Noise Delays Onset of Sustained Firing in a Minimal Model of Persistent Activity

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1   Introduction

The effects of random currents on the firing behavior of real and model neurons have received a considerable amount of attention in neurobiology and physics literature [1, 2, 3, 4, 5, 7]. Experimental work has identified noise induced signal amplification and resonance in a number of preparations e.g. [3]. In the Hodgkin-Huxley model pre-cursors of the Hopf bifurcation are revealed by the action of random currents [5]. In this sense noise "advanced" the bifurcation. Similar effects have also been found in a generic saddle-node driven oscillator where noise advances the onset of oscillations and upregulates the mean frequency [14, 15]

Modelling studies of noise effects on models of spatial working memory ("bump" attractors) have found that random noise tends to destabilize the position of the activity bump and induce a diffusion-like motion (Wang, personal communication). Laing and colleagues [18] looked at bumps of sustained activity destabilized to traveling waves due to spike frequency adaptation and reported that noise has a curious stabilizing effect: it reduces the velocity of the bump.

In view of these reports we decided to examine what random noise would do to a minimal neural circuit that is capable of synaptically evoked sustained activity. Here, we look in detail at the effects of noise on the onset of synaptically sustained firing in circuits of excitable neurons where activity occurs purely due to the recurrent synaptic interactions. Our finding is rather intriguing since we observe a noise induced **delay** of bifurcation in a purely positively coupled circuit of neural oscillators. Analysis of this system leads us to conclude that the relative width of the attractor basins for the quiescent and persistent asynchronous firing states determines whether noise has a delaying, neutral,

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or advancing effect on the bifurcation. We propose this as a noise dependent mechanism that could stabilize sustained activity by making it more difficult to recruit neurons just at the edges of the sustained bump.

Below we summarize the dynamics of the spiking neuron used in this circuit (the θ-neuron), and analyze the case of two coupled cells in the regimes of weak and strong excitatory coupling.

2 The Model: θ-neuron

The θ-neuron model [19, 20, 21] is derived from the center manifold reduction for type I spike generation dynamics (saddle-node bifurcation at a critical input current value). In the present case of the saddle-node bifurcation which is the simplest bifurcation type, this yeilds:

$$\frac{dx}{dt} = \lambda + x^2. \tag{1}$$

Here, the bifurcation parameter λ is the input to the neuron, x records its activity. One can change to a phase variable θ that is 2π-periodic via $x = \tan(\frac{\theta}{2})$; θ is then a variable with domain the unit circle S^1, and a spike now corresponds to a period of θ:

$$\frac{d\theta}{dt} = (1 - \cos(\theta)) + (1 + \cos(\theta))\lambda. \tag{2}$$

Due to the nonlinearity of the transformation from x to θ, the input λ is no longer additive. The bifurcation occurs at $\lambda = 0$, and there is a single degenerate rest point, namely $\theta = 0$. The sensitivity to the input λ is highest at $\theta = 0$ and lowest at $\theta = \pi$. There is no rest point when λ is positive. In this case, θ continually increases, and the neuron fires perpetually. When λ is negative, there are two rest points; a stable one denoted by θ_r and an unstable one $\theta_t > \theta_r$. If θ is larger than θ_t it increases until it completes a period and comes to rest at $\theta_r + 2\pi$ which is identified with θ_r as we are working on the unit circle S^1. Thus, if the phase is above the threshold value θ_t, a spike occurs and the neuron returns to rest. In general, the input can be decomposed as $\lambda = \beta + \sigma\eta + I(t)$, where β is a constant bias, $I(t)$ is a time dependent input, while η is (white) noise and σ its intensity. Sufficiently strong noise can push the phase θ beyond the threshold θ_t causing intermittent firing (Figure 1C).

In this report we consider a small circuit of two θ-neurons coupled reciprocally with excitatory synapses, with total input to a cell being:

$$\lambda_i = \beta_i + \sigma_i\eta_i + gs_is_i \tag{3}$$
$$ds_{ij}/dt = syn(x_i) * (1 - s_{ij}) - s_{ij}/tau \tag{4}$$
$$syn(x_i) = a * exp(-20 * (1 - cos(x_i - th))) \tag{5}$$

Where s_{ij} gives the dynamics of the synaptic coupling from cell i to cell j, and x_i is the presynaptic state variable, which triggers the synapses when

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the presynaptic cell fires a spike. For the numerical simulation we picked the duration of the synapse to be comensurate with fast AMPA-type glutamatergic excitatory synapses. Threshold th was set at 3, $a=1$, $tau=2$. The maximal synaptic strength gs_i is a free parameter.

3   Numercial Results

Figure 1A, upper trace shows the firing patterns of two cells whose spiking behavior results from mutual excitatory synapses. The cells are initially quiescent (they are not intrinsically spiking) and their activity results from an transient input to one cell. Activity can be terminated by small levels of noise (Figure 1A, middle trace), whilst increased noise levels cause intermittent firing (Figure 1A, lower trace).

Figure 1B plots the probability (M_1) of observing firing in the last 200 msecs of a 2000 msec run over an ensemble of 1000 sample paths as a function of the the strength of the synaptic coupling (g_s). In the noise free circuit, g_s^* is the critical value of coupling above which sustained firing occurs (i.e. $M_1=0$ for $g_s < g_s^*$, $M_1=1$ for $g_s \geq g_s^*$). At small noise levels (Figure 1B, traces 1,2), increasing the noise amplitude progressively shifts the curves of M_1 to the right with respect to the noise-free case. This behavior is surprising as addition of small amounts of noise for an autonomously spiking θ-neuron (or a generic saddle-node oscillator) induces the opposite effect - noise advanced bifurcation (see [21, 14]). Above a critical noise value, the onset of sustained firing appears to be shifted back to the left (Figure 1B, traces 3 and higher). Thus we can state that the bifurcation is delayed for low noise amplitudes and advanced with higher noise. Figure 1C shows that there is a non-linear relationship between the amount of injected noise and the firing probability. Similar effect is also seen in in a circuit of conductance based, Hodgkin-Huxley models for a pyramidal neuron [10] (simulations not shown).

4   Analysis of coupled neurons

We now consider the situation where we have two neurons (distinguished by subscripts $i = 1, 2$). The dynamics then takes place on the product of two circles, i.e. on a two-dimensional torus T, represented by the square $[-\pi, \pi] \times [-\pi, \pi]$ in the plane, with periodic boundary identifications. We first consider the simple case of two uncoupled, noise-free neurons ($\sigma_1 = \sigma_2 = 0$) with the same bias β. Their dynamics are independent. In the phase plot (Figure 2(i)) the diagonal is always an invariant curve, where the two neurons fire. If $\beta > 0$, both neurons continue to fire, although their phase difference, if not 0 initially, is not constant, due to non-linear effects. If $\beta = 0$, $(0,0)$ is a degenerate rest point (Figure 2(ii)). The two curves $\theta_1 = 0$ and $\theta_2 = 0$ are homoclinic orbits and all flow lines eventually terminate at this fixed point. One or both neurons will spike before returning to rest if their initial phase is between 0 and π.

If $\beta < 0$ (Figure 2(iii)), we have four fixed points - the attractor ($\theta_1 = \theta_2 = \theta_r$), the repeller ($\theta_1 = \theta_2 = \theta_t$), and the two saddles where one of the neurons has its phase at θ_r (rest) and the other one at θ_t (threshold). Some special heteroclinic orbits are given by the straight lines where one of the two neurons stays at θ_t while the other one moves from the threshold to the rest value, spiking if its initial phase was above threshold. All other flow lines terminate at the attractor. We now add an interaction term $s_i g_s$ to the input of neuron i. s_i is considered as the synaptic input from neuron j to neuron i ($i \neq j$) and g_s is the synaptic intensity. A precise equation for s_i can be derived from electrophysiological models, however for our qualitative study we only need the characteristic features that it stays bounded between 0 and 1. Typically, it is peaked near the spike of neuron j, i.e. where $\theta_j = \pi$. With this interaction term, the equation for neuron i is then

$$\frac{d\theta_i}{dt} = (1 - \cos(\theta_i)) + (1 + \cos(\theta_i))(\beta + g_s s_i + \sigma \eta). \tag{6}$$

Since s_i represents the input that neuron i receives from neuron j, s_i should essentially be considered as a function of the phase θ_j of j. Once more, we first consider the situation without noise, i.e. $\sigma = 0$. We also assume that we are in the excitable region, i.e. $\beta < 0$. g_s is positive (excitatory coupling), and so the coupling counteracts the effect of the bias to some extent. N.B. In contrast to the constant bias, the synaptic input to each neuron is time dependent. If g_s is sufficiently small, the qualitative situation is similar to the case with zero coupling, i.e. $g_s = 0$. We still have a heteroclinic orbit from the saddle ($\theta_1 = \theta_t, \theta_2 = \theta_r$) to the attractor ($\theta_r, \theta_r$), although θ_2 is no longer constant, but increases due to the input from neuron 1 before returning to the rest value. (Figure 2(iv)). If g_s reaches a critical value g_s^*, however, the heteroclinic orbit starting at (θ_t, θ_r) no longer terminates at the attractor. The synaptic input advances the phase of neuron 2 to the other saddle point at (θ_r, θ_t) (Figure 2v). Besides two heteroclinic orbits that go from the repeller to the two saddles as before, all other orbits still terminate at the attractor (θ_r, θ_r), for $g_s = g_s^*$. If g_s is increased beyond g_s^*, however, the heteroclinic orbit between the two saddles mutates into a stable attractor (Figure 2(vi)). This corresponds to sustained asynchronous firing of the two neurons. If the phase difference between the two neurons is too small, the dynamics converges towards the double rest point (except in some region in the vicinity of the node), and both neurons stop firing. This happens when the two neurons are close to synchrony as neither cell is sensitive enough to its synaptic input to maintain firing (an effective refractory period). Conversely, if they are out of synchrony, a single spike can induce the second neuron to fire at a time when the first cell is close to rest, and sensitive to synaptic input itself. If g_s is only slightly above the critical value, the basin of attraction of that limit cycle will still be relatively small, but as g_s is increased further, the basin grows in size until eventually it is larger than the basin of attraction of the double rest point. On the basis of the preceding analysis, it is now straightforward to predict the effect of noise. If g_s is only slightly above the critical value g_s^*, a small amount of noise is more likely to kick the dynamics

out of the narrow basin of attraction of the asynchronous limit cycle and into the large basin of the double rest point than vice versa. In effect, a small noise level increases the critical parameter value required for the qualitative transition to sustained asynchronous firing. A larger amount of noise, however, has the potential to move the dynamics from the rest point into the basin of attraction of the asynchronous limit cycle. Once in that basin, the neurons will fire. Thus, for large noise in that regime, one will observe that the neurons will fire, perhaps with some intermissions spent near the double rest point. So, a larger value of noise will cause intermittent periods of sustained firing of the two neurons even at somewhat smaller values of g_s. In effect, it decreases the value of the critical parameter. Thus, we observe a genuinely nonlinear effect of the noise level σ (Figure 1E). For values of the coupling g_s that are substantially larger than the critical value g_s^*, even small amounts of noise have a good chance of perturbing the dynamics out of the attracting vicinity of the double rest point into the attracting region of the asynchronous limit cycle. This will further enhance the sustained asynchronous firing pattern of the two neurons.

5   Conclusions

In this work we report a new and unusual effect of noise in a simple neural circuit. When the sustained firing in the circuit is induced by recurrent excitatory coupling, small noise levels can exert a strong influence on the circuit dynamics, often abolishing the firing. Noise induced delay of bifurcation can therefore occur in a completely positively coupled circuit. The same noise has the exact opposite effect of advancing the bifurcation when it is applied to a single autonomously firing neuron. The paradoxical effect of noise in this circuit can be understood by considering the structure of its phase plane. When the width of the attractor basin for asynchronous firing is small, weak noise can perturb the system into the larger basin of the stable quiescent state. Thus, as the coupling strength increases, the basin of attraction for the sustained firing solution grows at the expense of the quiescent state. In this circuit, low levels of noise ensure that sustained firing can only take place above a critical coupling threshold. Transitions in the opposite direction from the rest-state to a sustained firing state can only occur when noise fluctuations reach a critical value. Above this value, transitions into the firing state begin to counteract transitions into the quiescent state.

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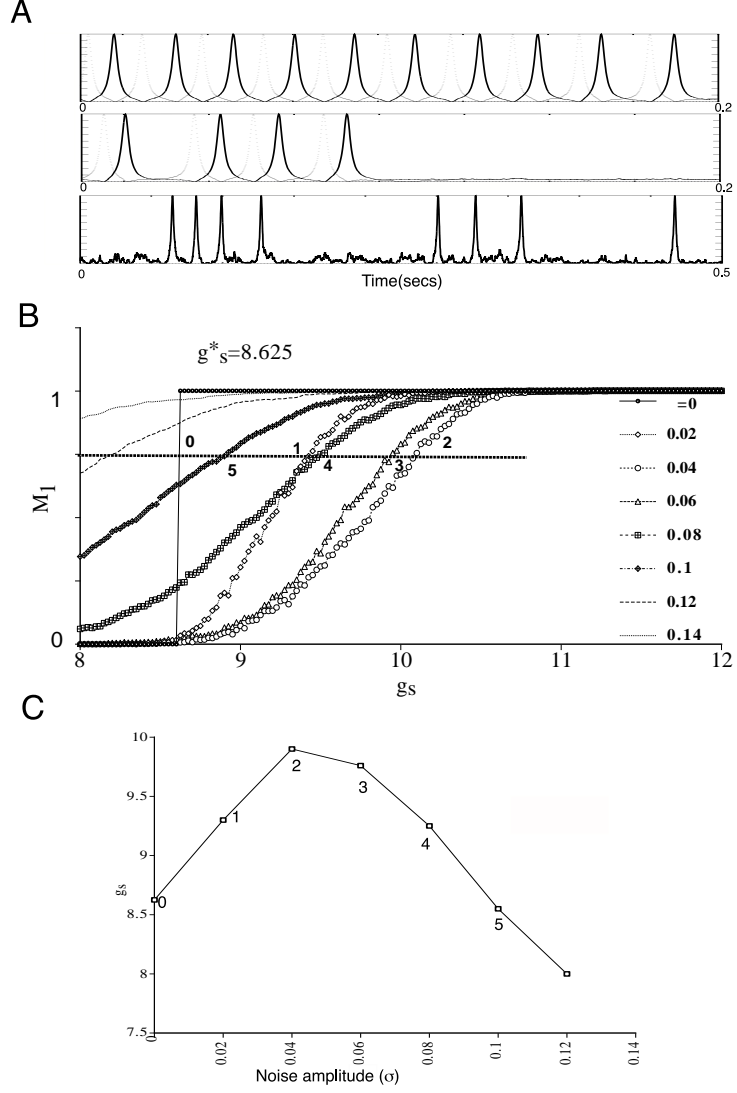
Figure 1: Asynchronous synaptically sustained oscillations in a positively coupled 2-cell circuit. A. Upper trace: sustained firing in the noise free circuit. Middle trace: sustained firing can be terminated by the action of small amplitude noise. Lower trace: larger amplitude noise induces an intermittent firing pattern. Noise injected into the two neurons is completely correlated, but the results are qualitatively identical for uncorrelated noises. B. Increasing noise delays sustained firing for low noise levels (traces 1,2) and advances firing for higher noise levels (traces 3,4,5). The horizontal dashed line and the numbers mark the test points $g_s^{2/3}$ - the values of synaptic coupling at $M_1 = 0.66$. C. Addition of noise has a non-linear effect on sustained firing in this coupled circuit. Here we plot the location of the test points $g_s^{2/3}$
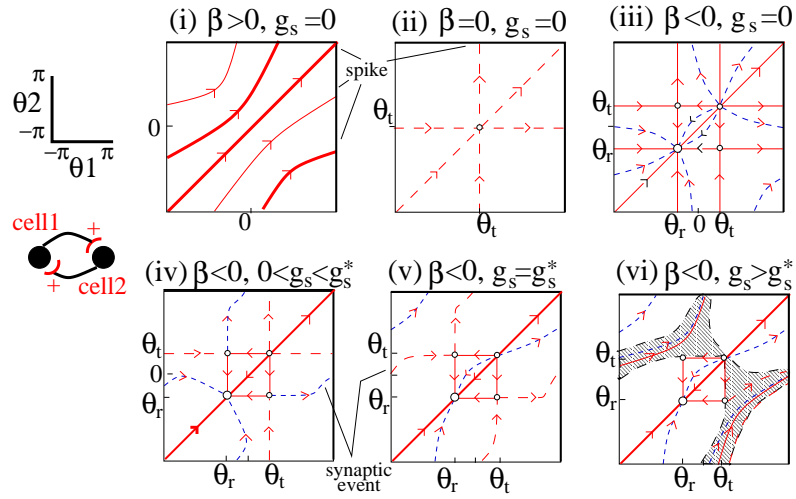
Figure 2: Different states of the network for various values of the intrinsic excitability of the cells, β, and the coupling strength, g_s. Axes plot the phase (θ_1, θ_2) of each cell. See text for details.