Rhythmic Bursting in Networks of Adaptive Spiking Neurons

C. van Vreeswijk†* and D. Hansel‡
†Racah Institute of Physics, Hebrew University
Jerusalem, 91904 Israel
‡Centre de Physique Théorique UPR014 CNRS
Ecole Polytechnique 91128 Palaiseau Cedex, France
Email: carl@fiz.huji.ac.il
hansel@orphee.polytechnique.fr

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1 Introduction

The Central Nervous System (CNS) displays a wide spectrum of macroscopic, spatially synchronized, rhythmic activity patterns, with frequencies ranging from 0.5 Hz (δ rhythm), to $40-80$ Hz (γ rhythm) [1]. The mechanisms involved in the emergence of these rhythms remains a matter of debate. An important issue is the respective contribution, in their emergence, of the cellular, synaptic and architectural properties.

Recent theoretical work has shown that synaptic excitation alone can hardly explain the occurrence of synchrony of neural activity [2]. This has lead to the suggestion that inhibition plays an important role in neural synchrony. Here we examine a different hypothesis in which rhythmic activity emerges from a cooperation between a strong excitatory feedback and a slow

*First Author, to whom correspondence should be addressed
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adaptation current. Since adaptation currents are widely spread in neurons of the CNS we suggest that they can contribute significantly to the rhythmogenesis in the CNS. Our study deals with stimulated and supra-threshold interacting neurons. The case of subthreshold excitatory neurons with adaptation has been recently addressed by Golomb and Amitai [3].

2 Model

We consider a network of N integrate and fire (IF) excitatory, all-to-all coupled neurons with spike adaptation. The neuron i is described by its voltage V_i and by an adaptation current A_i. When it is subthreshold ($V_i < \theta$) its dynamics obeys

$$\tau_m \frac{d}{dt} V_i = -V_i - A_i + I_0 + g_s E(t) \qquad (1)$$

$$\tau_A \frac{d}{dt} A_i = -A_i. \qquad (2)$$

Here I_0 is an external current and the last term describes the network feedback. Without loosing generality, we choose $\theta = 1$, $\tau_m = 1$. We assume a supra-threshold input, i.e. $I_0 > 1$. If at time t, $V_i(t^-) = 1$, neuron i fires, its voltage resets to 0, A_i is increased by an amount g_A/τ_A and the level of synaptic activity, $E(t)$, is augmented by a single synaptic response. If $t_{i,n}$ is the nth time neuron i fires, E is given by

$$E(t) = \frac{1}{N(\tau_1 - \tau_2)} \sum_{i=1}^{N} \sum_{n}^{t_{i,n}<t} \left(e^{-(t-t_{i,n})/\tau_1} - e^{-(t-t_{i,n})/\tau_2} \right). \qquad (3)$$

Throughout we will use $\tau_1 = 0.3$ and $\tau_2 = 0.1$. Assuming τ_m of 10 msec, this corresponds to a rise and decay time of the synaptic current of 1 and 3 msec respectively.

3 Results

Fig. 1A shows the response of a single cell to a constant input. For $t < 0$, $I_0 = 0$ and $V(t) = A(t) = 0$. At $t = 0$ a constant current $I_0 > 1$ is turned on and the neuron starts firing. As the adaptation increases the firing rate diminishes and the cell stabilizes in a periodic firing state. Thus the single model neurons are tonically firing with adaptation. This implies that in a large *asynchronous* network the neurons cannot fire in bursts. Indeed, in an asynchronous state (AS) the firing times of the neurons, are evenly distributed, and the input to the cells is constant (up to fluctuations of order $O(1/N)$).

We analyzed the stability of the AS. There are two ways in which the AS can become unstable.

- For weak adaptation: When the adaptation is weak, g_A is small, and the input I_0 is just above the threshold. For strong synaptic coupling, g_s large, the AS is stable. Decreasing g_s, the AS is destablized through a normal Hopf bifurcation to a *synchronized* state in which the cells continue to fire *tonically.*

- For strong adaptation: For larger g_A, the asynchronous spiking state is stable for small g_s. When g_s is increased, the destabilization occurs through an inverted Hopf bifurcation. Past this bifurcation, the neurons fire in *bursts* as shown in Fig 1B. The neurons' bursts are nearly synchronous, but the spikes are not.

The first scenario is similar to that in networks of IF neurons without adaptation [4]. Here we concentrate on the second scenario. A phase diagram of the network is shown in Fig. 2 as a function of τ_A and g_A. The parameters I_0 and g_s are fixed at values for which the network bursts for sufficiently large g_A. There is a region of bistability, in which the network state can evolve to a synchronized bursting state or an asynchronous spiking state depending on the initial conditions. For very slow adaptation the region of bistability is small, but it is considerable for faster adaptation.

4 Mechanism

In networks of IF neurons without adaptation there is a stable AS in which the neurons fire with a rate $R(I_0)$, for I_0 exceeding some critical input I_c. I_c depends on g_s and if g_s is large enough, I_c is below threshold ($I_c < 1$). In the following, we assume that this condition is satisfied. Since, below threshold, the network can also be in a quiescent state (QS) it is bistable for

$I_c < I_0 < 1$ and for $I < I_c$ it is in the QS. If I_0 decreases slowly (compared to the characteristic time of the network dynamics) and starts from above threshold the network settles in the AS it remains there until I_0 reaches I_c, where it abruptly switches to the QS. If I_0 increases slowly from below I_c the network remains in the QS until I_0 reaches 1, where it jumps to the AS.

We assume now that I_0 is constant, $I_0 > 1$, and that the neurons has a very slow adaptation ($\tau_A \to \infty$). After a transient period following the stimulation, A_i is the same for all the neurons, $A_i = A$. If $A < I_0 - 1$ the network is in the AS, with the cells firing at a rate $R(I_0 - A)$, and A obeys

$$\tau_A \frac{d}{dt} A = -A + g_A R(I_0 - A). \tag{4}$$

For sufficiently large g_A, A increases until it reaches $A_c = I_0 - I_c$ where the network goes to the QS. In the QS, A decreases, satisfying

$$\tau_A \frac{d}{dt} A = -A. \tag{5}$$

When A is sufficiently decreased ($A = I_0 - 1$) the network jumps back to the AS, and A increases again according to Eq. (4). Thus the network switches periodically from AS to the QS. This is illustrated in Fig. 3.

It should be noted that while all cells start to fire at the same time (on the time scale of τ_A), on a time of order 1, short compared to the burst duration the network desynchronizes. Thus the cells are synchronized on the burst time scale, but on the finer time scale of the spikes the cells are not synchronized.

There is no simple way to determine the behavior of the bursting state for finite τ_A, but the above analysis suggests that the cells fire nearly synchronously at the burst onset and desynchronize during the burst. This is confirmed by simulations.

Equations (4) and (5) allow us to calculate the dependence of burst duration and the inter-burst interval on the parameters in the large τ_A limit. For example, in this limit, both durations vary linearly with τ_A. That this is still qualitatively true for relatively small τ_A is shown in Fig. 4.

5 Conclusion

We have examined a simple scenario that can lead to rhythmic and stable collective patterns of neural activity. We have shown that states characterized by a synchronized and slow bursting emerge naturally from a cooperation between excitatory feedback and firing adaptation. The results presented here have been obtained in the framework of identical and all-to-all coupled IF neurons. The bursting this system exhibits is a *network* effect since the neurons cannot fire periodic bursts on their own. It relies on the fact that an excitatory network can display a region of bistability and on a slow process which drags the neurons back and forth in and out of this bistable region. This mechanism is very similar to those that have been suggested for single neuron bursting. However, here the bistability is a network property.

Adaptation currents display a wide spectrum of time constants ranging from tens to hundreds of msec. As shown above, this leads very naturally to rhythmic activity over a wide range of frequencies.

This bursting mechanism is general. In particular, we have checked that it is valid also in all-to-all networks of conductance-based neurons. We have also verified that it is robust to noise, to neuron inhomogeneities and to randomness in the connectivity. Finally, if the network architecture is of the "cortical type" (i.e each neuron is interacting with a finite fraction of its neighbors) collective bursting also occurs. However, if a population of inhibitory neurons is also present in the network, the collective and synchronous bursting state can be destabilized and replaced by a traveling pulse state as has been recently shown[5].

References

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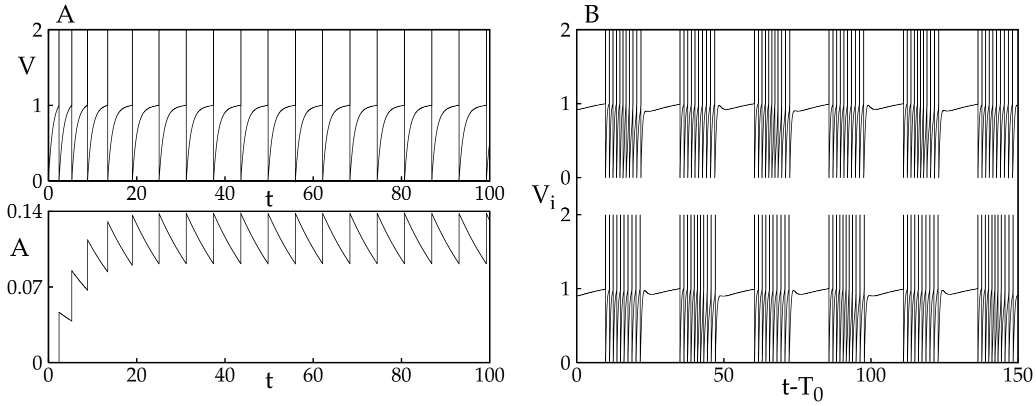

Figure 1: **A:** Response of a single cell to current injection. At $t = 0$ a constant current $I_0 = 1.1$ is injected. Top panel shows the neurons voltage, bottom the adaptation current. **B:** Voltage traces of two neurons in a network that is in the bursting state. Simulation was started at $t = 0$ with random initial conditions. Traces are shown after a time $T_0 = 10000$ has elapsed. Parameters used: $g_A = 0.7$, $\tau_A = 15$ in A, $I_0 = 1.1$, $g_A = 0.4$, $\tau_A = 15$ and $g_s = 0.675$ in B. Time is in units of τ_m.

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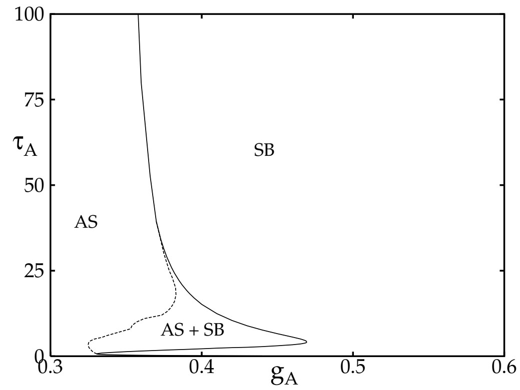

Figure 2: Phase diagram. Dependence of the final state of the network on τ_A and g_A is shown. AS is asynchronized spiking state, SB synchronized bursting state, AS+SB bistable region. Other parameters: $I_0 = 1.1$, $g_s = 0.675$.

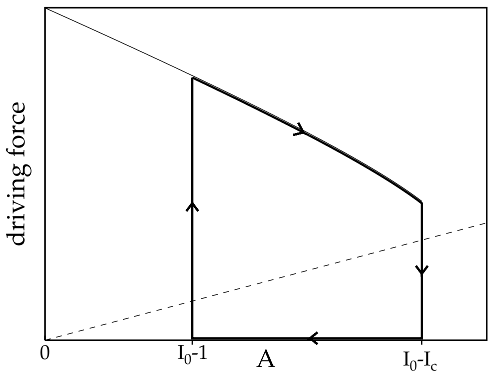

Figure 3: Limit cycle behavior of A in the large τ_A limit. If the network fires asynchronously A is driven to $g_A R(I_0 - A)$ (solid line). This is larger than A (dashed line) A increases. If A exceeds $I_0 - I_c$ the network becomes quiescent and A is driven to 0. When A is less than $I_0 - 1$ the network jumps back to the asynchronous state. A and its driving force periodically traverse a closed trajectory (thick solid line).

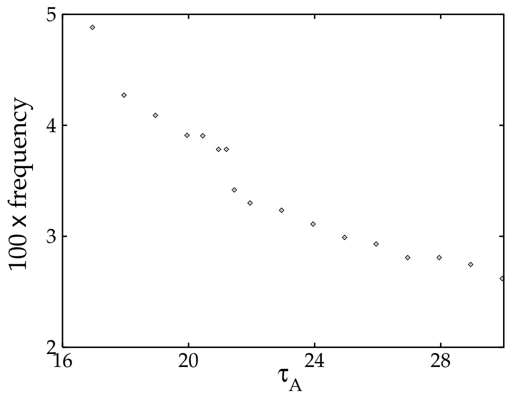

Figure 4: Dependence of the Rhythm on the adaptation time constant. The burst frequency (in units of $1/\tau_m$) is shown as function of τ_A (in units of τ_m). Rhythms were obtained from simulations of a network with 100 neurons with parameters $I_0 = 1.1$, $g_s = 0.675$ and $g_A = 0.45$. There is a qualitative agreement with $1/\tau_A$ dependence predicted by the theory.