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On spike synchronization

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Abstract

We start with historically founded reflections on the relevance of synchronous activity for neural information processing and we propose to differentiate between synchrony at the emitting and the receiving side. In the main part we introduce chains of impulse coupled and noisy formal neurons in which random spiking will most likely synchronize, if the local lateral coupling is either excitatory without delay or delayed inhibitory, and if the mean drive of all neurons is about uniform. Synchrony is maintained under temporally varying stimulations which result in aperiodic spike fronts. Although we present some hypotheses, the question of how actual neural systems deal with this almost inevitable synchronizing behavior, remains to be answered.

1. INTRODUCTION

Half a century ago Warren McCulloch and Walter Pitts [1] stated that neurons are in principle 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 mechanization 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 and much more importantly, this view marks the fundamental transition from regarding neurons as integrators to realizing them as possible coincidence detectors (cf. [2; 3]). Although coincidence detection is the principle of AND-gates, it did not become popular even in biological cybernetics and computational neuroscience. Instead, the generalized formal McCulloch/Pitts-neuron (graded output unit) that generates a mathematical construct, namely impulse rates, not impulses (action potentials or spikes), became standard – not only for simulations of neural networks but also for interpretations of neurobiological experiments. Thus, until recently, rather few scientists have investigated the temporal fine structure of neural signals, i.e., of spike trains and bursts as well as the associated post-synaptic potentials, and hypothesized about their possible 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 Norbert 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 that 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 sense 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 misconceptions in cybernetics by Taube [5] Chapter 6).

(ii) Obviously, the kind 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 pre-synapses of a target neuron – thus representing synchronous input activity – need not cause coinciding excitatory post-synaptic potentials at its cell body (cf. [6] Section 5). This discrepancy can be due to differences in conduction times or to delayed synaptic transmission that 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 synchronous as well. It should be realized that periodicity commonly refers to a *single* signal whereas synchrony exclusively concerns the (temporal) relation between *several* signals. Hence, it is somewhat surprising that a possible advantage of coincidence detection for the processing of neural information is often associated with oscillatory activity in the brain.

Of course, there is a seldom explicated reason for the association mentioned in comment (iii): The *generation* of synchronous spikes in neural populations by local cooperative processes, i.e., without central control (triggering or gating), is supported by short epochs of near to constant stimulation and consequently quasi periodic firing. Whether this kind of short-term constancy is regarded as oscillatory, is a matter of taste. We prefer the aspect of fairly rapid variations in (synchronous) neural activity [7, Section 4] rather than that of more or less stationary oscillations, or oscillations in the sense of slowly shifting, hence narrow spectral frequency bands. This emphasis appears justified by the notion that neurons are voltage controlled (stochastic) impulse generators (cf. [8]) that 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 to optimally stimulate *coincidence detectors* (cell somata) if a *transmission channel* (axons, synapses, dendrites) of spatially variant temporal properties must be assumed. However, one decade ago, a neural receiver mechanism was identified for which the emission of synchronous impulses could make sense, namely coincidence-detecting NMDA-type synapses [9; 10]. It can detect coincidences of action potentials that arrive at pre-synapses situated at essentially the same dendritic site [11]. In contrast to somatic coincidence detection of tonic potentials however, it is limited to only a few input signals. In short, one must be aware of what is or shall be synchronized and at which location.

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

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

3. NETWORKS OF LOCALLY COUPLED FORMAL NEURONS

Our investigations started from the question about necessary conditions for the generation of synchronous impulses in populations of locally interconnected formal neurons. To tackle this problem, we needed a formal spiking neuron (unit) that is computationally manageable in larger populations. Chains of such units that are laterally coupled to their neighbors by either excitatory or inhibitory interconnections and with or without delay was considered a promising and simple enough network structure.

3.1 The formal neuron

We use a formal neuron with the sub-threshold behavior of a leaky integrator that can be characterized by its δ-impulse response

$$h(t \geq 0) = \tfrac{1}{\tau} e^{-t/\tau}$$

and the time constant $\tau = 10\,\text{ms}$. We distinguish three kinds of input signals that are *summed* by these units (all potentials are normalized to the firing threshold θ):

- the *feeding* input $e(t)$ (drive, activation, or stimulation of the network)

  that represents stimulations from outside the network and that changes the somatic resting potential by $u_e(t) = e(t) \ast h(t)$, where "$\ast$" denotes convolution. For most of the investigations, we consider $E := e(t) = \text{const.}$ which – in conjunction with the applied noise – simulates incoherent input from many weakly transmitting – for example "apical dendritic" – synapses (Fig. 3) and changes the somatic potential (Fig. 2) according to

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

- 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 $p_\nu(t) = \sum_\kappa s\left(t - t_\kappa\right)$ – with spike times t_κ – that arrive delayed by $\vartheta = \text{const.}$ at – for example "basal dendritic or somatic" – synapses from $2k$ neighboring units, i.e., from inside the network (Fig. 3). This input alters the somatic potential according to $u_a(t) = a(t) \ast h(t)$. Figure 1 shows the assumed exponentially decaying action potential $s(t)$ of time constant $\tau_{AP} = 0.144\,\text{ms}$ and its post-synaptic response at the soma $u_{PSP}(t)$ normalized to the *coupling strength w*.

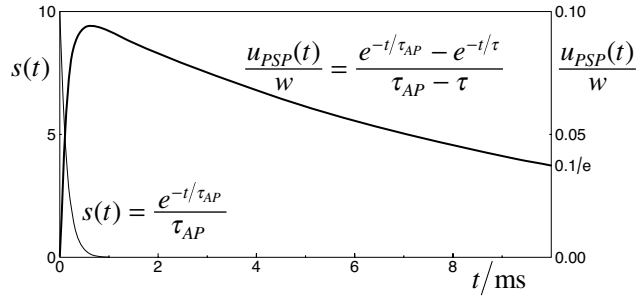

Figure 1. Action potential $s(t)$ and normalized post-synaptic potential $u_{PSP}(t)/w$

- the *noise* input $n(t)$ (individually computed for each unit)

  is a random process with uniformly distributed values from the range $\pm E/2$ that mimicks fluctuations $u_n(t) = n(t) \ast h(t)$ of the somatic potential.

In total, the change of the sub-threshold somatic potential from the resting potential is

$$u(t) = u_e(t) + u_a(t) + u_n(t) = \left[e(t) + a(t) + n(t)\right] \ast h(t)\,.$$

(Neither synaptic habituation nor non-linear synaptic transmission or interaction is considered.) An action potential $s(t)$ is triggered, when the somatic potential exceeds the threshold θ, and 1 ms later the somatic potential is set to the resting potential for a period of 0.5 ms before the integration can start again. The feeding input E is specified by the period T of the impulse train it evokes in a noise-free unit.

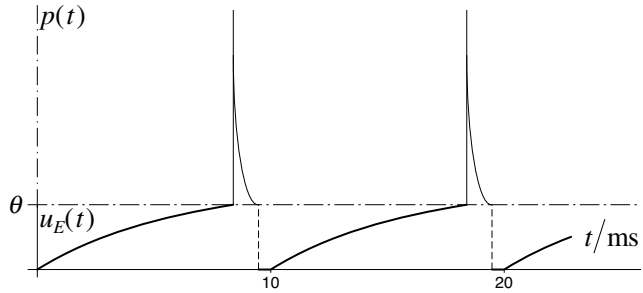

Figure 2. Typical periodic time-courses of the sub-threshold somatic depolarization $u_E(t)$ and of the associated spike train $p(t)$ for constant input E

3.2 The network

Figure 3 depicts the neighborhood of a formal neuron in the one-dimensional, single stage network. To avoid boundary problems in networks of manageable size the chain is cyclically closed. All of its N units are coupled in the same way: We consider either inhibitory or excitatory interconnections without direct feedback from units onto themselves. Every unit receives input from its immediate $k \ll N$ neighbors on either side with strengths $|w_\nu|$ that decrease 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 (ii) of Section 2). To characterize the strength of the interaction in the whole network, we introduce a neuron's *total coupling strength*

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

Of course, it is reasonable to demand for global stability which in turn necessitates an upper limit of the excitatory coupling strength that we choose to $W_{\text{crit}} = +0.78$. With this *critical coupling strength* a noiseless unit without feeding input starts spiking under synchronous unilateral input.

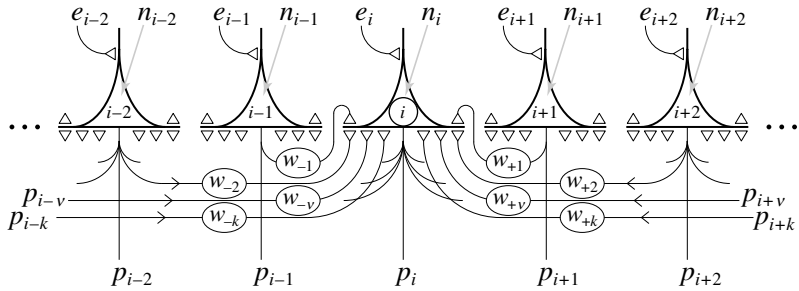

Figure 3. Local interconnection scheme of unit i in the one-dimensional network

Although we report about investigations of single stage or "single layer" networks, locally divergent forward coupling between "layers" – like that proposed by Moshe Abeles [16, Chapter 7] for the generation and transmission of synfire chains – appears more realistic for configurations without delay. Non-delayed lateral excitation can directly be achieved by forward coupling whereas non-delayed lateral inhibition requires the compensation of the delays between the direct excitatory signal and the divergent inhibitory ones that are inevitably relayed by inter-neurons.

3.3 Network simulation and measures of synchrony

We studied the *discrete* non-linear dynamics of the networks, i.e., we performed simulations on a digital computer with temporal resolution δt. As a consequence, the zero delay in the lateral links can only be approximated, i.e., one must accept the average intrinsic delay $\vartheta_0 = \delta t/2$. For most of the investigations reported here, all units received the same constant feeding input for times $t \geq 0$. However, their initial somatic potentials were individually set to uniformly distributed random values from the "refractory potential to threshold"-range. Unless stated otherwise, the values of the network parameters are:

$$\delta t_S = 0.1\text{ms}; \qquad N_S = 64; \qquad k_S = 8; \qquad E_S \text{ so that } T = 10\text{ms};$$

This standard setting turned out to be adequate for most of the investigations and does not represent an extraordinary choice. The effect of deviations from this standard on the quality of synchronization is explicated elsewhere [17].

To quantify different states of synchrony, we define the instantaneous *spike density* $S(t)$ which is the total spike activity of the ensemble in a running window of duration $M \cdot \delta t = 1\text{ms}$, divided by the maximum possible activity in this interval (cf. Fig. 5).

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

Consequently, $S = 1$ denotes perfect synchrony, i.e., all N units have triggered action potentials at the same time. (We also use the *envelope function* $\hat{S}(t)$ of the spike density.) To compare the synchrony obtained with different settings, we introduce the *qual-*

ity factor η_x which is the mean of the maximum spike density in an interval of 50 ms computed from 50 runs. (We display η_{50} and η_{200}.)

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

For an assessment of the quality factor, we provide the *reference quality* η_{ref} that results from "synchrony by chance" of uncoupled units. Because this 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 ensembles.

4. SIMULATION RESULTS

After the detailed description that is essential for judging the consequences of our findings, we now present a compilation of the main results in Figure 4. Obviously, significant synchrony can be achieved in networks with inhibitory or excitatory lateral interconnections. (Figure 5 shows examples of the corresponding spike densities.) Synchronization relies on the non-linear characteristic of the somatic integration: The efficacy of post-synaptic potentials 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 later/earlier ones.

On the whole and with regard to the delays, the networks' synchronization behavior is complementary:

ϑ/ms	Inhibitory coupling ($W < 0$)	Excitatory coupling ($W > 0$)
0.05	desynchronization $\eta_{200} < \eta_{\text{ref}}$	excellent synchrony for $W > 0.2$
2.00	good synchrony for $W < -1.0$	no significant synchrony $\eta_{200} \approx \eta_{\text{ref}}$

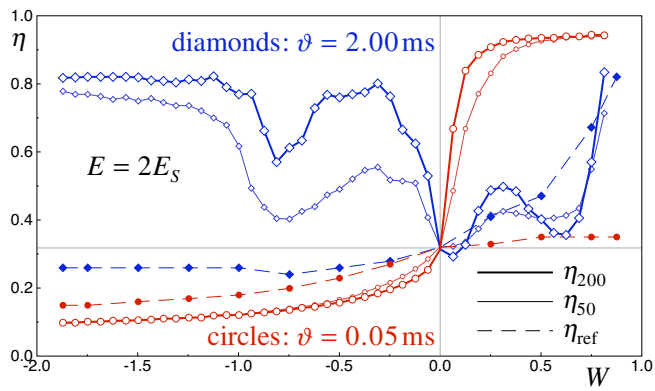

Figure 4. Quality of synchronization η as a function of the total coupling strength W for transmission delays $\vartheta = 0.05$ ms and $\vartheta = 2.00$ ms (from [7])

While non-delayed excitatory coupling and strong activation $E = 2E_S$ causes the spike rate $r_0(W_{\text{crit}}) \approx 195/\text{s}$, 2 ms delay results in pathological $r_2(W_{\text{crit}}) \approx 400/\text{s}$. The former implies that retarded action potentials are accelerated up to synchrony and the latter that additional impulses are generated. A comparable effect is observed for inhibition with $r_0(W=-1.87) \approx 60/\text{s}$ and $r_2(W=-1.87) \approx 142/\text{s}$. $[r_{\text{ref}} = r(W=0) \approx 180/\text{s}]$

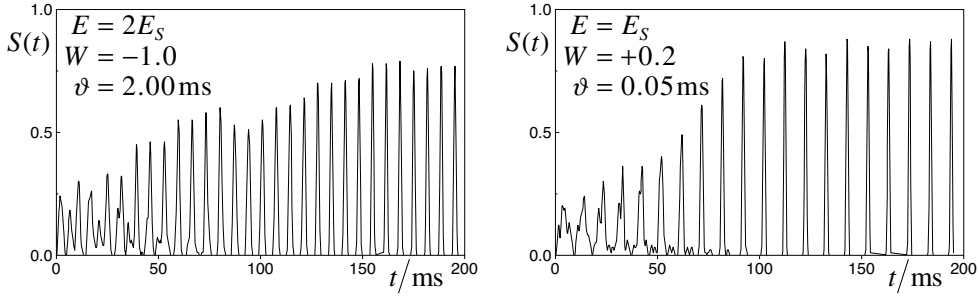

Figure 5. Examples of instantaneous spike densities $S(t)$ (note the different drive E)

Synchronization depends on the delay (graphs not shown): For inhibition ($W = -1.0$) and strong activation $E = 2E_S$ it continuously increases from desynchronization for $\vartheta = 0.05$ to good synchronization for $\vartheta = 2.00$ that is maintained, apart from 1 ms dips at multiples of $\Delta\vartheta = 6$ ms. However, for fairly weak excitation ($W = +0.2$) and standard activation $E = E_S$ it steeply decreases from excellent quality for $\vartheta = 0.05$ to the reference level for $\vartheta = 0.50$ where it stays, except for short peaks at multiples of $\Delta\vartheta = 6$ ms. These peaks decrease with ϑ and are less pronounced when considering measure η_{50}.

The response to a *linear downward sweep* of the feeding input $e(t)$ is 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 non-linear transfer characteristic $r[e(t)]$ of a coupled formal neuron.

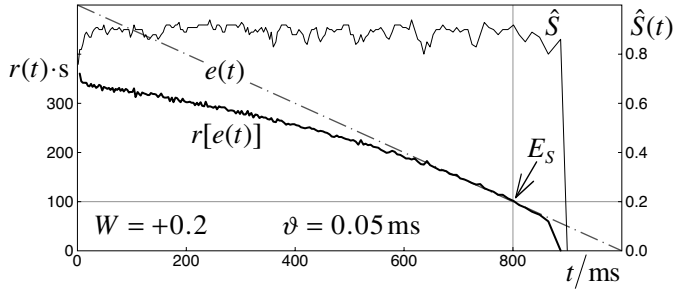

Figure 6. Envelope of the spike density $\hat{S}(t)$ and impulse rate $r(t)$ for a linear sweep of activation $e(t)$

5. DISCUSSION

According to our findings, synchronous spike activity results almost inevitably from homogeneously stimulated and in particular either excitatorily forward coupled or delayed (interneurons!) lateral inhibition networks. In contrast to the present euphoria about this *highly ordered* spatio-temporal behavior, we are not certain as to its relevance for the processing of neural signals (see Section 2): If a significant number of neurons are in fact acting as somatic coincidence detectors for complex spatio-temporal spike patterns, why then are synchronizing circuits required? However and aside from coincidence-detecting synapses, synchronous spike emission may turn out advantageous for reasons of neural self-organization.

After this more general consideration, we should like to return to the issue of synchronous aperiodic spike fronts. In addition to the results shown in Figure 6, we found that even abrupt changes in global activation do not significantly disturb an established synchrony. – Moreover, the time needed to reach a certain level of synchrony clearly depends on the spike rate, because spike synchronization happens stepwise.

In contrast to related work on excitatorily coupled networks, we have shown that neither non-linear synapses [18] nor different time constants for feeding and synchronizing inputs [19] are required for attaining reasonable synchrony. Although fully interconnected networks [20; 21] cannot truly be compared to the ones investigated here, they nevertheless show states similar to those observed in our study. Regarding comparisons, it may prove helpful to realize that our preferred excitatory total coupling strength of $W = +0.2$ means rather *weak coupling.*

We should like to conclude by indicating a general problem with temporally discrete simulations that arises from the fact that temporally discrete non-linear systems are not always approximations of their temporally continuous originals. Hence, results from such investigations must be regarded with utmost caution. So far, we could show a convergence for decreasing increments δt.

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