Synchronization and label-switching in networks of laterally coupled model neurons

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Abstract

Necessary conditions for the impulse synchronization in non-oscillating networks of laterally coupled 'integrate-and-fire' model neurons are investigated. The behaviour of such networks for homogeneous stimulations as well as for differently stimulated subpopulations is studied. In the first case, synchronization accurate to fractions of the impulse duration can be achieved by either lateral *inhibition* or lateral *excitation* and in the second case, good and independent synchronization is obtained within subpopulations, if they are separated by unstimulated units.

1. INTRODUCTION

Recent electrophysiological investigations of the visual cortex, mainly in the cat, revealed stimulus-dependent temporal correlations between spatially separate neural activity [1,2]. It is currently conjectured that the (spatial) coherence of visual data, such as direction and speed of motion, contrast, texture or color, is expressed (labeled) by synchronized neural activity. In this sense, neurons that fire asynchronously but perhaps at the same rate, indicate non-coherent features, e.g. those belonging to different objects. Obviously, a thorough judgement of this hypothesis must be based on some knowledge about necessary conditions for the synchronized spike generation in networks of 'integrate-and-fire' model neurons. Consequently, we resume and extend last year's report on this issue [3], before we deal with responses of such networks to spatio-temporal stimuli.

2. NETWORK MODEL AND MEASURE OF SYNCHRONIZATION

The model neuron (unit) considered here behaves like a leaky integrator with a time constant of 10ms. Hence, a unit's integral excitatory input increases its somatic potential $D_i(t)$ according to the step response of a first order lowpass. When this potential surpasses the threshold θ , an exponentially declining impulse $A_i(t)$ of 1ms duration is triggered and the somatic potential is reset to the refractory potential D_{re} . After a refractory period of 0.5ms the integration starts again (see Figure 1). The single stage networks are cyclically closed chains of n laterally coupled units (cf. Figure 2). All interconnections are either *inhibitory* or *excitatory* with coupling coefficients c_V that linearly decrease with the distance. In order to quantify the degree of synchronization the quality factor η is defined which is the maximum value of the spike density $S_d(t)$ within intervals of 50ms. The instantaneous spike density in turn is the ratio between the binarized activity within a spatio-temporal window (n units long and a spike duration wide) and the area of this window (see Figure 5 for examples of $S_d(t)$). The spike density $S_d(t)$ =1 and thus

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 η =1 is reached iff all n units start firing simultaneously. Two quality factors η_{50} and η_{200} are shown. They are the maxima of the spike densities $S_d(0...50\text{ms})$ and $S_d(150...200\text{ms})$ respectively. The synchronization η_{ref} refers to the uncoupled network and – due to the randomized starting potentials $D_i(t\le0)$ and to the noisy thresholds $\theta+N_i(t)$ – implies asynchronous impulse generation. It is evident that this reference value increases with the firing rate $R_A(C,\tau)$ and thus it depends on the lateral coupling strength C and, as it is shown below, on the lateral delay τ . More detailed descriptions of the neural network model are given in [3].

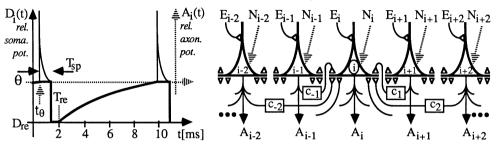


Figure 1. Typical time-course of the somatic potential and of the axonal impulses

Figure 2. Interconnection scheme of the onedimensional network

3. GLOBAL HOMOGENEOUS STIMULATION

Good synchronization with η >0.7 is observed for the following standard parameters: number of units in the network n_s =64, number of bilateral connections per unit k_s =8, integral lateral coupling strength per unit C_s = Σc_v =+0.2, same excitation $E_i(t>0)$ = E_s of every unit (which causes an uncoupled unit to fire at the rate R_{As} =100s⁻¹), lateral transmission delay τ_s =0ms and a temporal increment Δt_s =0.1ms of the simulation. The influence of deviations from these standard values on the synchronization is explicated in [3]. The extended investigations reported below include inhibitory lateral interconnections (C<0) and more realistic delays τ >0.5ms. In order to avoid very low impulse rates in the inhibitory networks, an excitation of $2E_s$ was used for the experiments reported in section ii.

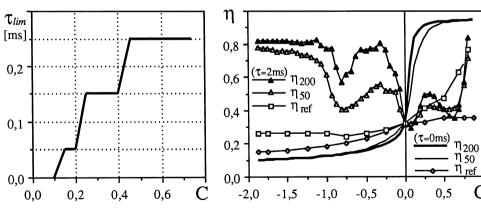


Figure 3. Tolerable time delay τ_{lim} for $\eta_{200} > 0.7$ as a function of the lateral coupling strength C

Figure 4. Synchronization as a function of the lateral coupling strength C for transmission delays τ =0ms and τ =2ms

i) In the case of purely *excitatory* interconnections, the tolerable delay τ for $\eta_{200}>0.7$ grows monotonously with the coupling strength C and has the value $\tau_{lim}=0.25$ ms at the critical coup-

ling strength C_{crit} =+0.78 (cf. Figure 3). (For $C>C_{crit}$ an externally unstimulated unit is triggered by synchronous lateral impulses from one side.) As a consequence of the temporal sampling – which implies a mean intrinsic delay of $\tau \approx \Delta t/2$ [4] – temporally discrete simulations must be performed with temporal increments $\Delta t \leq 0.5$ ms.

ii) The data displayed in Figure 4 clearly demonstrates that spike synchronization is feasible with either purely *inhibitory* or *excitatory* interconnections. The corresponding values for the lateral delay and coupling strength are complementary: Non-delayed lateral excitation results in excellent synchronization (η_{200} =0.9), whereas non-delayed lateral inhibition desynchronizes (η_{200} =0.1< η_{ref} =0.2). Delayed (τ =2ms) lateral excitation does not lead to a significant degree of synchronization – it remains in the range of the reference quality. (The increases in synchronization and reference quality beyond C=0.7 are due to a steep increase of the impulse rate which reaches R_A =500s⁻¹). However, delayed (τ =2ms) lateral inhibition produces very well synchronized activity (η_{200} =0.8). This is due to the temporally nonlinear influence of inhibitory postsynaptic potentials (IPSPs) on the generation of action potentials: The efficacy of an IPSP in delaying the triggering of a spike is stronger for somatic potentials near the threshold than for low somatic potentials. Hence, advancing impulses are stronger retarded than later arriving ones which results in synchrony.

4. SELECTIVE STIMULATION OF SUBPOPULATIONS

After this brief outline of the network behaviour under global and constant stimulation first results from simulations with spatio-temporal stimuli are reported. For this purpose the randomized starting potentials $D_i(t \le 0)$ are replaced by a deterministic pattern P_D which is superimposed with a differently shaped stimulation pattern P_E for times t > 0. Effects of this so-called label-switching on the spatio-temporal synchronization process are studied. Except for the initial potentials D_i and the stimulation E_i all network parameters conform to the standard settings.

Synchronization within each of two subpopulations for times $t \le 0$ is simulated by appropriate initialization of the somatic potentials of the corresponding units: While half of the units i=16...47, start with their somatic potentials at the threshold $D_{16...47}(t \le 0) = \theta$ (label 2) the other half i=48...15 (closed chain) is initialized with the potential $D_{48...15}(t \le 0) = (\theta + D_{re})/2$ (label 1). For times t>0 the units i=1...32 are stimulated by a sustained input E_s (label 1') and the units i=33...64 by $0.7 \cdot E_s$ (label 2'), i.e., at time t=0 the pattern P_E is shifted by 16 units with respect to pattern P_D as it is depicted in Figure 5A.

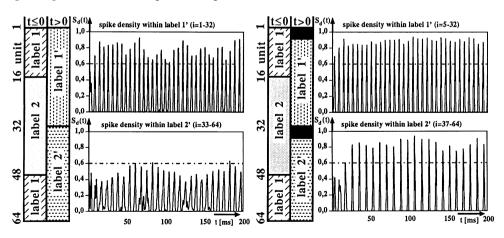


Figure 5. Stimulation patterns P_D and P_E , and the resulting spike densities $S_d(t)$ (A) for label-switching and (B) for label-switching with buffer units

Under these circumstances, the stronger stimulated (E_s) subpopulation is moderately synchronized, whereas the less stimulated $(0.7 \cdot E_s)$ subpopulation shows insufficient synchronization. Obviously, there is a destructive influence of label 1' on label 2'.

Good and undisturbed synchronization in both subpopulations is observed if the differently stimulated subpopulations are separated by externally unstimulated but still laterally coupled units (here: i=1...4, i=33...36), as it is shown in Figure 5B. Under this condition it is even possible to switch from 2 to 3 labels without loss of synchrony in the new subpopulations. If lateral transmission delays are introduced, more buffer units are required in order to maintain good and independent synchronization in the subpopulations.

5. DISCUSSION

An essential difference between the presented network and most of the presently discussed models [5,6,7,8,9], is the lack of explicitly implemented oscillator *circuits*. Consequently, any spiking activity is due to sufficiently stimulated model neurons that represent voltage-controlled and noisy generators for impulse trains. Every spatio-temporal binding in a network's activity results from the lateral couplings, from a possibly constancy of the external stimulus, or it occurs simply by chance. It is important to note that synchronization by lateral interconnections works over a large range (at least 1:10 in the case of lateral excitation) of impulse rates (cf. Figure 5B and [3]) and it is only recently that good neurophysiological evidence for spatial correlations during aperiodic neural activity was reported [10].

The superiority of networks with inhibitory lateral interconnections – especially with respect to realistic delay times – is obvious: Delays greater than τ =0.25ms permit only weak excitatory synchronization and neuroanatomically regarded, lateral inhibition is more likely as well [11].

The investigations clearly demonstrate that a synchronization accurate to fractions of the impulse duration and consequently, the encoding of up to 50 phase labels (at R_A =100s⁻¹), is feasible by either inhibitory or excitatory interconnections. There is no need for additional nonlinearities such as 'multiplicative synapses' [6] – i.e., the threshold nonlinearity is sufficient –, or different somatic integration time constants for external and lateral inputs [12].

According to Figure 5, the time needed for good synchronization after label-switching is quite short (less than 50ms) and thus it is well within a biologically relevant range. The interference between differently stimulated subpopulations demonstrates that there is only a minor spatial spread of synchronization and that synchronized activity in a coherently but lower stimulated subpopulation in the immediate neighbourhood can be destroyed. Whether lateral inhibition leads to a different behaviour after label-switching is up to future experiments.

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