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Effects of noise on recurrence in networks of spiking neurons

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Abstract

We use mathematical analysis and numerical simulations to study the effects of recurrent synaptic connections in neural networks. These recurrent effects can be analyzed by expanding a spike probability function about the membrane potential in the absence of connections. This expansion can be written as a series of loops representing the recurrent signal transmission. Conditions for convergence of the series reveal the number of loops that are significant for the system's dynamics. This method is applied to a pair of mutually coupled spiking neurons to show how the membrane potential and cross-correlation function is changed by recurrence.

Keywords: ■; ■; ■

1. Introduction

Recurrent connections are found in many biological neural systems. One type of recurrence arises from *lateral* connections between neurons within the same layer of processing [7]. The neurons within a layer are often interconnected, and the modulation of neighbors within the layer is an integral part of processing the sensory information. However, if these connections are too strong, then instabilities will develop that can be disastrous for sensory processing.

Another example of recurrence is due to *feedback* from layers at a higher level of processing leading to modulation of input layers [5]. The feedback gain itself must be modulated, because nonlinearities inherent in feedback systems can lead to

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uncontrollable consequences. Feedback control may improve feature identification by modulating sensory input, not by washing out the sensory information with feedback dynamics. We show in the following that the addition of noise can reduce the effects of recurrent nonlinearties by reducing the number of traversals that a signal can survive against noise.

Many studies of recurrence have concentrated on nonlinear effects such as bistability [3,4]. When two neurons are mutually coupled with strong synaptic connections, the output of the neurons has two stable states; one neuron is firing while the other is silent for inhibitory connections. Under certain conditions, the two neurons can switch between the two states. This type of neural dynamics is common in central pattern generators of invertebrate systems. By flipping between stable states, the system can generate a rhythmic output [9].

However, such bistable dynamics can be detrimental to sensory systems where lateral recurrence is used to sharpen contrasts. Nonlinear, bistable dynamics would blind the system with strong internal dynamics. In the mammalian cerebral cortex, it is likely that the nonlinear regime is a pathological state [1].

Methods of mathematical analysis are needed to determine how system characteristics, such as noise and synaptic strength, affect the dynamics of recurrence. Numerical methods have proven useful to find parameter sets where bistability and other nonlinear effects appear in specific models. Bifurcation analysis has been useful to determine the states of dynamical systems and analyze state changes [6]. However, many biological neural systems appear to have an equilibrium level of activity that is enforced by activity dependent mechanisms [8]. The presence of equilibrium fixed points in neural activity suggests that we should develop methods to compute corrections to neural activity arising from interconnectivity within neural networks.

In the following, we construct a model of synaptically coupled spiking neurons. Each neuron receives a non-adaptive input that maintains a baseline membrane potential resulting in a constant spike output rate. Gaussian noise is added to the membrane potential of the model neurons to randomly vary the instantaneous spike rate and simulate the presence of unpatterned synaptic inputs. We then expand the spike probability of one of the neurons about the baseline pike probability in the absence of the recurrent connections, and compute the correction due to the presence of the second neuron. The expansion is ordered in terms of recurrent loops so that the influence of information travelling through multiple recurrent loops becomes is quantified.

2. Model of coupled neurons

To represent the neurons, we use a spike-response model [2] with no relative refractory period. Time is discretized so that $t_n = n(\Delta t)$, with n an integer. The probability of a spike during the time interval t_n is a threshold (sigmoid) function of the membrane potential, $V(t_n)$. For neuron 1 in Fig. 1, with threshold θ, and noise parameter μ, the spike probability is given by the expression

$$f_1(t_n) = \frac{1}{1 + \exp[-\mu(V_1(t_n) - \theta)]}, \tag{1}$$

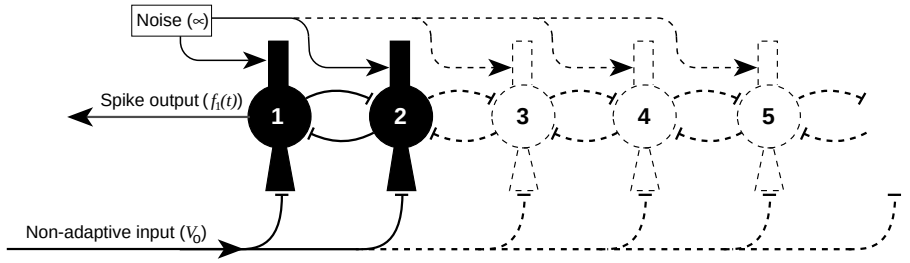

Fig. 1. Recurrent synaptic connectivity between model neurons. The model spiking neurons generate spikes that are the postsynaptic events coupling pairs of neurons. Each synaptic connection contributes a psp to the membrane potential represented by an psp waveform weighted by s. The text focuses on the first two neurons in the network.

representing the variability due to the Gaussian noise. The instantaneous spike frequency is obtained by multiplying the spike probability by the maximum spike frequency.

The membrane potential is the sum of all external inputs for each neuron. The baseline membrane potential maintained by non-adaptive inputs is represented by the term V_o. Synaptic connectivity is represented by weighted postsynaptic potential (psp) waveforms, $\varepsilon(t_n)$. The contribution from neuron 2 is represented by the spike probability convolved with the psp waveform, and weighted with the synaptic strength s. For an excitatory synapse $s > 0$, and for an inhibitory synapse, $s < 0$. The membrane potential of neuron 1 is

$$V_1(t_n) = +V_o + s\sum_m f_2(t_m)\varepsilon(t_m - t_n), \tag{2}$$

where the last term is the contribution from the activity of neuron 2. This formalism easily generalizes to any number of neurons in the network.

3. Results

The starting point of our analysis is to expand the spike probability about the spike probability that would result in the absence of recurrent synaptic connections, $\hat{f} = f_1(U_1(t_n))$,

$$f_1(t_n) = \hat{f}_1 + \mu(\hat{f}_1 - \hat{f}_1^2)(V_1(t_n) - U_1(t_n)) + \cdots . \tag{3}$$

The solution for the membrane potential of neuron 1 without the input of neuron 2 is the baseline membrane potential

$$\langle U_1(t_n)\rangle = V_o. \tag{4}$$

To compute the effects of recurrence, we include neuron 2 in our calculations and expand the membrane potential of 1 about the membrane potential in the absence or

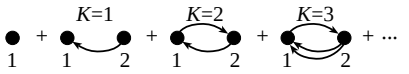

Fig. 2. Graphical representation of the loop expansion.

recurrence,

$$\langle V_1(t_n)\rangle = \langle U_1(t_n)\rangle + s\hat{f} + s\mu(\hat{f} - \hat{f}^2)\sum_m (\langle V_2(t_m)\rangle - V_o)\varepsilon(t_n - t_m). \tag{5}$$

Next, we expand $\langle V_2(t_m)\rangle$ about the baseline potential and collect terms to arrive at the recurrent loop expansion:

$$\langle V_1(t_n)\rangle = \langle U_1(t_n)\rangle + s\hat{f}\sum_{K=1}^{\infty}(s\hat{f})^{K-1}\mu^{K-1}(1-\hat{f})^{K-1} \times \left[1 + \mu(1-\hat{f})\sum_{i_1}\cdots\sum_{i_K}(U_a(t_n) - V_o)\varepsilon(t_{i_{K-1}} - t_{i_K})\cdots\varepsilon(t_n - t_{i_1})\right], \tag{6}$$

where

$$a = \left\{\begin{array}{ll} 1 & \text{if } K \text{ is odd} \\ 2 & \text{if } K \text{ is even} \end{array}\right\}. \tag{7}$$

Each term in this loop expansion represents the contribution to the membrane potential of 1 by going around the loop Fig. 2.

The first term has no recurrent influence, the second term ($K = 1$) includes only the contribution of only neuron 2 on neuron 1 as if neuron 2 had no other synaptic contact with neuron 1. The third term ($K = 2$) is the contribution of neuron 2 on 1 caused by neuron 1 on 2, etc.

The terms of expansion are weighted by the factor $[s\mu\hat{f}(1-\hat{f})]^K$. Thus, the series converges if the system has low synaptic strength or high noise. Low synaptic strength represents the weak coupling limit of the network. The convergence due to high noise is because the transmission of information is lost in the noise while traversing the recurrent loop. The series also converges faster if the fixed point is far from threshold because neurons are more sensitive to modulation near threshold.

Comparing the loop expansion with simulations: We may use this expansion to compute corrections to the neural dynamics due to coupling with other neurons in the network. Using reasonable values for the parameters of the model (see Fig. 3), the corrected membrane potential is

$$\langle V_1(t_n)\rangle = V_o + s\hat{f} + s^2\hat{f}^2\mu(1-\hat{f}) + s^3\hat{f}^3\mu^2(1-\hat{f})^2 + \cdots . \tag{8}$$

The membrane potential is increased for excitatory recurrence, and decreased for inhibitory recurrence. The correction to the baseline membrane potential is linear for recurrent synaptic weight values that are weak, or for noisy networks.

For stronger synaptic weights, the simulated results match the loop series closely up to $s \approx \pm 20$ (arbitrary units). When the synapses are excitatory, the higher order

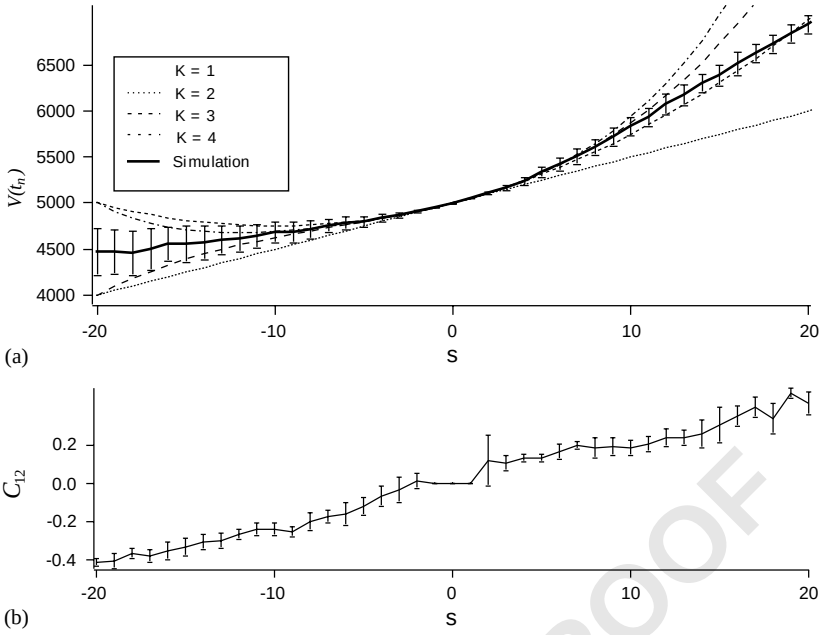

Fig. 3. Numerical simulation of the recurrent model: (A) The traces showing the membrane potential, V (arbitrary units), as a function of the recurrent synaptic weight, s. The solid is the result of a simulation and the broken lines are the series for the first two, three, four, and five terms and (B) the average of the cross-correlation function for five simulation runs. Parameter values: $\mu = 0.5$, $\hat{f} = 0.5$. The error bars represent one standard deviation.

corrections deviate more quickly than inhibitory synapses from the simulation result. This deviation is due to the saturation of the spike probability that was linearized in the first step of the analysis.

In the case of inhibitory synapses, the higher order terms seem to extend the accuracy of the prediction to stronger synaptic weights. However, at strong inhibition the membrane potential becomes bistable, and averaging over both states would yield a large variance in the simulation. The problem here is the distribution of the membrane potential is no longer Gaussian and higher order moments limit the reliability of the analysis.

How strong is 'weak'? In the noisy, weak limit, the first few terms of the loop expansion yield a good quantitative predictor of the membrane potential of neurons in a network with recurrent loops. To investigate the effect of one neuron on the firing rate of the other, we calculated the cross-correlation coefficient, C_{12}, for the two neurons in the simulation

$$C_{12} = \frac{\langle (V_1 - \langle V_1 \rangle)(V_2 - \langle V_2 \rangle) \rangle}{\sigma_1 \sigma_2}, \tag{9}$$

where, $\sigma_i^2 = \langle (V_i - \langle V_i \rangle)^2 \rangle$.

The averaged results of five simulations is shown in Fig. 3B. For the range of synaptic strengths where there is close agreement with the first few terms of the loop expansion, $-20 < s < 20$, the correlation coefficient value is, $-0.2 < C_{12} < 0.2$. This corresponds to a modulation of the spike probability of over 60%. Such a level of modulation could contribute significantly to the range of fluctuations observed in cortical neurons.

Another result of this analysis is that under these "weak and noisy" conditions, few neurons contribute to modulate the activity of a given neuron in a network. The match between the loop expansion and simulations tells us that neural modulations traverse only a few synapses in lateral recurrence, so that distant neurons have no effect. We have compared the graphs similar to Fig. 3A for networks of 4 and 50 neurons, and found the curves (V vs. s) to be within a standard deviation. This also implies that feedback connections will modulate those neurons across few.

4. Conclusion

The analysis and simulations presented here show that dynamical changes caused by recurrence can be calculated using an expansion in terms of signal transmission through recurrent loops. The loop expansion reveals what system parameters are responsible for convergence allowing one to truncate the series. This method can be used to estimate an upper bound on lateral synaptic connections in biological networks that do not exhibit nonlinear effects of recurrence.

The conclusion that the loop expansion series converges quickly for networks with high noise has consequences for sensory processing systems. Feedback recurrence in biological systems, such as cortical sensory systems, serve a first order role in modulating sensory input patterns so that only the first loop is important in the processing of sensory information.

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