that our approach can be extended to explain other extrapolatory phenomena in perceptual experiments such as flash-lag effects in color, pattern entropy, localization, or orientation. However, to do this, our single-neuron approach has to be extended. For example, unlike luminance, neurons sensitive to orientation only respond to a narrow range of orientations. This means that a whole range of orientation cannot be represented with a single neuron, thus increase or decrease in firing rate in these neurons cannot indicate the presence of varying orientations in the input. For this case, facilitation has to go across different orientation-selective neurons. Another interesting future direction would be to verify whether neurons with facilitating synapses are more often found in places where delay compensation is needed more, for example, at the end of long, slow axons, or where precise real-time information is needed.

## V. Conclusion

In this paper, we have shown that facilitatory (extrapolatory) dynamics found in facilitating synapses can be the basic neural mechanism for motion extrapolation. Experiments with a moving object trajectory showed that facilitatory activation can successfully reproduce the visual motion flash-lag effect (i.e., overshooting in continuous motion as well as the lack




($a$) Increasing firing rate ($b$) Decreasing firing rate

Fig. 5. **Extrapolation with facilitating synapses.**

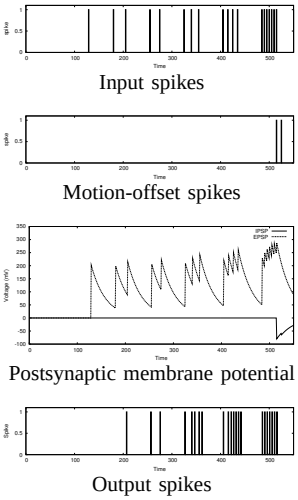



Fig. 6. **Facilitating synapse with motion-off signal.**

of such an effect at motion reversal). Also, an extended facilitating synapse model allowed the facilitation model to be firmly grounded on neurophysiology. Experiments with FLE with change in visual luminance turned out that facilitating postsynaptic activity can generate extrapolated neural activity under both increasing and decreasing firing rate conditions. In sum, we showed that facilitating synaptic dynamics can serve as a delay compensation mechanism, which may give rise to the various flash-lag effects, and help biological organisms to perceive the present environmental state in real time.

## ACKNOWLEDGMENTS

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