

Information processing with spiking neurons in a cortical architecture framework under the control of an oscillatory signal

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Abstract

In this work we analyze information processing in spiking neurons in the cortex by investigating how short (10 ms) pulse packets of spikes are processed in cortical neurons. These signals are compliant with constraints of rapid feedforward recognition in the visual cortex. We add an oscillatory membrane potential modulation that simulates the effect of 40 Hz (evoked) gamma oscillations. We find that depending on the oscillation signal different input features are decoded, processed and encoded in a single output spike. We demonstrate that a latency-coding of information is possible.

Key words: Latency-Coding, Spiking Neurons, Gamma Oscillation

1 Introduction

Spiking neurons in a realistic cortical environment show a remarkably complex input-output mapping. A number of different functions like integrator (rate count), coincidence detector or, recently, latency decoder have been proposed. In this work we analyze the input-output relation for a neuron roughly matching principal neurons in the monkey visual cortex. Experiments by Thorpe et al. on ultra-fast image recognition in monkeys and humans have brought up serious temporal constraints [8]. The extremely fast recognition of complex stimuli in about 200 ms leaves an upper limit of just 10–20 ms for information transfer between two neurons. Taking into account that cortical neurons fire with rates $\ll 100$ Hz a maximum of one spike per neuron must suffice to transmit information. This prohibits the classic single neuron rate-coding approach. It was therefore proposed [5,8] that the first, fast categorization of inputs is purely feedforward, based on one spike per neuron only and makes use of a temporal coding scheme. Essential for the coding scheme proposed in [5] are the initial (evoked) gamma oscillations which are triggered even by simple stimuli [6,7]. It was proposed that this oscillation signal is used as a reference and control signal for a latency-coding scheme that is able to transmit information with single spikes within the time limit of 10 ms. In this work we will analyze how information is decoded and encoded in a cortical neuron subjected to an external oscillatory signal. We investigate how

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the number of spikes a neuron receives (roughly corresponding to an ensemble firing rate), their temporal dispersion/variance (a measure of coincidence) and their latencies influence the neuron's output. The analysis is done via computer simulations at the behaviourally relevant time-scale of 10 *ms*.

2 Network set-up

As the basic element of our circuit (Fig. 1) we employ a spiking neuron, simulated within the spike response model (SRM) [4] framework, which is closely related to the Integrate & Fire model. The neuron's status variable is its membrane potential. Due to the one spike per neuron limit we focus on the generation of the first output spike only and neglect all refractory effects from previous spikes. Incoming spikes trigger excitatory post-synaptic potentials of the form (α function):

$$\eta_i(t) = w \frac{(t - t_i) \cdot e}{\tau} \cdot \exp\left(-\frac{t - t_i}{\tau}\right) \quad (t > t_i) \quad , \quad (1)$$

with τ as the EPSP's rise time, w as a scale factor (connection weight) and t_i being the arrival time of spike i . Contributions from different synapses are summed up linearly. In addition to these spikes, corresponding to inputs from the previous stage in the processing hierarchy, neurons are also subjected to an external oscillatory signal (see also [2]). This oscillation signal is modeled

as:

$$O(t) = \frac{A}{2} \left(1 + \sin \left(\omega t - \frac{\pi}{2} - \phi \right) \right) \quad , \quad (2)$$

with $\omega = 2\pi f$ and f being the frequency of the oscillation. The oscillation can be described by amplitude A , frequency f , and phase ϕ . This signal is added to the membrane potential. If the sum of EPSPs and oscillation exceeds the neuron's threshold θ , a spike is generated. The simulation is stopped at this point and the time (= latency) of the spike is recorded.

We describe input spike trains by a parameterized spike density function. We use the model of Gaussian pulse packets [1] for which spike arrival times follow a normal-distribution:

$$G(t) = N \cdot \frac{1}{\sqrt{2\pi}\sigma} \exp \left(-\frac{(t - t_m)^2}{2\sigma^2} \right) \quad . \quad (3)$$

The three density parameters correspond to the total number of spikes N to arrive at the neuron within the 10 ms time frame, the average arrival time t_m (mean input latency), and the width of the distribution σ (standard deviation of input spike latencies with σ^{-1} as a measure of spike coincidence). We note that the neuron takes three input variables (N , t_m , σ) and maps these to a single output spike. The neural output consists of two parts — whether a spike was generated or not and if yes, the latency of this spike. Neuronal parameters

are taken as fixed and not modified. We changed oscillation parameters to see the effect on the mapping between input variables and output characteristics. We used the following parameter settings: $\tau = 2 \text{ ms}$, $\Theta = 10 \text{ mV}$, $w = 0.1 \text{ mV}$, and $f = 40 \text{ Hz}$.

3 Results

Without the oscillation signal the decision whether the neuron generates a spike or not is independent of the mean input latency t_m . The output latency can be approximated by t_m plus a fixed offset. Both N and σ have only a small influence on the output latency, but determine whether the threshold is exceeded or not. For our choice of parameters the number of spikes has a greater influence on the maximum value of the membrane potential (maximum over time) than the width of the pulse packet. The number of spikes and the variance of input spike latencies are decoded in the neuron and a two-dimensional “threshold” operation (Fig. 2) determines whether a spike is emitted or not. If a spike is emitted its latency will encode the average latency of incoming spikes.

When adding the oscillation signal the neuron’s maximum membrane potential value becomes very sensitive to t_m . Therefore the spike generation process is latency dependent. The relative weighting of t_m compared to N and σ can be scaled up and down by increasing or decreasing the absolute value of the

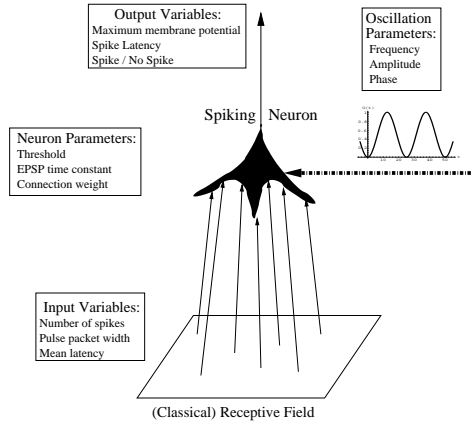


Fig. 1. The network element under investigation. A spiking neuron receives spikes from neurons within its receptive field and is furthermore subjected to a membrane potential oscillation.

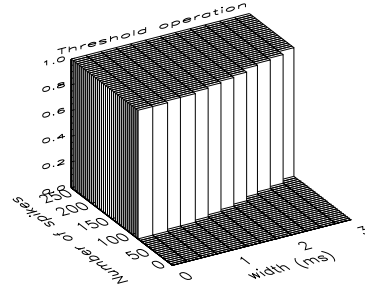


Fig. 2. Spike generation as a function of pulse packet width and number of spikes. The oscillation signal is disabled. The neuron spikes (output 1) if the number of spikes is high and the width of the pulse packet low enough.

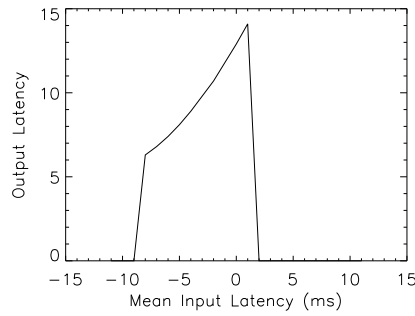


Fig. 3. Output latency as a function of the mean input latency relative to the peak of the oscillation signal. Parameters: $N = 200$, $\sigma = 1 \text{ ms}$, $A = -18 \text{ mV}$. Values of zero indicate that no spike was generated.

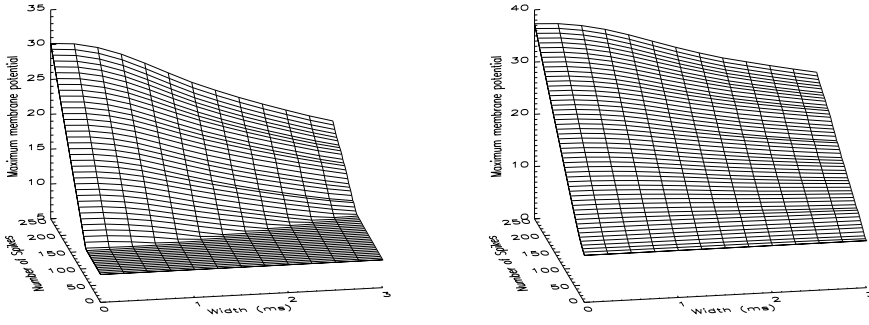


Fig. 4. Maximum membrane potential value as a function of pulse packet width σ and spike number N for mean input latencies of -5 ms (*left*) and $+5\text{ ms}$ (*right*) relative to the minimum of the oscillation. Results are for a positive oscillation with 9 mV amplitude.

oscillation amplitude. For a strong oscillation signal (excitatory or inhibitory) both the generation of an output spike and the latency of this spike depend strongly on input latencies. The neuron can thus decode input latencies. The output latency is still largely controlled by t_m but in a non-linear manner (Fig. 3). The maximum membrane potential is determined by all three input variables (see Fig. 4). A very interesting finding is the very strong dependence of the latency decoding mechanism on the phase relation between the pulse packet and the oscillation. If the pulse packet arrives in the rising phase of the oscillation, higher input latencies will lead to a higher maximum membrane potential (read: the later the better). If spikes arrive in the decreasing phase of the oscillation, earlier arriving spike will generate a higher membrane potential.

4 Summary and conclusion

We have used short pulse packets of spikes as inputs for biologically feasible spiking neurons in the visual cortex. These pulse packets are short enough to fit temporal constraints put forward by [3,8] for the fast feedforward recognition in mammals. We also added an oscillatory membrane potential modulation that mimics (evoked) gamma oscillations. Under these conditions the neurons input-output relation does not conform to standard models of neural operation. Instead we find that the neuron's output is highly sensitive to all three input parameters that characterize the pulse packet. Information can be encoded and decoded in all three of these parameters. Probably the most efficient variable to encode information in is mean input spike latency, at least when the oscillation is strong. The neuron can very efficiently encode the mean input latency in its output latency and also make a latency dependent threshold operation. Spike latencies can play a role analogous to firing rates in classical models. However, latencies are far more efficient in terms of processing time, information capacity per spike and energy consumption.

References

- [1] Markus Diesmann, Marc-Oliver Gewaltig, and Ad Aertsen. Characterization of synfire activity by propagating "pulse packets". In J. Bowers, editor, *Computational neuroscience: trends in research*, pages 59–64, San Diego, CA,

1996. Academic Press.
- [2] J. J. Hopfield. Pattern recognition computation using action potential timing for stimulus representation. *Nature*, 376:33–36, 1995.
 - [3] C. Keysers, D.-K. Xiao, P. Földiák, and D. I. Perret. The speed of sight. *Journal of Cognitive Neuroscience*, 13(1):90–101, 2001.
 - [4] Werner Kistler, Wulfram Gerstner, and J. Leo van Hemmen. Reduction of the Hodgkin-Huxley equations to a single-variable threshold model. *Neural Computation*, 9(5):1015–1045, 1997.
 - [5] Edgar Körner, Marc-Oliver Gewaltig, Ursula Körner, Andreas Richter, and Tobias Rodemann. A model of computation in neocortical architecture. *Neural Networks*, 12(7–8):989–1005, 1999.
 - [6] M. Schürmann, C. Basar-Eroglu, and E. Basar. Gamma responses in the EEG: elementary signals with multiple functional correlates. *Neuroreport*, 8:1793–1796, 1997.
 - [7] C. Tallon-Baudry and O. Bertrand. Oscillatory gamma activity in humans and its role in object representation. *Trends in Cognitive Sciences*, 3(4):151–162, 1999.
 - [8] Simon Thorpe, Arnaud Delorme, and Rufin Van Rullen. Spike-based strategies for rapid processing. *Neural Networks*, 14(6–7):715–725, 2001.