Stable Propagation of Activity Pulses in Populations of Spiking Neurons

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Abstract

We investigate the propagation of pulses of spike activity in a neuronal network with feed-forward couplings. The neurons are of the spike-response type with a firing probability that depends linearly on the membrane potential. After firing neurons enter a phase of refractoriness. Spike packets are described in terms of the moments of the firing-time distribution so as to allow for an analytic treatment of the evolution of the spike packet as it propagates from one layer to the next. Both analytic results and simulations show that depending on the synaptic coupling strength a stable propagation of the packet with constant wave form is possible. Crucial for this observation is neither the existence of a firing threshold nor a sigmoidal gain function – both are absent in our model – but the refractory behavior of the neurons.

1 Introduction

Recently, the propagation of sharp pulses of spike activity through various types of neuronal networks has attracted a lot of attention. There are basically two complementary scenarios where a temporally precise transmission of spikes has been investigated in model studies, namely spatially extended networks with distance-dependent couplings and layered feed-forward structures of pools of neurons. Spatially extended networks have properties similar to those of excitable media and exhibit, for example, solitary waves of spike activity [13, 6, 3, 11]. Layered feed-forward networks, also known as synfire-chains [1], can be seen as a discretized version of the former where the smooth propagation of a wave of activity is replaced by a discrete transmission of spikes from one layer to the next. Similar to solitary waves in spatially extended networks the transmission function for spikes can produce an attractive fixed point for the shape of the firing-time distribution in each layer. Such a "spike packet" can propagate in a stable way from one layer to the next [2, 15, 10, 5].

In this paper we consider a chain of M pools of identical neurons with feed-forward coupling. Each neuron is described by the Spike Response Model – a generalization of the integrate-and-fire model. The spike train of neuron i is formalized as a sum of δ functions, $S_i(t) = \sum_f \delta(t - t_i^f)$, where the firing times t_i^f of neuron i are labeled by an upper index f. The membrane potential u_i of a given neuron is the linear response to pre- and postsynaptic action potentials,

$$u_i(t, \hat{t}_i) = \sum_{j, j \neq i} \omega_{ij} \int_0^\infty \mathrm{d}t' \, \epsilon(t') \, S_j(t - t') + \eta(t - \hat{t}_i) \, . \tag{1}$$

Here, the response kernel ϵ describes the form of an elementary postsynaptic potential, ω_{ij} is the synaptic coupling strength, and η is a (negative) afterpotential that accounts for the reset of the membrane potential after the last spike at $\hat{t}_i = \max\{t_i^f | t_i^f < t \, , \; f = 1, 2, \dots\}$ and for refractoriness [9, 8, 12]. The influence of the last-but-one and earlier spikes is neglected so that spike triggering can be described by an input-dependent renewal process [4].

Noise is implemented in the model by a stochastic spike triggering mechanism. New spikes are defined through a stochastic process that depends on the value of the membrane potential. The probability of a spike to occur in the infinitesimal interval $[t, t + \mathrm{d}t)$ is

$$\mathrm{prob}\{\text{spike in } [t, t + \mathrm{d}t)\} = f[u_i(t, \hat{t}_i)] \, \mathrm{d}t \, . \tag{2}$$

The function f is called *escape rate* (or *hazard function*) [16]. For the sake of simplicity we assume a semi-linear dependence of the firing probability and the membrane potential,

$$f(u) = [u]_+ \, , \tag{3}$$

with $[u]_+ = u$ if $u > 0$ and $[u]_+ = 0$ elsewhere. This completes the definition of our single-neuron model.

If we assume that neuron i has fired its last action potential at time $\hat{t}_i$, we can calculate the probability $s_i(t, \hat{t}_i)$ that it will 'survive' without firing until time $t > \hat{t}_i$, viz.,

$$s_i(t, \hat{t}_i) = \exp\left\{ - \int_{\hat{t}_i}^{t} f[u_i(t, \hat{t}_i)] \right\} , \tag{4}$$

cf. [4, 7]. The probability density for the next firing time is thus

$$p_i(t, \hat{t}_i) = -\frac{\partial}{\partial t} s_i(t, \hat{t}_i) = f[u_i(t, \hat{t}_i)] \, \exp\left\{ - \int_{\hat{t}_i}^{t} f[u_i(t, \hat{t}_i)] \right\} . \tag{5}$$

We consider M pools containing N neurons each that are connected in a purely feed-forward manner, i.e., neurons from pool n project only to pool $n+1$ and there are no synapses between neurons from the same pool. We assume all-to-all connectivity between two successive pools with uniform synaptic weights $\omega_{ij} = \omega/N$. The membrane potential of a neuron i from pool $n+1$ is thus

$$\begin{aligned} u_i(t, \hat{t}_i) &= \frac{\omega}{N} \sum_{j \in \Gamma(n)} \int_0^\infty \epsilon(t') \, S_j(t - t') \, \mathrm{d}t' + \eta(t_i - \hat{t}_i) \\ &= \omega \int_0^\infty \epsilon(t') \, A_n(t - t') \, \mathrm{d}t' + \eta(t_i - \hat{t}_i) \, , \end{aligned} \tag{6}$$

with $i \in \Gamma(n+1)$, $\Gamma(n)$ the index set of all neurons that belong to pool n, and $A_n(t) = N^{-1} \sum_{j \in \Gamma(n)} S_j(t)$ the *population activity* of pool n.

The spike trains S_i and the population activity A_n are random variables. Each pool is supposed to contain a large number of neurons ($N \gg 1$) so that we can replace the population activity A_n in Eq. (6) by its expectation value $\bar{A}_n$, which is given by a normalization condition [7],

$$\int_{-\infty}^{t} s_n(t, \hat{t}) \, \bar{A}_n(\hat{t}) \, \mathrm{d}\hat{t} = 1 - s_n(t) \, . \tag{7}$$

Here, $s_n(t) = s_n(t, -\infty)$ accounts for those neurons that have been quiescent in the past, i.e., have not fired up to time t. The strong law of large numbers ensures that the population activity A_n converges in probability to $\bar{A}_n$ (in the weak topology) as the number of neurons in the pool goes to infinity, i.e.,

$$\mathrm{prob}\left\{ \lim_{N \to \infty} \int_{-\infty}^{\infty} A_n(t) \, \phi(t) \, \mathrm{d}t = \int_{-\infty}^{\infty} \bar{A}_n(t) \, \phi(t) \, \mathrm{d}t \right\} = 1 \, , \tag{8}$$

for any test function $\phi \in \mathcal{C}^\infty(\mathbb{R})$, cf. e.g. [14].

2 Pulse Propagation

Simulation studies suggest that a pronounced refractory behavior is required in order to obtain a stable propagation of a spike packet from one layer to the next [5, 10]. If neurons were allowed to fire more than once within one spike packet the number of spikes per packet and therewith the width of the packet would grow in each step. Therefore, we use a strong and long-lasting after potential η so that each neuron can fire only once during each pulse. The survivor function thus equals one for the duration τ_{AP} of the afterpotential, i.e., $s_n(t,\hat{t}) = 1$ for $0 < t - \hat{t} < \tau_{\mathrm{AP}}$ and τ_{AP} being large as compared to the typical pulse width. Differentiation of Eq. (7) with respect to t (and dropping bars in order to keep notation simple) leads to

$$A_n(t) = -\frac{\partial}{\partial t} s_n(t) = f[u_n(t)]\, \exp\left\{ -\int_{-\infty}^{t} f[u_n(t')]\, \mathrm{d}t' \right\} , \tag{9}$$

with

$$u_n(t) = \omega \int_0^{\infty} \epsilon(t')\, A_{n-1}(t-t')\, \mathrm{d}t' . \tag{10}$$

Equation (9) provides an explicit expression for the firing-time distribution $A_n(t)$ in layer n as a function of the time course of the membrane potential. The membrane potential $u_n(t)$, in turn, depends on the time course of the activity $A_{n-1}(t)$ in the previous layer, as shown in Eq. (10). Both Eq. (9) and Eq. (10) can easily be integrated numerically; an analytic treatment, however, is difficult, even if a particularly simple form of the response kernel ϵ is chosen. Following [5] we therefore concentrate on the first few moments of the firing-time distribution in order to characterize the transmission properties. More precisely, we approximate the firing-time distribution $A_{n-1}(t)$ by a gamma distribution and calculate – in step (i) – the zeroth, first, and second moment of the resulting membrane potential in the following layer n. In step (ii), we use these results to approximate the time course of the membrane potential by a gamma distribution and calculate the moments of the corresponding firing-time distribution in layer n. We thus obtain an analytic expression for the amplitude and the variance of the spike packet in layer n as a function of amplitude and variance of the spike packet in the previous layer.

In step (i) we assume that the activity $A_{n-1}(t)$ in layer $n-1$ is given by a gamma distribution with parameters α_{n-1} and λ_{n-1}, i.e.,

$$A_{n-1}(t) = a_{n-1}\, \gamma_{\alpha_{n-1},\lambda_{n-1}}(t) . \tag{11}$$

Here, a_{n-1} is the portion of neurons of layer $n-1$ that contribute to the spike packet, $\gamma_{\alpha,\lambda}(t) = t^{\alpha-1}\, \mathrm{e}^{-t/\lambda}\, \Theta(t)/[\Gamma(\alpha)\, \lambda^{\alpha}]$ the density function of the gamma distribution, Γ the complete gamma function, and Θ the Heaviside step function with $\Theta(t) = 1$ for $t > 0$ and $\Theta(t) = 0$ else. The mean μ and the variance σ^2 of a gamma distribution with parameters α and λ are $\mu = \alpha\,\lambda$ and $\sigma^2 = \alpha\,\lambda^2$, respectively.

The membrane potential $u_n(t)$ in the next layer results from a convolution of A_{n-1} with the response kernel ϵ. This is the only point where we have to refer explicitely to the shape of the ϵ kernel. For the sake of simplicity we use a normalized α function,

$$\epsilon(t) = \frac{t}{\tau^2}\, \mathrm{e}^{-t/\tau}\, \Theta(t) \equiv \gamma_{2,\tau}(t)\,, \tag{12}$$

with time constant τ. The precise form of ϵ is not important and similar results hold for a different choice of ϵ.

We want to approximate the time course of the membrane potential by a gamma distribution $\gamma_{\tilde{\alpha}_n,\tilde{\lambda}_n}$. The parameters[1] $\tilde{\alpha}_n$ and $\tilde{\lambda}_n$ are chosen so that the first few moments of the distribution are identical to those of the membrane potential, i.e.,

$$u_n(t) \approx \tilde{a}_n\, \gamma_{\tilde{\alpha}_n,\tilde{\lambda}_n}(t)\,, \tag{13}$$

with

$$\int_0^\infty t^n\, u_n(t)\, \mathrm{d}t \stackrel{!}{=} \int_0^\infty t^n\, \tilde{a}_n\, \gamma_{\tilde{\alpha}_n,\tilde{\lambda}_n}(t)\, \mathrm{d}t\,, \qquad n \in \{0,1,2\}\,. \tag{14}$$

As far as the first two moments are concerned, a convolution of two distributions reduces to a mere summation of their mean and variance. Therefore, the convolution of A_{n-1} with ϵ basically translates the center of mass by 2τ and increases the variance by $2\tau^2$. Altogether, amplitude, center of mass, and variance of the time course of the membrane potential in layer n are

$$\left.\begin{aligned} \tilde{a}_n &= \omega\, a_{n-1}\,, \\ \tilde{\mu}_n &= \mu_{n-1} + 2\tau\,, \\ \tilde{\sigma}_n^2 &= \sigma_{n-1}^2 + 2\tau^2\,, \end{aligned}\right\} \tag{15}$$

respectively. The parameters $\tilde{\alpha}_n$ and $\tilde{\lambda}_n$ of the gamma distribution are directly related to mean and variance, viz., $\tilde{\alpha}_n = \tilde{\mu}_n^2/\tilde{\sigma}_n^2$, $\tilde{\lambda}_n = \tilde{\sigma}_n^2/\tilde{\mu}_n$.

In step (ii) we calculate the firing-time distribution that results from a membrane potential with time course given by a gamma distribution as in Eq. (13). We use the

[1]We use a tilde in order to identify parameters that describe the time course of the membrane potential. Parameters without a tilde refer to the firing-time distribution.

same strategy as in step (i), that is, we calculate the first few moments of the firing-time distribution and approximate it by the corresponding gamma distribution,

$$A_n(t) \approx a_n \, \gamma_{\alpha_n,\lambda_n}(t)\,. \tag{16}$$

The zeroth moment of $A_n(t)$ (i.e., the portion of neurons in layer n that participate in the activity pulse) can be casted in a particularly simple form; the expressions for higher order moments, however, contain integrals that have to be calculated numerically. For amplitude, center of mass, and variance of $A_n(t)$ we find

$$\left.\begin{aligned} a_n &= 1 - \mathrm{e}^{-\tilde{a}_n}\,, \\ \mu_n &= m_n^{(1)}\,, \\ \sigma_n^2 &= m_n^{(2)} - \left[m_n^{(1)}\right]^2\,, \end{aligned}\right\} \tag{17}$$

with

$$\begin{aligned} m_n^{(k)} &= \left(1 - \mathrm{e}^{-\tilde{a}_n}\right)^{-1} \int_0^\infty u_n(t)\, \exp\left[-\int_{-\infty}^t u_n(t')\,\mathrm{d}t'\right] t^k\,\mathrm{d}t \\ &= \frac{\tilde{a}_n\,\tilde{\lambda}_n^k}{(1-\mathrm{e}^{-\tilde{a}_n})\,\Gamma(\tilde{\alpha}_n)} \int_0^\infty \exp\left[-t - \tilde{a}_n\,\Gamma(\tilde{\alpha}_n,0,t)/\Gamma(\tilde{\alpha}_n)\right]\, t^{k-1+\tilde{\alpha}_n}\,\mathrm{d}t \end{aligned} \tag{18}$$

being the kth moment of the firing-time distribution (9) that results from a gamma-shaped time course of the membrane potential. $\Gamma(z,t_1,t_2) = \int_{t_1}^{t_2} t^{z-1}\,\mathrm{e}^{-t}\,\mathrm{d}t$ is the generalized incomplete gamma function. The last equality in Eq. (18) has been obtained by substituting $\tilde{a}_n\,\gamma_{\tilde{\alpha}_n,\tilde{\lambda}_n}(t)$ for $u_n(t)$.

A combination of Eqs. (15) and (17) yields explicit expressions for the parameters (a_n,μ_n,σ_n) of the firing-time distribution in layer n as a function of the parameters in the previous layer. The mapping $(a_{n-1},\mu_{n-1},\sigma_{n-1}) \to (a_n,\mu_n,\sigma_n)$ is closely related to the neural transmission function for pulse-packet input as discussed in [5].

Particularly interesting is the iteration that describes the amplitude of the spike packet,

$$a_n = 1 - \mathrm{e}^{-\omega\, a_{n-1}}\,, \tag{19}$$

which is independent of the shape of the spike packet. If $\omega \le 1$, the mapping $a_{n-1} \to a_n$ has a single (globally attractive) fixed point at $a=0$. In this case no stable propagation of spike packets is possible since any packet will finally die out[2]. For $\omega > 1$ a second fixed point at $a_\infty \in (0,1)$ emerges through a pitchfork bifurcation. The new fixed point is stable and its basin of attraction contains the open interval $(0,1)$. The fact that the all-off state

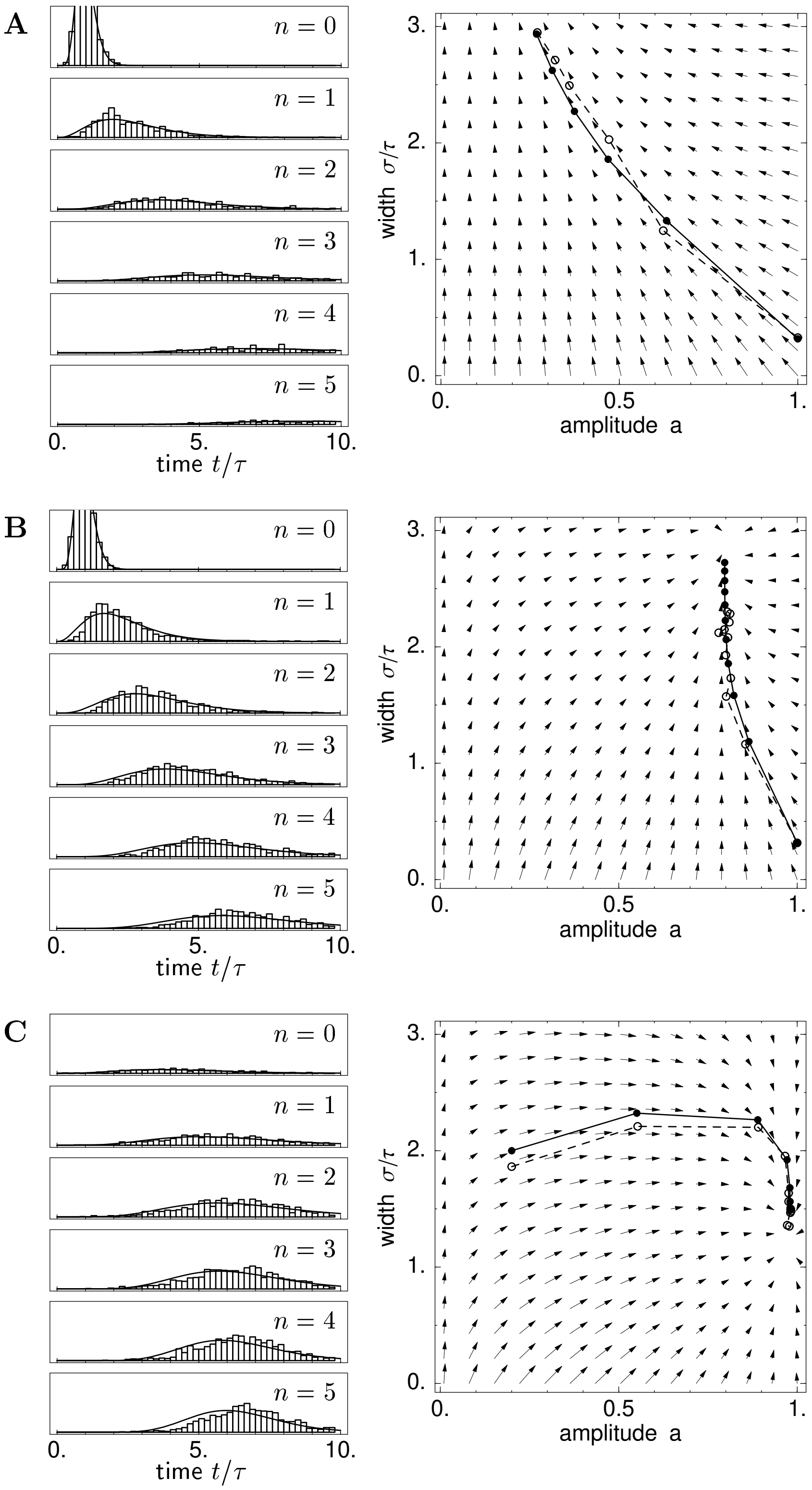

Figure 1: see next page.

at $a = 0$ is unstable for $\omega > 1$ is related to the fact that there is no real firing threshold in our model.

Figure 1 shows examples of the propagation of a spike packet for various synaptic coupling strengths and initial conditions. Theoretical predictions based on Eqs. (15) and (17) are compared to simulations of a network with $N = 1000$ neurons per layer. In each sub-figure a series of bar charts shows the firing-time distribution of neurons from layers $n = 0$ to $n = 5$. The flow field illustrates the evolution of the amplitude and the width of the spike packet as it propagates from one layer to the next.

In Fig. 1A a small coupling strength has been chosen ($\omega = 1$) so that iteration (19) has only a single fixed point at $a = 0$. Therefore, any spike packet will die out whatever the initial firing-time distribution in layer $n = 0$. Figure 1B is another example for $\omega = 2$. Here, the iteration (19) has a stable fixed point at $a \approx 0.80$ and both simulations and theory show that this fixed point corresponds to a stable propagation of spike packets from one layer to the next. Finally, in Fig. 1C ($\omega = 4$) we demonstrate that the iteration of $(a_{n-1}, \mu_{n-1}, \sigma_{n-1}) \to (a_n, \mu_n, \sigma_n)$ converges to a mere translation of the spike packet with an approximately fixed wave form. This wave form is globally attractive so that even a weak and broadly tuned initial firing-distribution will become sharper and form a narrow spike-packet.

Figure 1 (previous page): Propagation of spike packets through a feed-forward network. **A**. Evolution of the firing-time distribution of a spike packet as it propagates from one layer to the next ($n = 0, 1, \ldots, 4$). The neurons in layer $n = 0$ are driven by an external input that creates a sharp initial spike-packet given by a gamma distribution with $\alpha_0 = 10$ and $\lambda_0 = 0.1$. Initial amplitude is $a_0 = 1$. The bars (bin width 0.2) represent the results of a simulation with $N = 1000$ neurons per layer; the solid line is the firing-time distribution as predicted by the theory; cf. Eqs. (15) and (17). The "flow field" to the right characterizes the transmission function for spike-packets in terms of their amplitude a_n and width $\sigma_n = \sqrt{\alpha_n}\,\lambda_n$. Open symbols connected by a dashed line represent the simulations shown to the left, filled symbols connected by solid lines represent the corresponding theoretical trajectories. Neurons between layers are only weakly coupled ($\omega = 1$) so that the packet will fade out. Time is given in units of the membrane time constant τ. **B**. Same as in A but with increased coupling strength $\omega = 2$. There is an attractive fixed point of the flow field at $a = 0.80$ and $\sigma = 2.9$ that corresponds to the stable wave form of the spike packet. **C**. Similar plots as in A and B but with a strong coupling strength ($\omega = 4$). Initial stimulation is weak ($a_0 = 0.2$) and broad ($\sigma_0 = 2$). As the packet propagates through a few layers it quickly reaches a stable wave form with amplitude $a = 0.98$ and $\sigma = 1.5$.

[2] The decay of the activity is exponential in n if $\omega < 1$; for $\omega = 1$ the decay is polynomial in n.

3 Discussion

Any information processing scheme that relies on the precise timing of action potentials obviously requires a means to transmit spikes without destroying their temporal structure. In this paper we have shown that, despite the noise in the spike generating mechanism, packets of (almost) synchronous spikes can propagate in a feed-forward structure from one layer to the next so that their width is preserved provided that the synaptic coupling strength is sufficiently large. This property is not based on a non-linear transfer function or a firing threshold – both are absent in our model – but on the refractory behavior of the neurons. Noise and broad postsynaptic potentials tend to smear out initially sharp spike packets. If, however, the synaptic coupling is strong enough, then postsynaptic neurons will start firing already during the raising phase of their membrane potential. If, in addition, these neurons show pronounced refractory behavior, then firing will cease even before the postsynaptic potentials have reached their maximum. With respect to a precise timing, refractoriness thus counteracts the effects of noise and synaptic transmission.

As a consequence of the linear transfer function no bistability could be observed in our model. Depending on the synaptic coupling strength there is always only one stable fixed point – either the all-off state or a propagating pulse. Recent simulation studies [5], however, have shown that a slightly more general model with a non-linear transfer function can indeed exhibit bistability where neurons are either firing asynchronously at a low rate or participate in the transmission of a sharp spike packet. This bistability does not only seem to be necessary to explain experimental observations but also extends the computational power of the whole system considerably.

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