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State space analysis of synchronous spiking in cortical neural networks

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Abstract

Recent proposals that information in cortical neurons may be encoded by precise spike timing have been challenged by the assumption that neurons in vivo can only operate in a noisy fashion, due to large fluctuations in synaptic input activity. Here, we show that despite the background, volleys of precisely synchronized action potentials can stably propagate within a model network of basic integrate-and-fire neurons. The construction of an iterative mapping for the transmission of synchronized spikes between groups of neurons allows for a two-dimensional state space analysis. An attractor, yielding stable spiking precision in the (sub-)millisecond range, governs the synchronization dynamics.

Keywords: Spike synchronization; Integrate-and-fire; Synfire chain; Feed-forward; Pulse packets

1. Introduction

Recently, evidence is accumulating that cortical neurons in vivo are capable of firing action potentials with high temporal accuracy. Independent evidence for precise spike timing in cortical neurons also comes from intracellular recordings in vitro. But,

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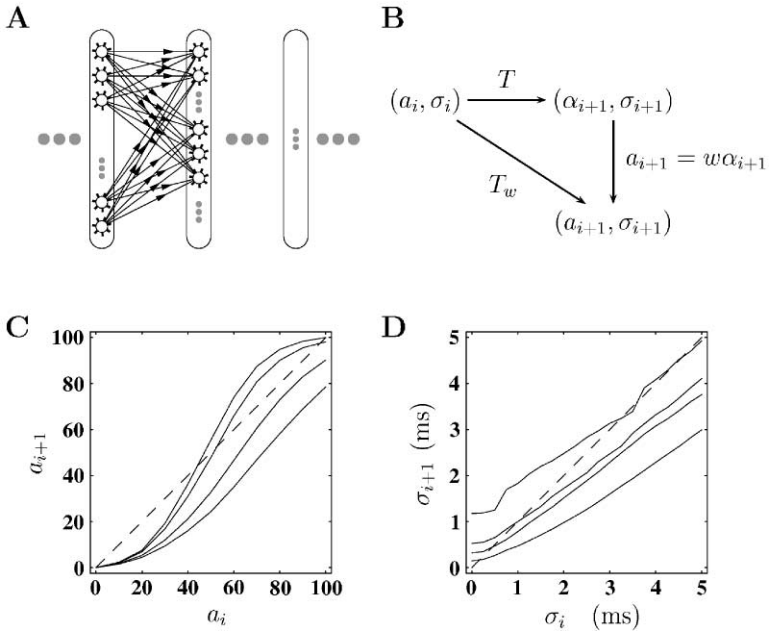

Fig. 1. (A) Locally feed-forward network structure: (1) Convergent synchronous input from a group of neurons (left group) causes transients in target neurons. (2) Neurons receiving shared input (right group) due to divergence tend to generate simultaneous spikes. (B) Transmission function T combined with the network structure w define an iterative mapping T_w. (C) a-section of T_w for different levels of input spread (from left to right $\sigma_i = 0, 1, 3, 5$ ms, $w = 100$). (D) σ-section of T_w and T for different input spike numbers (from top to bottom $a_i = 45, 65, 75, 115$).

can an instance of synchronous spiking be successfully propagated by subsequent groups of cortical neurons? The aim of this study is to investigate under which conditions a group of cortical neurons can engage in precisely coordinated spike timing, and to explore whether such conditions are feasible in the cortical network.

To address these questions, we studied the fine-grained temporal response properties of integrate-and-fire neurons. We focused on spike responses to transient membrane potential excursions, implied by physiological findings. Interestingly, the occurrence of transients can be interpreted in terms of local network structure (Fig. 1A). Thus, a "locally feed-forward" network where a group of neurons projects to another group of neurons in an all-to-all fashion can in principle propagate synchronous activity from the first group to the second group. Repeating this arrangement, the second group in turn can act as source of synchronous input to a consecutive group (synfire chain [1]). "Pulse packets" are used [2] to quantify the degree of synchrony in spike volleys propagating in this structure. A pulse packet is a spike volley characterized by two parameters: activity a and temporal dispersion σ. Activity is defined as the number of spikes in the volley; their temporal dispersion is measured by the standard deviation of the underlying pulse density. Examples of the

types of activity occurring in such a network are shown in [4] (this volume). In an earlier contribution [2] we studied the response of a cortical model neuron to pulse packets in the presence of background activity and introduced the neuronal transmission function T for transient input activity. T is defined by the transformation of the input pair $(a_{\mathrm{in}},\sigma_{\mathrm{in}})$ into the output pair: response probability α_{out}, and temporal spread of the response spikes σ_{out}. In numerical simulations the input is generated by drawing a_{in} spike times from a Gaussian distribution of width σ_{in}.

2. State space analysis

In the present work we computed T for a further reduced I&F model to demonstrate that the described effects are generic to this class of models. Post-synaptic currents (PSCs) are modeled as α-functions.

$$A = \begin{bmatrix} -\frac{1}{\tau_\alpha} & 0 & 0 \\ 1 & -\frac{1}{\tau_\alpha} & 0 \\ 0 & \frac{1}{C} & -\frac{1}{\tau_{\mathrm{m}}} \end{bmatrix}, \tag{1}$$

$$P(h) = \begin{bmatrix} \mathrm{e}^{-h/\tau_\alpha} & 0 & 0 \\ h\mathrm{e}^{-h/\tau_\alpha} & \mathrm{e}^{-h/\tau_\alpha} & 0 \\ \frac{1}{C}\left(\frac{\mathrm{e}^{-h/\tau_{\mathrm{m}}} - \mathrm{e}^{-h/\tau_\alpha}}{(1/\tau_\alpha - 1/\tau_{\mathrm{m}})^2} - \frac{h\mathrm{e}^{-h/\tau_\alpha}}{(1/\tau_\alpha - 1/\tau_{\mathrm{m}})}\right) & \frac{1\,\mathrm{e}^{-h/\tau_{\mathrm{m}}} - \mathrm{e}^{-h/\tau_\alpha}}{C\,(1/\tau_\alpha - 1/\tau_{\mathrm{m}})} & \mathrm{e}^{-h/\tau_{\mathrm{m}}} \end{bmatrix}, \tag{2}$$

$$\tau_{\mathrm{r}}(h) = \max\left(\left\lfloor \frac{\tau_{\mathrm{r}}}{h} + \frac{1}{2} \right\rfloor - 1, 0\right). \tag{3}$$

Eq. (1) is the coefficient matrix of the subthreshold dynamics $\dot{y} = Ay$, where the first two components of y describe the sub-system generating the PSCs and the third component the membrane potential. If input spikes are restricted to occur on a temporal grid $t = hk$, the sub-threshold dynamics can be integrated by repeatedly applying the propagator matrix $P(h) = \mathrm{e}^{Ah}$ of the system Eq. (2) and changing the state according to the incoming events in each time step. See [5] for a detailed description of the general approach and the particular system. After-spike-effects of the I&F model can be consistently incorporated into the discrete time simulation scheme (Fig. 2). At low spontaneous firing rates, the effects of after hyperpolarization and adaptation on T are small. Therefore, we only included an absolute refractory period τ_{r} during which the membrane potential is clamped to the resting level to prevent unrealistically short spike intervals. Eq. (3) is the discrete time version of τ_{r}. Including the detection of threshold crossings (Fig. 2), however, simulation on the grid becomes an approximative method. The pitfalls of approximate numerical integration of such

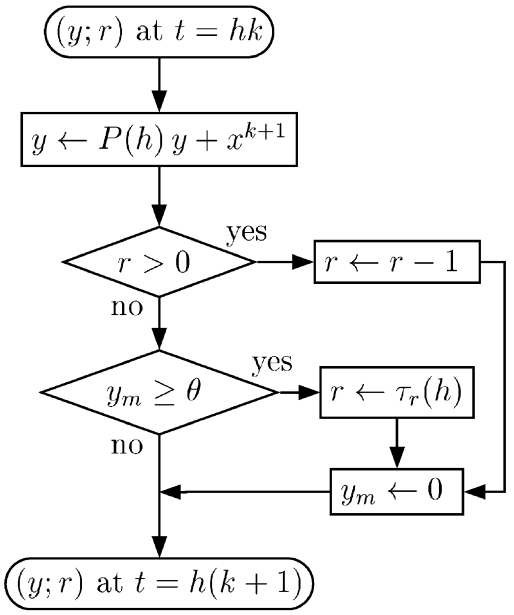

Fig. 2. The state of the neuron is fully described by vector y for the sub-threshold dynamics and integer r measuring time in the absolute refractory period $\tau_r(h)$. Time is advancing in discrete steps h. A constant propagator matrix updates the sub-threshold dynamics (this can be done in place because $P(h)$ is lower triangular). Point events arriving at $t = h(k+1)$ cause changes x^{k+1} which add linearly to the state. Two state variables (y_1, y_2) are necessary to describe the effect of all synaptic inputs. y_m is the membrane potential. A network of such point event exchanging units is consistent if the minimal delay of interaction is h.

systems are discussed in [5]. In addition to pulse packet input, the model neuron is supplied with uncorrelated Poisson input from 20,000 background neurons (88% excitatory, 12% inhibitory). Rates are adjusted to obtain a membrane potential y_3 with mean about 7 mV below threshold ($\theta = 15$ mV) and a standard deviation of 2.5 mV. Membrane time constant is $\tau_m = 10$ ms and capacity $C = 250$ pF. All post-synaptic potentials (PSPs) have an amplitude of 0.14 mV and a rise time of 1.7 ms.

We recently found [3] that the ability of the neuron to support spike synchronization cannot be judged from the transformation T alone. The network architecture has to be incorporated to answer this question. Since each neuron responds to an incoming pulse packet with at most one spike, stable propagation of synchronous spike volleys inevitably requires activation of successive, sufficiently large groups of neurons. For a group of identical, independent neurons, the distribution of response spikes to an input pulse packet is identical to the response distribution for a single neuron. The expected number of response spikes in a group equals the single neuron response probability, multiplied by the group size w. Thus, using T we can construct an iterative mapping T_w (Fig. 1B) describing the transformation of (a,σ) from one group to the next. The iterative mapping for the model neuron described above, choosing $w = 100$ is shown in Fig. 1C and D. Assuming that the group's response to

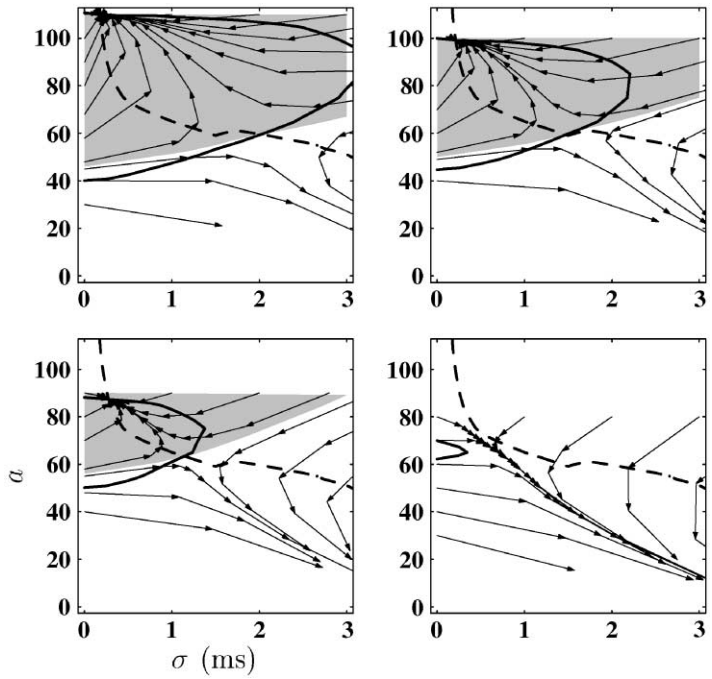

Fig. 3. State space portrait of dynamic variables a and σ. Group size w decreasing from left to right and from top to bottom $w = 110,100,90,80$. Gray area indicates the basin of attraction. The a-isocline (loci of horizontal flow, thick solid curve) depends on w. The σ-isocline (loci of vertical flow, dashed curve) remains unchanged.

a spike volley is adequately described by (a, σ) (see [4], this volume), we can capture the evolution of synchronous spiking activity in a chain by repeatedly applying T_w. Thus, the evolution of synchronous activity is described by a trajectory in the two-dimensional state space. Apart from the trivial fixpoint at vanishing activity, the state space portrait can exhibit two fixpoints (Fig. 3): an attractor and a saddle point. A separatrix divides the state space into two regimes. In the basin of attraction, all trajectories converge into the attractor. A spike volley starting anywhere inside this regime rapidly reaches a stable configuration with sub-millisecond dispersion and close to full group activation. The finite rise time of the PSP limits the precision of spike timing. Volleys starting outside the stable regime decay after only few stages: too weak or too dispersed activity rapidly dies out. Neither the relation between input and output activity alone, nor between input and output jitter alone (Fig. 1C and D) determines whether synchronous activity survives. An initial increase in temporal spread may still support stable propagation, provided that the number of spikes in the volley is large enough. Conversely, synchronous activity may still vanish with an initial decrease in dispersion, unless the volley is large enough. Thus, the system dynamics are governed by the interaction of the two state variables.

To determine how many neurons in a group are needed to guarantee that synchronous activity survives, we examined how the structure of the state space depends on the group size w (Fig. 3). For decreasing groups size, the two fixpoints approach each other, until at some critical value they merge into a saddle node. Below this value, no fixpoint exists: all trajectories lead to extinction. Hence, w is a bifurcation parameter of the system. This lower bound is essentially determined by the ratio of the distance from mean membrane potential to spike threshold and the PSP amplitude.

3. Conclusions

The results obtained for the reduced model are practically identical to the ones reported in [3]. This demonstrates that the described effect is a generic property of I&F dynamics. Firing probability in the attractor is close to unity because of the low spontaneous firing rate and the fast recovery from reset. The attractor describes a stationary configuration of activity in (a,σ)-space. However, unlike the Hopfield attractor, this attractor describes a dynamic activity configuration in neuron space: different neuron groups, one after the other, contribute single spikes to the propagating synchronous wave. The basin of attraction guarantees robustness of the propagating synchrony against perturbations exceeding the response variability accounted for by the transmission function. In the stable state, essentially all response spikes in a volley fall within ± 1 ms. This temporal precision is consistent with the accuracy of observed spike patterns in cortical recordings. Thus, precise synchronous firing of cortical neurons is feasible, in spite of a membrane time constant of 10 ms or more. We conclude that the cortical network may indeed provide the substrate for computation on the basis of precise spike timing.

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