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On spike synchronization

H. Glünder¶ and A. Nischwitz*

Institut für Medizinische Psychologie, Ludwig-Maximilians-Universität
Gœthestraße 31, D-8000 München 2, Germany

*Institut für Nachrichtentechnik, Technische Universität München
Arcisstraße 21, D-8000 München 2, Germany

Abstract

We start with historically founded reflections on the relevance of synchronized activity for the neural processing of information and we propose to differentiate between synchrony at the emitting and the receiving side. In the main part we introduce model networks which consist of chains of locally coupled and noisy spiking neurons. In the case of lateral excitation without delay as well as for delayed lateral inhibition these basic structures can turn homogeneous stimulations into synchronized activity. The synchrony is maintained under temporally varying stimulations thus evoking aperiodic spike fronts. Although we present some hypotheses, the question of how the nervous system deals with this network property remains to be answered.

1. INTRODUCTION

Half a century ago McCulloch and Pitts [1] stated that neurons are principally suited to perform Boolean operations. Undoubtedly, the authors were strongly influenced by the developing theory of automata and especially by the incredible perspectives of an effective mechanisation of the logical calculus. At that time, for instance, electronic AND-gates consisted of a resistive network for the summation of electric currents followed by an active thresholding device and therefore, they represented an attractive structural and functional analogue to nerve cells. However, much more importantly, this view marks the fundamental transition from regarding neurons as integrators to realizing them as coincidence detectors (cf. [2; 3]). Although coincidence detection is the principle of AND-gates, it did not become popular even in biological cybernetics and theoretical neuroscience. Instead, the generalized McCulloch/Pitts-(model)neuron which computes with a mathematical construct, namely instantaneous impulse rates, and not with impulses (action potentials or spikes), became standard – not only for most simulations of neural networks but also for interpretations of neurobiological experiments. Thus, until recently, only few scientists investigated the temporal fine structure of neural signals, i.e. of spike trains and bursts as well as the associated postsynaptic potentials, and hypothesized about its putative significance for the processing of neural information.

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2. THE RELEVANCE OF SPIKE SYNCHRONIZATION

In one of the early comments on this issue Wiener [4, chapter 10] points out that coincident or synchronized spikes at a neuron's input terminals will be much more efficient for the triggering of action potentials than asynchronous, for instance stochastic impulses. Owing to this basic functional property of nerve cells, Wiener concludes that synchronously oscillating nervous activity should be found in the brain – a notion which appears rather modern, although he concentrated on the α-rhythm while nowadays oscillations in the γ-band are favoured. Although Wiener's conclusion and its presently discussed versions are appealing, they lack stringency:

(i) In the same way as the statement of McCulloch and Pitts does not imply that neurons really act as AND-gates, the similarly reductionistic view of neurons as coincidence detectors does not imply the actual use of this faculty for neural processing (cf. the analysis of basic misconceptions in cybernetics by Taube [5] chapter 6).

(ii) Obviously, the type of coincidence detection considered here takes place at a neuron's axon hillock. Such somatic coincidences generally differ from synchronous input to a neuron. Consequently, action potentials that appear at the same time at various presynapses of a target neuron – thus representing synchronous input activity – need not cause coinciding excitatory postsynaptic potentials at its cell body (cf. [6] section 5). This discrepancy can be due to differences in conduction times as well as to delayed synaptic transmission which can be caused by molecular processes such as second messenger cascades. One may even conjecture that, within limits, neurons are able to produce somatic coincidences between non-coinciding action potentials by (learning) appropriate synaptic delays.

(iii) At least phenomenologically, synchrony need not be bound to periodic processes because aperiodic events may be synchronized as well. It should be realized that periodicity commonly refers to a *single* signal whereas synchronization exclusively concerns the (temporal) relation between *several* signals. Hence, it is somewhat surprising that the putative advantage of coincidence detection for the processing of neural information is quite often associated with oscillatory activity in the brain.

Of course, there is a rarely explicated reason for the association mentioned in comment (iii): The *generation* of synchronous spikes in neural populations by means of local cooperative processes, i.e. without central control (triggering or gating), is supported by short epochs of near to constant stimulation and consequently quasiperiodic firing. (This need not hold for already synchronized ensembles.) Whether this kind of short-term binding is recognized as oscillatory is a matter of taste. We prefer the aspect of fairly rapid variations in (synchronized) neural activity [7, section 4] rather than that of more or less stationary oscillations – or oscillations in the sense of slowly shifting and thus narrow spectral frequency bands. This emphasis appears justified by the notion that neurons are voltage controlled (stochastic) impulse generators (cf. [8]) which obviously serve the processing of time-varying signals.

According to remark (ii) there is generally little reason for the *emission* of synchronous action potentials from a neural population in order to optimally stimulate *coincidence detectors* (cell somata) if a *transmission channel* (axons, synapses and dendrites) with space-variant temporal properties must be assumed. However, one decade ago, a neural receiver mechanism was identified for which the emission of synchronized impulses could make sense, namely coincidence-detecting NMDA-type synapses [9; 10]. It can detect coincidences between action potentials that are transmitted at essentially the same dendritic site [11] but, in contrast to the somatic coinci-

dence detection of tonic potentials, it is limited to small numbers of input signals. In short, one must be aware of what is or should be synchronized at which location.

Other reasons for the generation of synchronous events are their direct behavioural use, such as the synchronous emission of light flashes by populations of certain fireflies [4; 12], and the suspected relevance for general timing purposes [13; 14; 15].

We like to conclude this argumentation with yet another statement which anticipates the essence of our own investigations: We found that synchronization of neural spiking activity generally *must be expected* in populations of homogeneously stimulated neurons which are locally coupled – either in an excitatory space-invariant feedforward fashion or by delayed (recurrent) lateral inhibition. Because both are quite common interconnection schemes, especially in cortical structures, we conclude that synchronized action potentials should not be regarded as particular network states – at least unless these synchronizing mechanisms are commonly paired with desynchronizing ones, such as inhibitory and space-invariant forward couplings – or that our simulations turn out to be too simplistic.

3. NETWORKS OF LOCALLY COUPLED MODEL NEURONS

Our investigations started from the question regarding necessary conditions for the generation of synchronous impulses in populations of locally interconnected model neurons. In order to tackle this problem, we needed an appropriate, i.e. in larger populations still computationally manageable model neuron (unit) with spiking output. A chain of such units that are laterally coupled with their neighbours by either excitatory or inhibitory interconnections – with or without delay – was considered as a promising and simple enough network structure.

3.1 The model neuron

We use a model neuron with the subthreshold behaviour of a leaky integrator which can be characterized by its δ-impulse response

$$h(t) = e^{-t/\tau}/\tau \quad \text{for } t \geq 0$$

with the time constant τ =10ms. We distinguish three kinds of input signals that are *linearly summed* by such units (all potentials are normalized to the threshold θ):

- the *feeding* input $e(t)$

  which represents stimulations from outside the network and changes the somatic resting potential by $u_e(t) = e(t) * h(t)$ ("$*$" denotes convolution). For most of the experiments reported here, we consider $E := e(t) = const.$ which simulates incoherent input via many weakly transmitting, e.g. "apical-dendritic" synapses (Figure 3) and changes the somatic potential according to the step response (Figure 2)

$$u_E(t) = E\left(1 - e^{-t/\tau}\right) \quad \text{for } t \geq 0 \ .$$

- the *lateral* input $a(t) = \sum_\nu w_\nu \cdot p_\nu(t - \vartheta)$ ($w_\nu > 0$: excitatory; $w_\nu < 0$: inhibitory) is the weighted sum of impulse trains $p_\nu(t) = \sum_\kappa s(t - t_\kappa)$ that are transmitted with delay $\vartheta = const.$ from ν neighbouring units via, e.g. "basal-dendritic or somatic" synapses, i.e. from inside the network (Figure 3). (Times t_κ denote the spike positions in an impulse train.) This input alters the somatic potential according to $u_a(t) = a(t) * h(t)$. Figure 1 shows the assumed exponentially decaying action po-

tential $s(t)$ (time constant $\tau_{AP} = 0.144\text{ms}$) together with its postsynaptic response at the soma $u_{PSP}(t)$ which is normalized to its *coupling strength* w.

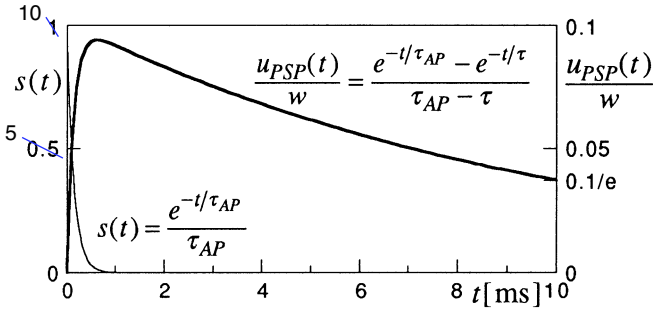

Figure 1. Action potential $s(t)$ and normalized postsynaptic potential $u_{PSP}(t)/w$

- the *noise* input $n(t)$ (individually computed for each unit) is a random process with uniformly distributed values from the range $\pm E/2$ which mimicks stochastic fluctuations $u_n(t) = n(t) * h(t)$ of the somatic potential.

In total, the change of the subthreshold somatic potential from the resting potential is

$$u(t) = u_e(t) + u_a(t) + u_n(t) = [e(t) + a(t) + n(t)] * h(t) \ .$$

(We neither consider synaptic habituation nor nonlinear synaptic transmission or interactions.) When the somatic potential exceeds the threshold θ, then an action potential $s(t)$ is triggered and 1ms later the somatic potential is set to the refractory potential for a period of 0.5ms before the integration can start again. The feeding input E is specified by the period T of the impulse train it evokes in a noisefree unit.

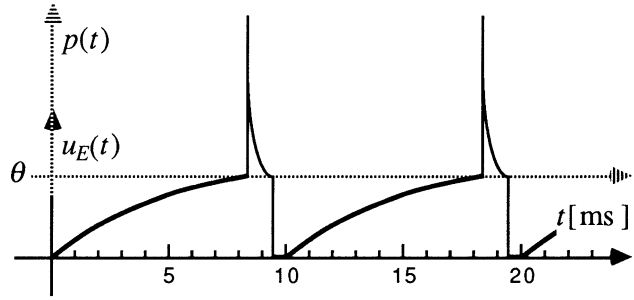

Figure 2. Typical time courses of the subthreshold somatic potential $u_E(t)$ as well as of the emitted spike train $p(t)$ with the period $T = 10\text{ms}$

3.2 The network

Figure 3 depicts the neighbourhood of a model neuron in our one-dimensional, single stage network. In order to avoid boundary problems in networks of manageable size the chain is cyclically closed and all its N units are coupled in the same way. We consider either purely inhibitory or excitatory interconnections without direct feedback from units onto themselves. Every unit receives input from its immediate $k \ll N$ neighbours on either side with a strength w_ν that linearly decreases with the distance, i.e. with $|\nu|$. Unlike the coupling strength, the transmission delay is assumed constant which implies similar axonal conduction times as well as synaptic and post-

synaptic processing (see point (ii) of section 2). In order to characterize the efficacy of interaction in the whole network, we introduce the *total coupling strength*

$$W = \sum_{\substack{\nu=-k \\ \nu\neq 0}}^{+k} w_\nu = const.$$

of every model neuron. Of course, there is a reasonable desire for global stability which in turn necessitates an upper limit of the excitatory coupling strength which we choose to $W_{crit} = +0.78$. For this *critical coupling strength* an unstimulated and noiseless unit begins to spike under synchronous unilateral input.

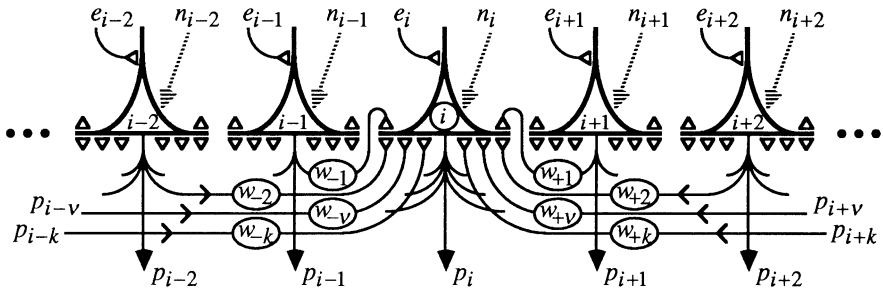

Figure 3. Local interconnection scheme of a unit in the one-dimensional network

Although we report on investigations of single stage or "single layer" networks, locally divergent forward coupling between "layers" – similar to the one proposed by Abeles [16, chapter 7] for the generation and transmission of synfire chains – appears more realistic for configurations without delay. Non-delayed lateral excitation can be directly achieved by forward coupling whereas non-delayed lateral inhibition requires the compensation of different delays between the central excitatory path and the divergent inhibitory ones which are due to the inevitable inhibitory interneurons.

3.3 Network simulation and measures of synchrony

We studied the *discrete* nonlinear dynamics of the networks, i.e. we performed simulations on a digital computer with the temporal resolution Δt. As a consequence, the zero delay in the lateral links can only be approximated, i.e. one must accept the mean intrinsic delay of $\vartheta_0 = \Delta t/2$. For the experiments reported here, all units of the network received the same feeding input for times $t \geq 0$. However, their initial somatic potentials ($t < 0$) were individually set to uniformly distributed random potentials from the "refractory potential to threshold"-range. Unless stated otherwise, the parameter values of the networks were choosen as follows:

$$\Delta t_s = 0.1\text{ms}\ ; \quad N_s = 64; \quad k_s = 8\ ; \quad E_s \text{ so that } T = 10\text{ms}\ ;$$

This standard setting turned out to be useful for most of the investigations and does not represent an extreme choice. The influence of deviations from these standard values on the quality of synchronization is explicated elsewhere [17].

In order to quantify the degree of synchronization we define the instantaneous *spike density* $S(t)$ which is given by the binarized spike aktivity of the network in a

spatio-temporal window which is N units long and one spike duration $M \cdot \Delta t = 1\text{ms}$ wide, devided by the maximum possible activity in this window (Figure 5).

$$S(t) = \frac{1}{NM} \sum_{i=1}^{N} \sum_{j=0}^{M-1} P_i(t - j\Delta t) \quad \text{with} \quad P_i(t) = \begin{cases} 0 & p_i(t) = 0 \\ 1 & \text{else} \end{cases}$$

Consequently, $S(t) = 1$ denotes an instant of perfect synchrony, i.e. all N units must have triggered action potentials at exactly the same time. Occasionally, we display the *envelope function* $\hat{S}(t)$ of the spike density. Finally, the *quality factor* η is defined as the mean over 50 runs of the maximum spike density that is determined during intervals of 50ms. We display $\eta_{50} = \max_{0<t\le 50} \{S(t)\}$ and $\eta_{200} = \max_{150<t\le 200} \{S(t)\}$.

$$\eta_x = \frac{1}{50} \sum_{\mu=1}^{50} \max_{(x-50<t\le x)} \{S_\mu(t)\}$$

For an assessment of the quality factor we provide the *reference quality* η_{ref} which results from "synchrony by chance" in the uncoupled ensemble but, since the reference quality depends on the impulse rate $r(W)$, which in turn can be converted to an equivalent reference quality, η_{ref} can also be specified for coupled networks.

4. SIMULATION RESULTS

After this detailed description which appears indispensible for judging the consequences of our findings, we present a compilation of the main results in Figure 4. Obviously, significant synchronization is feasible by either purely inhibitory or excitatory lateral links. (Figure 5 shows examples of corresponding spike densities.) In both cases, synchronization is based on the nonlinear characteristic of the somatic integration: The efficacy of a postsynaptic potential in delaying/accelerating the triggering of an action potential is higher for somatic potentials near the threshold than for small depolarizations. Therefore and with respect to the spike emission, advanced/retarded impulses are more strongly retained/impelled than late/early ones.

On the whole, a complementary behaviour is observable with respect to delays:

ϑ	$W < 0$	$W > 0$
$\vartheta_0 = 50\mu\text{s}$	desynchronization: $\eta_{200} < \eta_{ref}$	excel. sync.: $\eta_{200} > .9$ for $W > +.2$
$\vartheta_2 = 2\text{ms}$	good sync.: $\eta_{200} > .75$ for $W < -1$	no significant sync.: $\eta_{200} \approx \eta_{ref}$

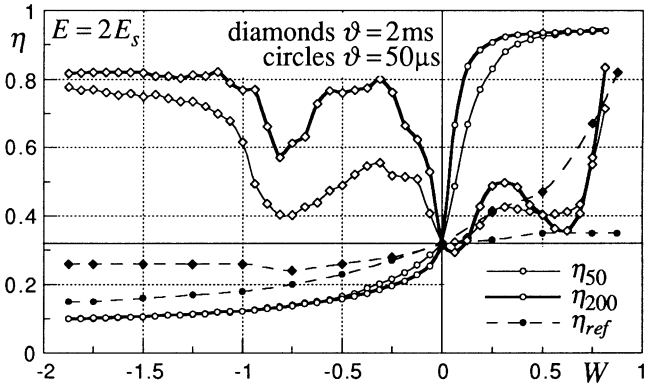

Figure 4. Synchronization quality $\eta(W)$ as a function of the total coupling strength W for transmission delays $\vartheta = 50\mu\text{s}$ and $\vartheta = 2\text{ms}$

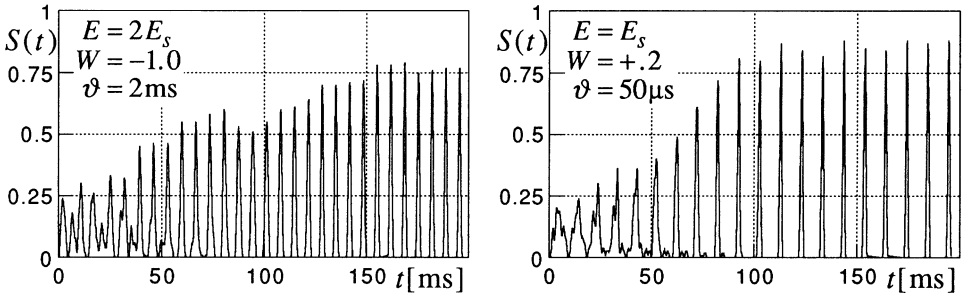

Figure 5. Examples of instantaneous spike densities $S(t)$ (note the different inputs)

Compared to the impulse rate $r_0(W_{crit}) \approx 195\text{s}^{-1}$ for the non-delayed transmission, delays considerably increase the impulse rate to $r_2(W_{crit}) \approx 400\text{s}^{-1}$ which means that with delay additional impulses are generated while without it retarded action potentials are accelerated up to synchrony. A similar effect holds for inhibitory links with $r_0(W=-1.87) \approx 60\text{s}^{-1}$ and $r_2(W=-1.87) \approx 142\text{s}^{-1}$. ($r_{ref} = r(W=0) \approx 180\text{s}^{-1}$)

Actually, synchronization depends on the amount of the delay. For $W=-1.0$ and $E=2E_s$ we observed a continuous increase from desynchronization $\eta_-(\vartheta_0)$ to the maximum $\eta_-(\vartheta_2)$ which is sustained apart from 1ms dips at multiples of 6ms. However, for $W=+.2$ and $E=E_s$ we found a steep decrease from excellent synchronization $\eta_+(\vartheta_0)$ down to the reference level at $\vartheta=.5$ms which is maintained except for peaks around multiples of 6ms that are less pronounced for η_{50} and decrease with ϑ.

Responses to a *linear downward sweep* of the feeding input are plotted in Figure 6. The envelope of the spike density $\hat{S}(t)$ indicates a consistently high degree of synchrony over the whole range of stimulation. Furthermore, this experiment nicely reveals the nonlinear transfer characteristic $r(e)$ of a coupled model neuron.

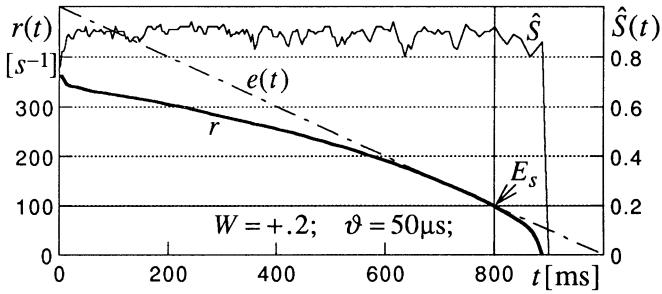

Figure 6. Spike density envelope $\hat{S}(t)$ and impulse rate $r(t)$ for a linear sweep of the feeding input $e(t)$

5. DISCUSSION

According to our findings, synchronized spiking activity results almost inevitably from homogeneously stimulated and especially excitatorily forward coupled or delayed (interneurons!) lateral inhibition networks. Contrary to the present euphoria over this *highly ordered* spatio-temporal binding, we are not certain as to its relevance for the processing of neural signals (see section 2): If a significant number of neurons really act as somatic coincidence detectors for *complex* spatio-temporal ac-

tivity patterns, why then are synchronizing circuits required? However, aside from coincidence-detecting synapses, synchronous spike emission may turn out advantageous for reasons of selforganization.

After this general consideration we return to the issue of synchronized aperiodic spike fronts. In addition to the demonstration in Figure 6, we found that even abrupt variations of the global stimulation do not significantly disturb an already existing synchrony. – Moreover, spike synchronization happens stepwise and therefore, the time needed to reach a desired degree of synchrony depends on the spike rate.

Compared with related work on excitatorily coupled networks we have shown that neither nonlinear synapses [18] nor different time constants for feeding and synchronizing inputs [19] are a requisite for good synchronization. Although fully interconnected networks [20; 21] cannot really be compared they show states similar to those occuring in our experiments. In this context we would like to mention that our preferred total coupling strength $W = +.2$ means a rather *weak coupling*.

Finally, we must indicate a limitation of temporally discrete simulations arising from the fact that discrete nonlinear systems are not necessarily approximations of their continuous originals. Hence, all such simulations must be taken with extreme caution. Up to now we could only show a convergence for decreasing increments Δt.

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BRAIN THEORY

SPATIO-TEMPORAL ASPECTS OF BRAIN FUNCTION

Edited by

A. AERTSEN
Institut für Neuroinformatik
Ruhr-Universität Bochum
Bochum, Germany

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