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Multiple Phase Clustering of Globally Pulse Coupled Neurons with Delay

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1 Introduction

Recently it was shown that synchronizations of neuronal populations may depend systematically on Gestalt properties of the stimulus [1, 2]. Such temporal structures have gained increased attention when it has been suspected that they might relate to higher brain functions. It has been suggested that synchronization plays an important rôle in object perception [3], and that it might even reflect consciousness [4]. The basic mechanisms leading to synchronization and desynchronization in realistic neuronal groups are still not completely understood. Finite transmission delays and pulselike coupling yield a rich phenomenology including multiple clustering and spontanous desynchronization. Our rigorous analysis shows that these phenomena are generic properties of realistic neuronal groups.

2 Analysis of Synchronization

We consider pulse coupled neurons extending methods from Ref.[6] to the situation of finite transmission delays. The neuron is characterized by a function (e.g. membrane potential) $f(\Phi)$ depending on the time Φ elapsed since its last spike. When $f(\Phi)$ reaches a threshold Θ, the neuron fires and the phase Φ is set to zero. For convenience we choose $f(T=1)=\Theta=1$. The mathematical analysis only requires that the inverse g of f exists and that $f'' > 0$.

The interaction of such neurons is considered to be pulselike with no additional temporal structure. When a spike of neuron B reaches neuron A, $f(\Phi_A)$ is increased (excitatory coupling) or decreased (inhibitory coupling) by an amount of ϵ, which is equivalent to a jump in phase (Fig. 1a):

$$\Phi_A \longrightarrow \Phi_A + \delta\Phi_A = g\left(min[f(\Phi_A)+\epsilon, 1]\right) \quad ; \quad \epsilon > 0 \qquad (1)$$

$$\Phi_A \longrightarrow \Phi_A + \delta\Phi_A = g\left(max[f(\Phi_A)+\epsilon, 0]\right) \quad ; \quad \epsilon < 0 \qquad (2)$$

We include a synaptic transmission delay, which means that the spike from neuron A reaches neuron B after a time $\tau < 0.5$. We rigorously construct a return-map $R(\Phi)$ which maps the phase-difference $\Delta\Phi(t_1) = \phi_B(t_1) - \phi_A(t_1)$ when Neuron A fires onto $\Delta\Phi'(t_2)$ when neuron A subsequently fires again [7]. We find two stable and two unstable fixed points in R leading to desynchronisation for $\tau > 0$ in case of excitatory coupling $\epsilon > 0$ (Fig. 1b). With inhibitory

couplings, we get either perfect synchronization or total desynchronization, depending only on the initial configuration (Fig. 1c). For large ϵ, two of the four fixed points vanish and the neurons either desynchronize ($\epsilon > 0$) or synchronize ($\epsilon < 0$) for all possible initialisations (for the complete analysis see [7]).

3 Delay-Induced Phase Clustering

We now consider an ensemble of N such neurons mutually coupled with a delay τ. We furthermore assume that a neuron cannot fire twice during a time τ which holds for $\tau < 0.5$ and sufficiently small ϵ. The state of N globally coupled neurons at time t_i is characterized by $\vec{\Pi}(t_i) = (\vec{\Phi}(t_i), \vec{\tau}(t_i))$ with $\vec{\Phi}(t_i) = \{\Phi_n(t_i); n = 1, .., N)\}$ and $\vec{\tau}(t_i) = \{\tau_n(t_i); n = 1, .., N\}$, where $\tau_n(t_i)$ denotes time since the most recent spike of neuron n. The network evolves under the same dynamics as mentioned above. Choosing a reference neuron j, we can define a similar return-map R' which maps $\vec{\Pi}(t_i)$ onto $\vec{\Pi}(t_{i+1})$, where t_i denotes the time when neuron j fires the i'th time. In our simulations, we found stable phase clustering as the result of inhibitory connections (Fig 2b). Decreasing the delay, more and more subsequently firing clusters of neurons appear (Fig.3a), corresponding to an effective frequency increase in the network. With excitatory couplings and nonvanishing delays, we found spontaneous synchronization and desynchronization of several clusters. Also the emergence of new clusters seems to destabilize others (Fig.2a).

4 The Mechanism

In case of inhibition the basins of attraction of the fixed points of R explain the emergence and number of stable clusters. The smaller the delay, the smaller is the basin for synchronization and the more clusters can coexist (Fig 2b,3). For excitation clusters become unstable which relates to the unstable fixed point at $\Phi = 0$. However, clusters may still emerge when sufficient synchronous neurons with a different phase override the self desynchronization (Fig.2a). Heuristically, the delay induces an efficient and instantanous force on the phase differences which can be derived as follows. Consider the dynamics:

$$\frac{d}{dt}\Phi_n(t) = \omega + \frac{const}{N-1} \sum_{m=1; m \neq n}^{N} V[(\Phi_n - \Phi_m)(t)] + \eta(t). \qquad (3)$$

Discretizing this function for $N = 2$ and comparing it to the exact mathematical analysis of the dynamics, we find $V(\Delta\Phi) := \frac{1}{2 \cdot const. \cdot \Delta t}[R(\Delta\Phi) - \Delta\Phi]$ and $V(-\Delta\Phi) := -V(\Delta\Phi)$, i.e. that the phase description is identical to the return-map formalism. In case of $N > 2$ we performed simulations which show excellent agreement with the full dynamics. In the inhibitory case, the clusters are reproduced for all τ (Fig.3). The excitatory case is not completely reproduced by (Eq.3) (not shown). Here the delay cannot be eliminated in this way because it governs the time course of decay and emergence of unstable clusters.

5 Conclusion

We analyzed the mechanisms leading to phase clustering and desynchronization in a simple model of pulse coupled neurons with delay. The number and stablility of clusters is explained by the fixed points and their basins of attraction of a return map which can be rigorously derived from the dynamics of two interacting neurons. We related this description to a model of instantanously coupled phase oscillators for which we show that the corresponding interaction function has higher Fourier modes, a fact which has already been shown to induce phase clustering [8]. The underlying mechanism is generic as it depends only on monotonity and convexity of the membrane potential. Therefore our analysis should be relevant for many much more detailed models and we claim that it explains high frequency network oscillations as e.g. recently found in the hippocampus [9].

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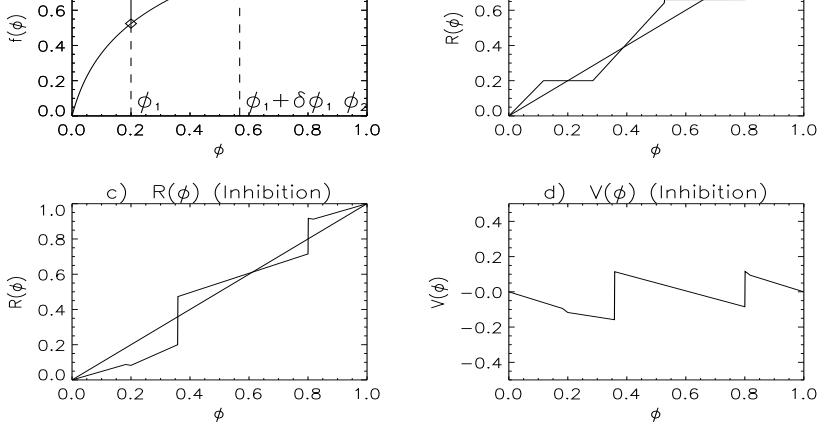

Figure 1: a) State f of an abstract neuron depending only on the time Φ since the last spike. For finite delay $0 < \tau < 0.5$ the return map R has a fixed point at $\Phi = 0$ which is *never* stable when the coupling is excitatory (b: $\tau = 0.2, \epsilon = 0.1$) and *always* for inhibition (c: $\tau = 0.2, \epsilon = -0.1$). d) Plot of the efficient phase interaction for case c).

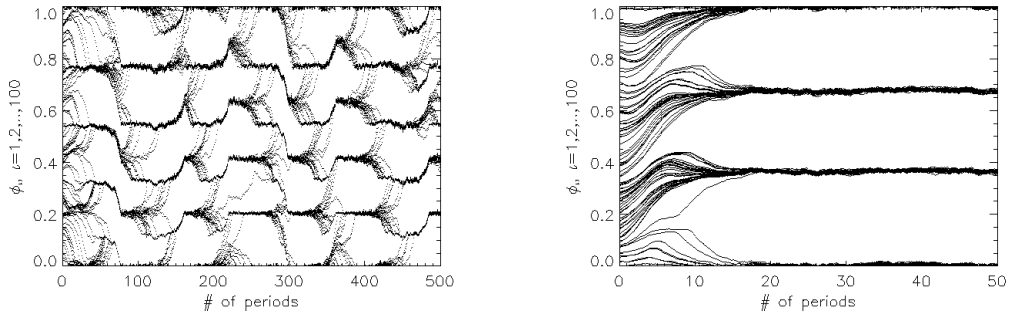

Figure 2: Return plots of the phases Φ_i in a population of $i = 1, \ldots, N = 100$ neurons. Depicted are the phases each time a fixed but arbitrary neuron fires (left: $\tau = 0.2, \epsilon = 0.1$ and right: $\tau = 0.2, \epsilon = -0.1$).

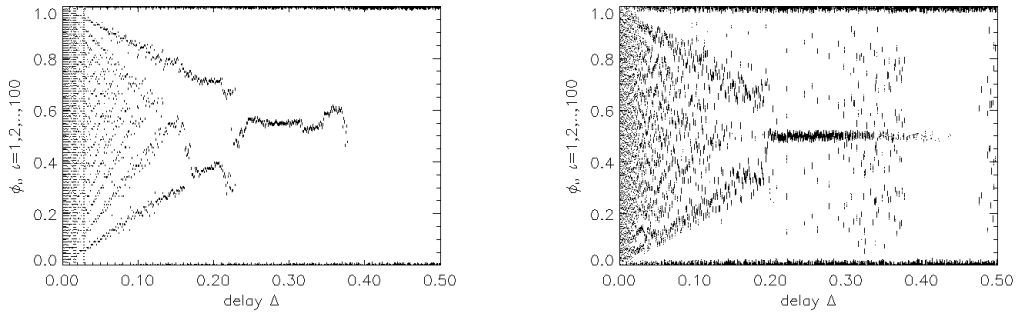

Figure 3: Final positions of the relative phases Φ_i for different delays τ ($\epsilon = -0.1$,left) and the corresponding result for the dynamics (Eq.3) involving the instantaneous phase interaction V derived from R (right).