Spike-latency codes and the effect of saccades

Rüdiger Kupper *, Marc-Oliver Gewaltig, Ursula Körner, and Edgar Körner

Honda Research Institute Europe GmbH, Carl-Legien-Str. 30, D–63073 Offenbach/Main, Germany

Abstract

Spike-time based models of visual processing often cover only a few spike waves. This disregards the continuous character of visual processing and the fact that the visual field is scanned by series of saccades to different fixation points. Thus, some form of *reset* is needed in order to bring the neurons into a well defined state after each saccade. In this contribution we investigate the ability of various tentative reset mechanisms to maintain a latency-based temporal code in a model of layer 4 spiny stellate cells in the primary visual cortex of primates. We conclude that input suppression and inhibition of neurons are effective ways of setting temporal reference frames for spike-time processing.

Key words: latency, spike rank, saccadic suppression, temporal reference frame

1 Introduction

Many models of visual processing start from the assumption, that an image or scene is analyzed as a whole and that at the start of the analysis all neurons are essentially in the same state (e.g. [1,2]). However, at least in primates, the visual field is scanned by a series of fixations and saccades. During fixations, the stimulus is almost static, and thalamic relay cells respond by transient activation, followed by tonic firing [3]. This sustained activity is transmitted to cortical neurons throughout the fixation. It follows a saccade to the next fixation point. The neurons are then in a state which depends on the pre-saccadic fixation point and whatever happened during the saccade. This history dependence is problematic for models which rely on a consistent

* Corresponding author
Email address: Ruediger.Kupper@Honda-RI.De (Rüdiger Kupper).

temporal reference, like spike rank [1,2] or spike latency models [4]. These models need some form of "reset" in order to reduce history dependence after each saccade. For cortical neurons of the magno-cellular pathway, where a latency based code is most plausible, *saccadic suppression* has been demonstrated [5,6]. But also for the other visual pathways some form of saccadic reset seems plausible. Processing in the retina and thalamus appears to be fast enough to deliver information which is not tainted by traces of the previous input [7]. Spiny stellate cells in layer 4 of the primary visual cortex are the main targets of thalamic afferents. From here, information propagates to other cortical layers. Thus, at least for the spiny stellate cells in layer 4, we expect a reset mechanism which maintains or re-establishes the necessary conditions for an assumed latency or rank order code. History dependence of spike times will only originate from processes whose dynamics is at a time scale between that of saccades (10–40 ms) and fixation periods (100–1000 ms). Processes which are slower will have approximately the same effect in successive fixations and can be regarded as unproblematic. Processes which are faster will have subsided in time for the next fixation. Several factors contribute to the history dependence in the cortical neural response. Among them are the membrane dynamics, use dependent synaptic transmission, and intra-cortical connections. The influence of use dependent synaptic properties on the history dependence is very difficult to assess. Although the associated processes work at time-scales comparable to those of fixations, the time constants for onset and recovery differ considerably [8]. While the onset of synaptic short term dynamics is very fast, recovery takes a very long time and may be too slow to contribute to the history dependence discussed here. Here, we focus on the dynamics of the membrane potential in a feed-forward network as the most basic setting. In ongoing studies, we extend this to networks with lateral and feed-back interactions.

2 Simulation and Methods

We investigate the ability of various tentative reset mechanisms to provide a consistent temporal reference for a latency-based temporal code across saccades, in a model of layer 4 spiny stellate cells in the primary visual cortex of primates. To this model, we present input sequences, resembling those produced by a natural scan-path, assuming saccades as trigger for the reset. We discuss the following tentative reset mechanisms (fig. 1): *No action*, image patches are presented for 100 ms in direct succession and no action is performed between fixations. *Input suppression*, each presentation is followed by a 10 ms period of suppressed input, allowing the neurons to relax towards their resting potentials. And *inhibition*, strong inhibitory bursts are applied to the neurons during the last 10 ms of a fixation. A larger set of paradigms was

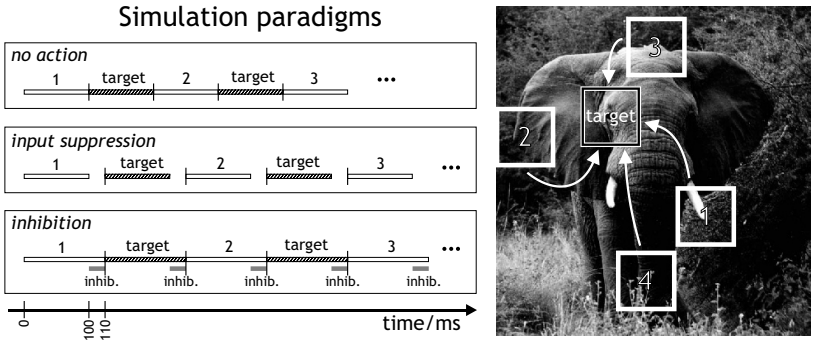

Figure 1. Simulation paradigms. Image patches are presented consecutively, separated by different tentative reset mechanisms. Patches were derived from a natural image. The target patch appears repeatedly, with varying predecessors.

shown in the conference contribution.

Directly changing from one input to the next (no action) corresponds to the idea that the transient response to a changed stimulus is sufficient to overcome the stimulus history. Input suppression corresponds to a saccadic suppression of the preceding network stages [5]. Similar effects could also be created by retinal "smear" of the visual stimulus, caused by the fast eye movement (cf. e.g. [9]). The inhibition paradigm operates at the site of the processing neurons, where high-frequency inhibitory bursts could be delivered with great effect by the peri-somatic inhibitory cells (*basket* and *chandelier cells*) in cortex [10].

Our model network consists of a 100×100 lattice of (a) standard Integrate & Fire (I&F) neurons [11], and (b) modified I&F model neurons where currents are scaled by factors $U_{rev}^{ex} - U(t)$ and $U_{rev}^{inh} - U(t)$, to approximate excitatory and inhibitory reversal potentials. The modified I&F model is biologically more plausible and will be used throughout this paper, unless otherwise indicated. "Visual" stimulation is implemented via injection of currents into the neurons, with amplitudes corresponding to grey levels, and waveforms imitating the transient and tonic parts of convergent LGN responses [3]. The input consists of 50 patches (100×100 pixels) from a natural image, each presented for 100 ms. One target patch appears alternating with different predecessors (see fig. 1), resulting in a sequence of 100 patches which resembles repeated saccades from different fixation points to one target. After each fixation, one of the tentative reset mechanisms is applied. During the input sequence, the reset mechanism is not changed. Simulations are done using the *NEST Initiative* simulator [12], at a temporal resolution of 0.2 ms.

We record the latency of the first response spike of all neurons, i.e., the time from the change of visual input until the neuron's next spike. A reset mechanism must ensure that for a particular stimulus (a particular fixation point) the response is *independent of stimulus history* (i.e., the preceding points of fixation), as well as *reproducible*, meaning that spike times should be the same when fixating the same spot again. Thus, we compute the mean and standard deviation of each neuron's spike latency, over all presentations of the target patch. This way, we quantify the reproducibility of the response to the target

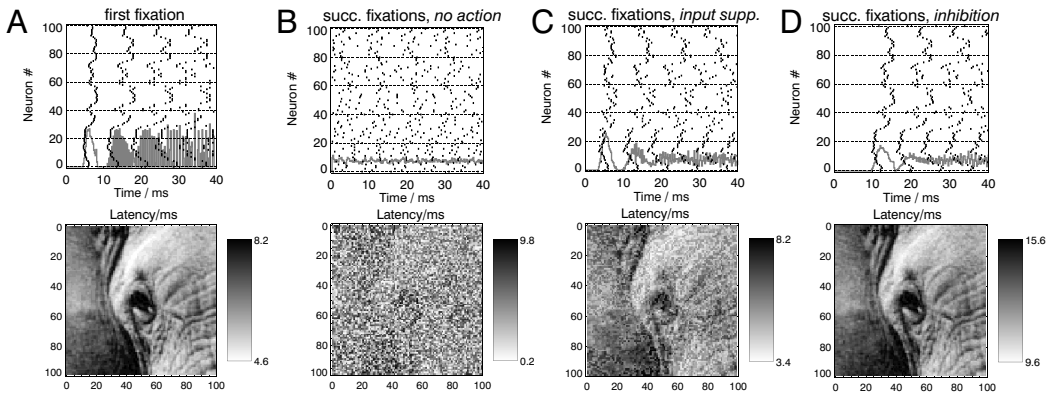

Figure 2. Network response to the target patch during presentation of the input sequence. **A:** Response to the first presentation of the target patch, starting with all neurons in the same state. **B–D:** Typical response to subsequent presentations of the target patch for different tentative reset mechanisms. **Top:** Spike trains of 100 selected neurons with spike time histograms (grey) superimposed. **Bottom:** Grey-coded first-spike latencies of all 100×100 neurons. Note the different value ranges.

patch depending on the applied reset mechanism.

3 Results and Discussion

Figure 2 shows the spike trains of 100 selected neurons, for a single presentation of the target patch. Initially all neurons are in the same state and their first spikes form a sharp spike wave (A). The latencies correspond well to the input strength. The grey-coded panel below demonstrates the good quality of the latency code: The stimulus can be reconstructed with high fidelity from the first response spikes. However, beyond the first 10 ms, the states of the neurons diverge and the spike waves dissolve. Figures 2B–D show typical results for the subsequent presentations of the target patch, with different reset mechanisms applied (fig. 1). At the beginning of each fixation, the neurons are in a state which is determined by the stimulus history and the reset mechanism. From fig. 2B, the *no action* paradigm, it is obvious that the stimulus-related information in the spike latencies of the first fixation (A) is no longer present. The transient response to a new input does not suffice to provide a consistent temporal reference for a spike time code. Panels C and D illustrate that the respective reset mechanisms are able to provide the temporal reference for a latency code. The differences between the two paradigms depend on the duration and strength of the respective reset mechanism (not shown).
To further quantify the reproducibility of the latency code, we collect the spike latencies from all presentations of the target patch. A good latency code should depend only on the current stimulus and not on the stimulus history. Figure 3A shows the raster diagram of the first response spike of one neuron

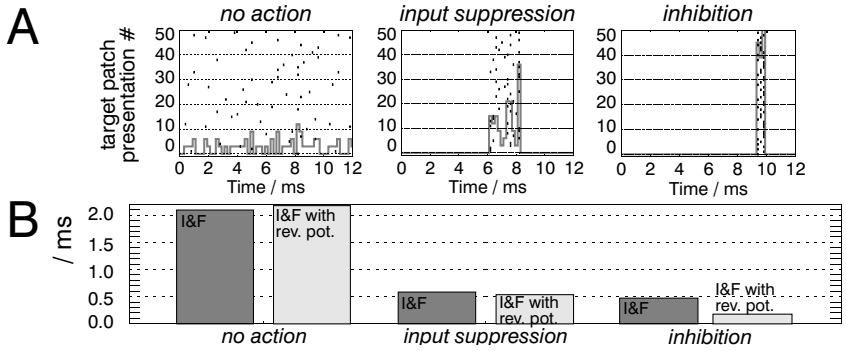

Figure 3. **A:** Raster diagram of the first response spike of one neuron to the target patch, collected over all presentations in a sequence. Spike time histograms (grey) superimposed. **B:** Standard deviation of response spike latency, averaged over all neurons, for both neuron models (I&F and modified I&F).

to the target patch, collected over all presentations in a sequence. If no reset mechanism is applied, the spike time histogram is flat, resulting in a wide standard deviation of the response spike latency (left panel). For the *input suppression* and *inhibition* paradigm, histograms are sharper, as the respective action has "reset" the neuron to a state which depends less on stimulus history (middle and right panel). Figure 3B shows the standard deviations of the first response spike latencies, averaged over all neurons. Results are shown for both neuron models (I&F and modified I&F). Inhibition acts as a better reset signal in the modified I&F model with reversal potentials, but not in the standard I&F model: The lower bound imposed by the reversal potential levels out differences in the membrane potential U when inhibition is applied, while in the standard model U is not bounded.

Figures 2 and 3 demonstrate the effect of the tentative reset mechanisms: Both *input suppression* and *inhibition* succeed in re-establishing the time-frame for latency coding that had been lost due to the history dependence. This is reached in very short time: In our model, 10 ms of input suppression or inhibition of neurons is enough to reduce the standard deviation in spike timing considerably below the typical inter spike interval, separating the spikes into distinct waves (fig. 2). The relative latencies can easily be decoded by a next stage of neurons, e.g. using a mechanism of feed-forward inhibition [2]. The temporal structure imposed on the firing sequence is also a good foundation for the spike-time based integration of feed-back signals from higher processing areas [4].

The typical duration of saccades (10–40 ms) is long enough to remove history traces caused by the membrane dynamics, and by short-term effects. Mechanisms operating at longer timescales, e.g. NMDA dynamics or recurrent cortical interactions, cannot be efficiently reset by input suppression. Here, active inhibition at the site of processing is the only choice for a reset. However, for spiny stellate cells in layer 4, input suppression is still a viable option, since the observed EPSPs at the thalamocortical synapses are short, probably mostly mediated by AMPA and not so much NMDA receptors. Such input

suppression could e.g. be produced in the LGN by actively shutting down transmission to the cortex.
We conclude that (1) spike latency codes require some reset of the processing neurons performed upon stimulus change, i.e., at saccades. (2) Both input suppression and coherent inhibition of neurons can serve as appropriate reset signals. (3) Coherent inhibition of neurons can create a more effective temporal reference than input suppression, provided that (4) a biologically reasonable neuron model like the modified I&F model is used. The standard I&F model fails to produce the effect of (3), due to missing bounds of the membrane potential.

References

[1] R. van Rullen, S. J. Thorpe, Rate coding vs. temporal order coding: What the retinal ganglion cells tell the visual cortex, Neur. Comp. 13 (2001) 1255–1283.

[2] S. Thorpe, A. Delorme, R. van Rullen, Spike-based strategies for rapid processing, Neur. Netw. 14 (6–7) (2001) 715–725.

[3] K. Funke, F. Wörgötter, On the significance of temporally structured activity in the dorsal lateral geniculate nucleus (LGN), Progr. Neurobiol. 53 (1997) 67–119.

[4] E. Körner, M.-O. Gewaltig, U. Körner, A. Richter, T. Rodemann, A model of computation in neocortical architecture, Neur. Netw. 12 (7–8) (1999) 989–1005.

[5] D. C. Burr, M. C. Morrone, J. Ross, Selective suppression of the magnocellular visual pathway during saccadic eye movements, Nature 371 (1994) 511–513.

[6] A. Thiele, P. Henning, M. Kubischik, K.-P. Hoffmann, Neural mechanisms of saccadic suppression, Science 295 (2002) 2460–2462.

[7] P. Reinagel, R. C. Reid, Temporal coding of visual information in the thalamus, J. Neurosci. 20 (14) (2000) 5392–5400.

[8] M. V. Tsodyks, H. Markram, The neural code between neocortical pyramidal neurons depends on neurotransmitter release probability, Proc. Natl. Acad. Sci. USA 94 (1997) 719–723.

[9] R. W. Rodieck, The first steps in seeing, Sinauer Associates, Inc., Sunderland, Massachusetts, 1998.

[10] M. Beierlein, J. R. Gibson, B. W. Connors, Two dynamically distinct inhibitory networks in layer 4 of the neocortex, J. Neurophysiol. 90 (2003) 2987–3000.

[11] H. C. Tuckwell, Introduction to Theoretical Neurobiology, Vol. 1, Cambridge University Press, Cambridge, 1988, Ch. 3, pp. 85–123.

[12] M. Diesmann, M.-O. Gewaltig, NEST: An environment for neural systems simulations, in: T. Plesser, V. Macho (Eds.), Beiträge zum Heinz-Billing-Preis 2001, Ges. f. wiss. Datenv. mbH Göttingen, 2003, pp. 43–70.