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Universality in neural networks: the importance of the 'mean firing rate'

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Abstract. We present a general analysis of highly connected recurrent neural networks which are able to learn and retrieve a finite number of static patterns. The arguments are based on spike trains and their interval distribution and require no specific model of a neuron. In particular, they apply to formal two-state neurons as well as to more refined models like the integrate-and-fire neuron or the Hodgkin–Huxley equations. We show that the mean firing rate defined as the inverse of the mean interval length is the only relevant parameter (apart from the synaptic weights) that determines the existence of retrieval solutions with a large overlap with one of the learnt patterns. The statistics of the spiking noise (Gaussian, Poisson or other) and hence the shape of the interval distribution does not matter. Thus our unifying approach explains why, and when, all the different associative networks which treat static patterns yield basically the same results, i.e., belong to the same universality class.

1 Introduction

Whenever networks with formal neurons have been studied (McCulloch and Pitts 1943; Little 1974; Hopfield 1982; Amit et al. 1985), there have been doubts concerning the relevance of the results to neurobiology. The assumption of formal two-state neurons *à la* McCulloch and Pitts seems extremely simplified compared to the rich behaviour of real neurons. To bring theory closer to neurobiology, a number of more refined models of a neuron have been incorporated in a network description (Buhmann and Schulten 1986; Horn and Usher 1989; Treves 1990; Gerstner 1990; Amit et al. 1991; Amit and Tsodyks 1991; Gerstner and van Hemmen 1992). The results show, however, that the basic associative features of the network remain unchanged with respect to networks with McCulloch-Pitts neurons, even though more details have been added to the description of the neurons. This leads to the question *why* the network behaviour always exhibits the same collective features, despite the fact that quite different models of a neuron have been used.

On the other hand, it is known from general results of statistical physics that many features of the elements of a network do not matter for its collective behaviour as long as the system is large enough. Only a couple of fundamental parameters determine how the system behaves qualitatively and gather the various models into a small number of universality classes. In theoretical approaches to neural networks, the mean firing rate or mean firing frequency is often considered as such a fundamental parameter and used in a macroscopic description of the network states (Wilson and Cowan 1972; Hopfield 1984; Schuster and Wagner 1990; Abbott 1991). This is related to the idea that information on the environment is encoded in biological neural systems by the mean firing rate of the neurons (Adrian 1926), an idea which has been put into question recently (Bialek et al. 1991).

It is important to realize that the arguments presented in this paper do not rely on any a priori assumption concerning the importance of the mean firing rate. Rather it is the *result* of our considerations that in a fully connected (recurrent) network that functions as an associative memory for stationary patterns, i.e., in generalized associative networks of the 'Little-Hopfield'-type, the mean firing rate is the *only* relevant parameter of the network. Second-order features of the spiking, such as the exact distribution or the variance of the spiking intervals, do not matter.

The above universality is a result that is independent of any particular model of the neurons and is based only on the assumption of high connectivity. Thus, all model neurons which have the same gain function (mean firing rate versus input current) will lead to the same stationary states. This generalizes results of Hopfield (1984) and Kühn et al. (1991) who have argued in favor of the equivalence of formal

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neurons and those with graded response. The equations that we derive are identical to those of the mean-field solution which is often used as an *approximation* in a simplified theory of neural networks. Our results for the stationary solutions, however, are exact.

An argument similar to ours has been put forward by Amit and Tsodyks (1991) who also use the notion of mean firing rate in the context of neural nets. But while they appeal to the high connectivity in biological networks to justify the use of the 'law of large numbers' as a good approximation to reality, we present an exact derivation of the mean-field results and show that there is a controlled and well-defined way to go from spike trains to a description by gain functions.

In the next section we calculate the interval distribution and the mean firing rate for a spike train of a single neuron. In the subsequent Sect. 3 we treat a network of neurons and show analytically that the mean firing rate and the strength of the synaptic feedback are the only variables that determine the existence of retrieval states. As an application of this general result we analyze some examples of networks with various model neurons including the Hodgkin–Huxley model (Sect. 4). The discussion in Sect. 5 concentrates on the validity and limitations of the assumptions we made in the derivation of the results of Sect. 3. We close our considerations with a number of conclusions in Sect. 6.

2 Spike trains of a single neuron

In an experimental setup, stationary input conditions can be defined by a constant input current forced upon the neuron by an electrode into the soma. The spike train can then be recorded by an extracellular electrode near the axon. After the current is switched on, it takes a few spikes before the neuron adapts and regular spiking evolves. Depending on the input current I, a spike train length T (Fig. 1) contains only a few spikes (low 'firing rate') or many spikes (high 'firing rate'). To analyze such a spike train, we can plot its *interval distribution* $D_I(s)$ where the subscript I refers to the dependence upon the input current I (Fig. 2). In this plot, $D_I(s)\Delta s$ is the probability that a neuron fires between s and $s+\Delta s$ *given* that the last spike has occurred at $s=0$. Thus it is the probability that the interval between two subsequent spikes has a length between s and $s+\Delta s$ (see Fig. 1). The normalization of probabilities yields $\int_0^\infty D_I(s)\,\mathrm{d}s = 1$.

We find that long intervals are extremely unlikely and so are very short intervals. The latter fact is due to the refractory period of the neuron which prevents the emission of another spike immediately after firing. Roughly speaking, the neuron remembers the emission of the last spike. This idea can be made more precise, if we look at the conditional probability $P_I(s)$ that a neuron has not yet spiked at time s under the condition that there was a spike at $s=0$. The '*survivor function*' (Perkel et al. 1967) $P_I(s)$ remains at 1 (i.e., no firing can occur) during the absolute refractory period and decays to zero afterwards (Fig. 2, bottom). The two quantities $D_I(s)$ and $P_I(s)$ are connected by the relation

$$D_I(s) = -\frac{\mathrm{d}}{\mathrm{d}s} P_I(s)\,. \tag{1}$$

To see this, we consider the probability to fire between s and $s+\Delta s$,

$$D_I(s)\Delta s = P_I(s) - P_I(s+\Delta s) = -\frac{\mathrm{d}}{\mathrm{d}s} P_I(s)\Delta s + O(\Delta s)^2\,. \tag{2}$$

Since each of the two functions can be derived from the other, it is completely equivalent whether we describe neuronal spiking by an interval distribution or a survivor function. Both contain most of the information of a spike train under stationary conditions. In particular, we can get the averaged quantities, especially the mean interval length. It is straightforward to define the *mean*

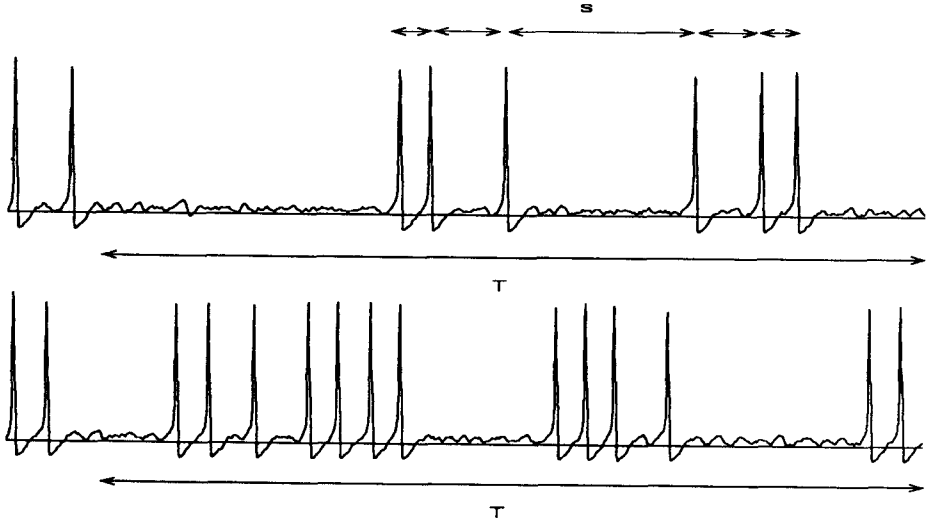

Fig. 1. *Spike trains*: Only a few spikes occur at low input current (*top*), whereas at higher input currents spiking is more frequent (*bottom*). Spikes are counted during a measurement period of length T; intervals between subsequent spikes have a variable length s

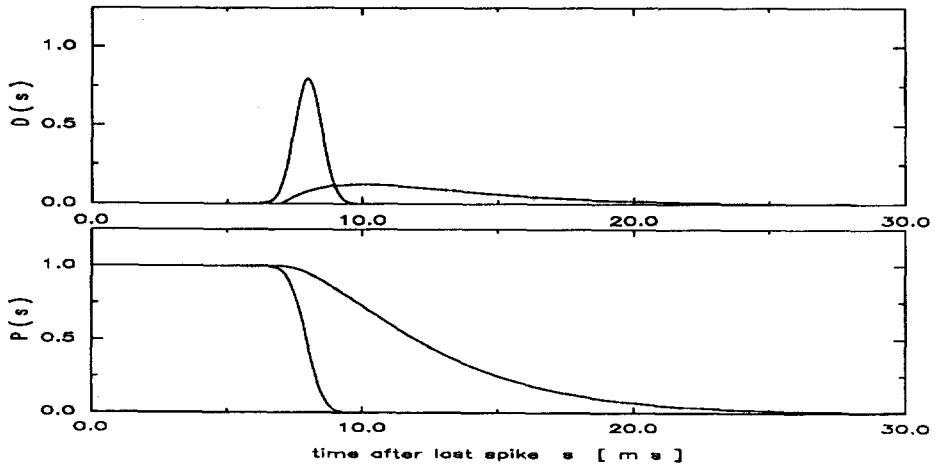

Fig. 2. *Spike train analysis*: The interval distribution $D_I(s)$ (*top*) gives the differential probability to find a interval of length between s and $s + ds$. The 'survivor' function $P_I(s)$ (*bottom*) is the probability that a neuron stays quiescent during a time s after firing of a spike at $s = 0$. Both functions are given for a regularly firing neuron (sharply peaked interval distribution and set-like survivor function) and a randomly firing neuron (long exponential tail)

interval length

$$\bar{s}(I) = \int_0^\infty s \times D_I(s)\, \mathrm{d}s = \int_0^\infty s\left(-\frac{\mathrm{d}}{\mathrm{d}s} P_I(s)\right) \mathrm{d}s$$

$$= \int_0^\infty P_I(s)\, \mathrm{d}s\,. \tag{3}$$

The *mean firing rate* is then defined as the inverse of the mean interval length (cf Perkel et al. 1967)

$$f(I) = \bar{s}(I)^{-1}\,. \tag{4}$$

This definition is useful for theoretical considerations as well as for experimental studies. Since for a long measurement time T the mean interval length is given directly by the total time T divided by the number of events (spikes) Z, we have $\bar{s}(I) = T/Z(I)$ and therefore

$$f(I) = \frac{Z(I)}{T}\,. \tag{5}$$

So there is a simple procedure to determine the mean firing rate at a given input current. We count the number of spikes and divide by the measurement time. The mean firing rate as a function of I is called the *gain function* of the neuron. Typical gain functions of real neurons are plotted in Fig. 3 (Jahnsen and Llinas 1984).

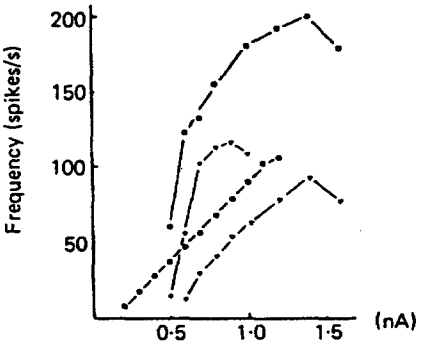

Fig. 3. *Gain function*: Experimental gain functions of thalamic neurons in the Guinea-pig. Taken from Jahnsen and Llinas (1984)

Note that if we talk of 'mean firing frequency' instead of 'mean firing rate' a different definition should be adopted. In this case the procedure would be to determine the 'instantaneous frequencies' (e.g. the inverse of an interval length) first and take the mean afterwards, a procedure that yields a different result. Our derivation in the next section, however, shows that the mean firing rate as defined above is the relevant variable in the context of neural nets.

To this end, it is convenient to introduce the activity A. If a neuron is prepared in a state of input current I, we can ask for the expectation value $A[I(t), t]\Delta t$ of finding a spike in a short interval Δt. In the case of a stationary input, i.e., $I(t) \equiv I$, the expectation value is simply the mean number of spikes per unit time multiplied by Δt, so that

$$A[I(t), t] \equiv f(I)\,. \tag{6}$$

If, however, the input varies in time, we can still ask for the expectation value of firing events, but to give this question a precise meaning we have to consider a large ensemble of n identically prepared neurons. The *activity* $A[I(t), t]$ is then defined by

$$A[I(t), t] = \lim_{\Delta t \to 0} \lim_{n \to \infty} n^{-1} Z/\Delta t, \tag{7}$$

where Z gives the number of spikes of the ensemble during the time between t and $t + \Delta t$.

3 The network

3.1 Definition of the model

The associative retrieval qualities of a neural system are not the result of any individual neuron but rather the collective effect of a whole network of neurons. To get some insight into the macroscopic properties of a neural system, we have to define a network that is appropriate for formal considerations and takes into account some relevant neurobiological facts. It is, of course, practically impossible to reach both aims – analytical transparency *and* detailed neurobiology – at the same

time. In the approach presented below, only a few restrictions are posed on the type and characteristics of the neurons. Thus the reader may imagine any realistic model of a biological neuron. We are, however, fairly strict on the type of connection topology and synaptic weights that we assume. The limitations of these assumptions are pointed out in this section and will be discussed more thoroughly in Sect. 5.

To keep track of the spikes, we describe the spike train of each neuron by a sequence of δ-functions

$$\sigma_i(t) = \sum_{k_i=1}^{n_i} \delta(t - t_{k_i}), \tag{8}$$

where the t_{k_i} with $1 \leqslant k_i \leqslant n_i$ and $0 \leqslant t_{k_i} \leqslant t$ are the spiking times of neuron i. As long as we assume linear dendritic summation (as we do below), (8) is just a convenient way to describe the spikes and includes no additional approximation, because the only thing that matters is the effect of the spikes on other neurons and not the shape of the spikes themselves. This effect is described by the synaptic weights and the time course of the postsynaptic response in the soma of the receiving neuron. Let us assume that neuron j has fired a spike at $t = 0$ and is connected to neuron i via a synapse of strength J_{ij}. The *current* from neuron j to neuron i is then

$$I_i(t) = J_{ij}\epsilon(t) \tag{9}$$

where $\epsilon(\tau)$ is the normalized current response function ($\int_{\tau=0}^{\infty} \epsilon(\tau)\, d\tau = 1$). It is a measurable quantity with some typical rise and decay time in the *ms*-range. The argument τ measures the time since the emission of the spike at the soma of neuron j. Thus $\epsilon(\tau)$ also includes the axonal transmission delay of the spike travelling from neuron j to neuron i (Fig. 4). We assume that the shape of $\epsilon(\tau)$ is independent of the particular pair of neurons or, equivalently, that all specific synaptic properties are in the efficacy factor J_{ij} only. In a fully connected network of N neurons the total current into neuron i is then given by

$$I_i(t) = \sum_{j=1}^{N} J_{ij} \int_0^{\infty} d\tau \epsilon(\tau)\sigma_j(t - \tau). \tag{10}$$

As we have mentioned before, we assume that the contributions of the neurons add up linearly (spatial summation).

At this point, it may be appropriate to recall and discuss the assumptions made so far. We have introduced a network of neurons which are coupled by synaptic weights. The most important assumption here is that the synaptic signals which are collected by the postsynaptic neuron add up in a linear fashion. Thus all nonlinearities of the dendrites, e.g., effects of the reversal potential and shunting inhibition, are excluded; see Abbott (1991) and Ekeberg (1991) for a model of these effects. Under the assumption of linear synaptic summation, the shape of the presynaptic spikes is irrelevant. This allows us to describe the spikes as a sequence of δ-functions (8). The second assumption in our description of the network is that all neurons and synpases are taken to be identical. We thus neglect that various types of neurons and synapses are found in the brain, in particular we neglect the effects of synaptic placement in terms of dendritic distance to the soma. In Sect 5.2 we will show how this assumption can be dropped and various types of neuron and synapse can be included.

A third and strong assumption concerns the synaptic efficacy. Here we take the simple Hebbian learning matrix

$$J_{ij} = \frac{2J_0}{N} \sum_{\mu=1}^{q} \xi_i^{\mu}\xi_j^{\mu}, \tag{11}$$

where J_0 is a parameter and $\mu = 1, \ldots, q$ labels the patterns. Each pattern μ is a set of independent, identically distributed random variables $\{\xi_i^{\mu}, i = 1, \ldots, N\}$ which assume the values ± 1 with equal probability. Furthermore q is fixed and finite. This choice of the synaptic matrix is certainly a strong simplification, but we have voted for it to keep the argument transparent and allow a comparison to other theoretical models. A generalization to patterns of arbitrary activity and asymmetric synaptic connections is straightforward; see Sect 5.1 for some details.

To summarize this subsection, we have introduced a model network and discussed the assumptions concerning the theoretical description of the model. These assumptions might seem strong as compared to the variety of details and phenomena found in biological systems and the model is certainly not a fully realistic description of the cortex. If, however, the model Ansatz is contrasted with traditional theoretical approaches to associative memory in recurrent neural nets (Little 1974; Hopfield 1982; Amit et al. 1985), it compares favorable in terms of biological realism. In particular, a postsynaptic response function is included into the description of the synaptic transmission and the type of model neuron ('binary', 'graded-response', or 'spiking')

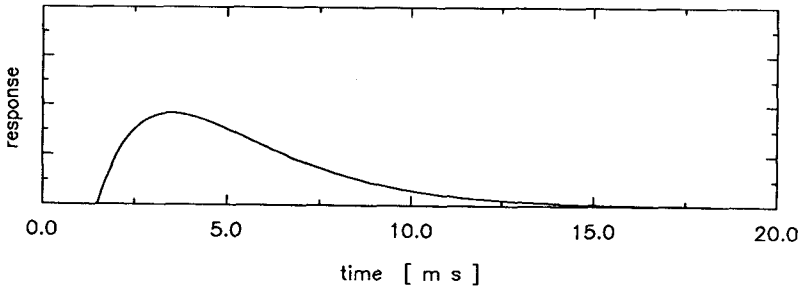

Fig. 4. *Postsynaptic current*: The synaptic response function $\epsilon(\tau)$ gives the time course of the current into the soma of the postsynaptic neuron after the firing of a presynaptic neuron at $\tau = 0$

need not be specified. This general model is solved in terms of stationary retrieval solutions in the following subsection.

3.2 Derivation of the equations for the mean firing rates

To give an analytical solution of the network behaviour, it is convenient to divide the ensemble of neurons into *sublattices* $L(\mathbf{x})$ (van Hemmen et al. 1986; van Hemmen and Kühn 1986) of those neurons that have learnt the same 'information', i.e., $L(\mathbf{x}) = \{i; \boldsymbol{\xi}_i = x\}$ where $\boldsymbol{\xi}_i$ is the information $(\xi_i^\mu;\ 1 \leqslant \mu \leqslant q)$ which has been presented to neuron i. Thus, the sublattice $L(\mathbf{x})$ contains all neurons i with $\boldsymbol{\xi}_i = \mathbf{x}$. Let the number of neurons in the sublattice $L(\mathbf{x})$ be $|L(\mathbf{x})|$. With these abbreviations we can write the current into neuron i (Herz et al. 1988)

$$I_i(t) = 2J_0 \sum_{\mu=1}^{q} \xi_i^\mu \int_0^\infty \mathrm{d}\tau \epsilon(\tau) \frac{1}{N} \sum_{j=1}^{N} \xi_j^\mu \sigma_j(t-\tau)$$

$$= 2J_0 \sum_{\mu=1}^{q} \xi_i^\mu \int_0^\infty \mathrm{d}\tau \epsilon(\tau) \sum_{\mathbf{y}}{}' \frac{|L(\mathbf{y})|}{N} \times y^\mu \left[\frac{1}{|L(\mathbf{y})|} \sum_{j \in L(\mathbf{y})} \sigma_j(t-\tau) \right] \tag{12}$$

where the prime at the summation sign indicates that the sum goes over all $\mathbf{y}$ with $|L(\mathbf{y})| \neq 0$.

All neurons j that belong to the same sublattice as neuron i, i.e. $\boldsymbol{\xi}_j = \boldsymbol{\xi}_i = \mathbf{x}$, experience the same current

$$I_i(t) = I(\mathbf{x}, t) = 2J_0 \sum_{\mu=1}^{q} x^\mu \int_0^\infty \mathrm{d}\tau \epsilon(\tau)$$

$$\times \sum_{\mathbf{y}}{}' \frac{|L(\mathbf{y})|}{N} y^\mu \left[\frac{1}{|L(\mathbf{y})|} \sum_{j \in L(\mathbf{y})} \sigma_j(t-\tau) \right]. \tag{13}$$

We now keep the number of patterns fixed and take $N \to \infty$. The sum in square brackets, integrated over a short time Δt, approaches the probability to find a spike at a neuron of the sublattice $L(\mathbf{y})$ in the time interval between $(t-\tau)$ and $(t-\tau+\Delta t)$. Since there are extensively many neurons in $L(\mathbf{y})$ and since all $j \in L(\mathbf{y})$ are equivalent, receive the same input and are otherwise independent, it follows that the quantity in square brackets equals the activity $A(\mathbf{y}, t) = A[I(\mathbf{y}), t]$ defined in Sect. 2, (7). Hence we find as $N \to \infty$

$$I(\mathbf{x}, t) = 2J_0 \sum_{\mu=1}^{q} x^\mu \int_0^\infty \mathrm{d}\tau \epsilon(\tau) \sum_{\mathbf{y}} p(\mathbf{y}) y^\mu A(\mathbf{y}, t-\tau), \tag{14}$$

where $p(\mathbf{y}) = |L(\mathbf{y})|/N$ is the probability of a neuron to belong to the sublattice $L(\mathbf{y})$.

Later on we will be interested in solutions that 'retrieve' the stationary patterns the system has been trained on. For the moment we only assume that there is some kind of stationary solution. A *stationary solution* is defined by the condition that the mean activity is constant in each sublattice, i.e., $A(\mathbf{y}, t) \equiv A(\mathbf{y})$. In this case the time-dependent postsynaptic current $\epsilon(\tau)$ drops out (due to the normalization $\int_0^\infty \varepsilon(\tau)\, \mathrm{d}\tau = 1$) and the input is constant at each sublattice, $I(\mathbf{x}, t) \equiv I(\mathbf{x})$. Given a constant input, the mean activity equals the mean firing rate $A(\mathbf{y}) = f[I(\mathbf{y})]$, as we have pointed out at the end of Sect. 2. We thus arrive at the following implicit equation for the sublattice current

$$I(\mathbf{x}) = 2J_0 \sum_{\mu=1}^{q} x^\mu \sum_{\mathbf{y}} p(\mathbf{y}) y^\mu f[I(\mathbf{y})]. \tag{15}$$

This is the main result of the present section. The solution of (15) yields all stationary states of the network.

Let us step back for a moment and look at what has happened so far. The main step in the above derivation is from (13) to (14) where the limit $N \to \infty$ has been taken. Here we have made use of the law of large numbers which can be applied under the condition that all neurons of the ensemble are *independent and equivalent*. They are independent because the firing of a neuron depends only on the *total* postsynaptic current into the neuron whatever the presynaptic origin of the signals may be. They are equivalent because all neurons of a sublattice experience the same postsynaptic current, viz. (13). But are these arguments correct? Is it not true that the firing should also depend on the refractoriness of the neuron, i.e., on the time that has passed since the last firing occurred (see, e.g., Fig. 2)?

Let us therefore assume for the moment that the firing depends not only on the total synaptic current I, but also on the refractory time s defined as the time which has passed since the last firing event. In this case, it is convenient to define new sublattices $L(\mathbf{y}, s)$ of those neurons i which have learnt the same information *and* have fired their last spike at time $t_{k_i} = t - s$. Using discrete time steps of duration Δt, we can argue as before, and in the limit $\Delta t \to 0$ we find instead of (14)

$$I(\mathbf{x}, t) = 2J_0 \sum_{\mu=1}^{q} x^\mu \int_0^\infty \mathrm{d}\tau \varepsilon(\tau) \times \sum_{\mathbf{y}} \int_0^\infty \mathrm{d}s\, p(\mathbf{y}, s) y^\mu A(\mathbf{y}, s, t-\tau), \tag{16}$$

where $p(\mathbf{y}, s)$ is the differential probability of a neuron to belong to the sublattice $L(\mathbf{y}, s)$ and $A(\mathbf{y}, s, t)$ is mean activity of this sublattice at time t. To continue, we assume as before a stationary solution, so that the explicit time dependence may be dropped, and obtain

$$I(\mathbf{x}, t) \equiv I(\mathbf{x}) = 2J_0 \sum_{\mu=1}^{q} x^\mu \sum_{\mathbf{y}} \int_0^\infty \mathrm{d}s\, p(\mathbf{y}, s) A(\mathbf{y}, s). \tag{17}$$

Let us try to understand the meaning of the integral $\int_0^\infty \mathrm{d}s\, p(\mathbf{y}, s) A(\mathbf{y}, s)$. According to its definition, $A(\mathbf{y}, s)\mathrm{d}s$ is the firing probability of neurons in sublattice $L(\mathbf{y}, s)$, i.e., neurons which are subject to a current $I(\mathbf{y})$ *and* have not fired during a time s. The quantity $p(\mathbf{y}, s)$, on the other hand, is the probability of belonging to the sublattice $L(\mathbf{y}, s)$ with refractory time s and is therefore proportional to the survival function $P_I(s)$ which gives the neuron's probability to survive a time s without spiking; cf. (1). We have

$$p(\mathbf{y}, s) = p(\mathbf{y}, 0) P_I(s) \tag{18}$$

where $I = I(\mathbf{y})$ is the current at sublattice $L(\mathbf{y})$. If we

recall the notion of the interval distribution (Sect. 2), we see that $D_{I(\mathbf{y})}(s) = P_{I(\mathbf{y})}(s)A(\mathbf{y}, s)$. We therefore arrive at

$$\int_0^\infty \mathrm{d}s\, p(\mathbf{y}, s)A(\mathbf{y}, s) = p(\mathbf{y}, 0) \int_0^\infty D_{I(\mathbf{y})}(s)\, \mathrm{d}s = p(\mathbf{y}, 0) = p(\mathbf{y})f[I(y)]\,. \tag{19}$$

The last step follows from (3), (4), using (18) and the normalisation condition $\int_0^\infty p(\mathbf{y}, s)\, \mathrm{d}s = p(\mathbf{y})$.

Thus the more complicated model where the firing probability depends explicitly on the refractory time s also leads to the result (15). That is, the mean firing rate as defined by (3) and (4) is the only quantity that matters. The exact shape of the interval distribution $D_I(s)$ – see Fig. 2 – drops out since only the integral $[\int_0^\infty sD_I(s)\, \mathrm{d}s]^{-1}$ is contained in (17) and (19). Hence the type of noise that leads to a broadening of $D_I(s)$ is irrelevant.

As we have mentioned before, the stationary states of the network are given as the solutions of (15). As a measure of the quality of the solutions regarding retrieval of a pattern μ we define the *overlap* m^μ

$$m^\mu = 2 \sum_{\mathbf{x}} p(\mathbf{x}) x^\mu A(\mathbf{x})\,. \tag{20}$$

If we assume that the retrieval solution has a non-vanishing overlap with one pattern only, i.e., $m^\mu = m\delta^{\mu\nu}$, then we find from (15) the self-consistent equations for $\mu \neq \nu$

$$\begin{aligned} m^\mu &= 2 \sum_{\mathbf{x}} x^\mu p(\mathbf{x}) f\left(J_0 \sum_{\mu=1}^{q} x^\mu m^\mu \right) \\ &= \tfrac{1}{2}[f(J_0 m) + f(-J_0 m) - f(J_0 m) - f(-J_0 m)] \\ &= 0\,, \end{aligned} \tag{21}$$

while for $\mu = \nu$

$$\begin{aligned} m^\nu = m &= 2 \sum_{\mathbf{x}} x^\nu p(\mathbf{x}) f\left(J_0 \sum_{\mu=1}^{q} x^\mu m^\mu \right) \\ &= f(J_0 m) - f(-J_0 m)\,. \end{aligned} \tag{22}$$

If we plot

$$g(I) := f(I) - f(-I) \tag{23}$$

as a function of I we find the stationary overlap by a straightforward graphical solution. Since the current is given by $I = J_0 m$, the intersection of a straight line of slope $1/J_0$ with the function $g(I)$ yields m.

4 Qualitative solution for various model neurons

What are the conditions for the existence of retrieval solutions? Figure 5 gives some examples of typical behaviour for various types of model neuron.

The majority of the more realistic models of neuronal spiking is based on the classic work of Hodgkin and Huxley (1952) who have summarized their extensive experimental studies on the giant axon of the squid in four differential equations. The first describes the conservation of electric charge on a piece of membrane of capacitance C under the influence of a current and a voltage V

$$C \frac{\mathrm{d}V}{\mathrm{d}t} = I_i = I \tag{24}$$

where I is the external driving current and I_i is the sum of the ionic currents through the cell membrane

$$I_i = -g_{Na} m^3 h (V - V_{Na}) - g_K n^4 (V - V_K) - g_L (V - V_L)\,. \tag{25}$$

The constants V_{Na}, V_K, and V_L are the equilibrium potentials of the three components sodium, potassium, and 'leakage', the g's are parameters of the respective ion-conductances which depend on the variables m, n, and h determined by the differential equations

$$\begin{aligned} \frac{\mathrm{d}m}{\mathrm{d}t} &= \alpha_m(V)(1-m) - \beta_m(V)m \\ \frac{\mathrm{d}n}{\mathrm{d}t} &= \alpha_n(V)(1-n) - \beta_n(V)n \\ \frac{\mathrm{d}h}{\mathrm{d}t} &= \alpha_h(V)(1-h) - \beta_h(V)h\,. \end{aligned} \tag{26}$$

The α and β are given functions of V that have been adjusted empirically to fit the data of the giant axon.

In realistic models of a neuron which include the dendrites and the soma analogous equations for additional ion currents can be added, in particular a calcium current and a calcium dependent slow potassium current $k_{[Ca]}$ (Koch and Segev 1989; Ekeberg et al. 1991; Traub et al. 1991). The equilibrium potentials and the α- and β-functions can then be adjusted so as to fit the data of various types of neuron.

For the sake of simplicity, however, we have restricted our numerical work to the set (24)–(26) and we have used the original parameters of Hodgkin and Huxley. Solving (24)–(26) for different driving currents I we find a threshold θ above which repetitive firing occurs. The rate of firing depending on the input current, i.e., the gain function of the neuron, is shown in Fig. 5 (top left, solid line). This gain function which shows a sharp step at threshold is typical for the squid axon. A more refined 'realistic' model which includes the slow $K_{[Ca]}$-current has a continuous transition at threshold and a gain function which is roughly linear over a wide range of frequencies (Ekeberg et al. 1991).

Realistic models based on the Hodgkin–Huxley equations describe a wide range of phenomena found in experiments on diverse neurons quite accurately. Because of the complexity of (24)–(26) and analogous sets of equations, which have many free variables and tens of parameters that must be fitted, various simpler models have been proposed which capture the key features of neuronal spiking. One approach aims at a systematic reduction of (24)–(26) to two variables, so as to allow a phase plane analysis of the stationary and oscillatory states of the system. This has been done by FitzHugh (1961) and Nagumo et al. (1962) and more recently by Abbott and Kepler (1991). The gain function of these nonlinear oscillators is similar to that of the Hodgkin–Huxley model; in particular, there is a discontinuity at threshold.

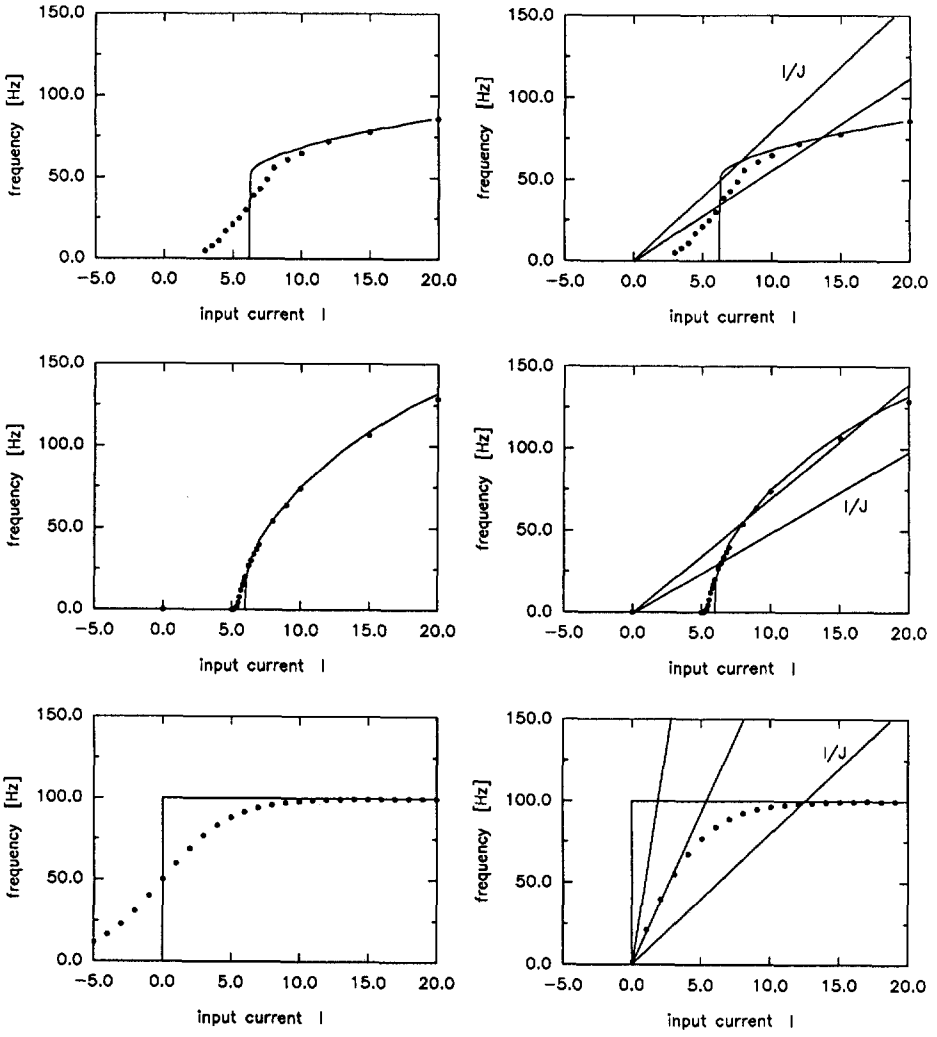

Fig. 5. *Gain function of various model neurons. Top* – Hodgkin–Huxley model, all parameters are taken from the original paper of Hodgkin and Huxley. Middle – Integrate-and-fire model with absolute refractory period $t_r = 4$ ms, integration time $\tau = 10$ ms and threshold current $I_\theta = 6\,\mu A$. Bottom – Two-state neuron without refractory period. The *solid lines* in the left column give the gain function $f(I)$ in the noiseless case, *dotted lines* show the mean firing frequency at a finite noise level (block-shaped amplitude distribution); see text for details. In the three plots of the right column, the function $g(I) = [f(I) - f(-I)]$ is plotted. The intersection of $g(I)$ with a *straight line* of slope $1/J_0$ yields the stationary overlap during the retrieval of a pattern

A different approach takes the neuron as a leaky integrator which is reset, if firing occurs. This leads to the class of 'integrate-and-fire' models which have been studied extensively by Stein (1967). The membrane voltage evolves after each spike according to

$$V(t) = IR[1 - \exp(-t/RC)], \tag{27}$$

where R and C are the membrane resistance and capacitance respectively and I is the driving current. If the threshold voltage V_θ is reached, the neuron fires and the voltage is reset to zero. If we also include an absolute refractory period t_r during which the membrane is insensitive to external stimuli, we find a firing rate

$$f(I) = \left[t_r + \tau \log\left(1 - \frac{I_\theta}{I}\right)\right]^{-1}, \tag{28}$$

where $\tau = RC$ and $I_\theta = V_\theta/R$. This is the gain function that is plotted in Fig. 5 (middle left, solid line).

The most dramatic simplification is a reduction of neuronal spiking behaviour to two states only: firing ($S = +1$) or not firing ($S = -1$). This model is known as two-state or formal neuron and has been used in theoretical considerations (McCulloch and Pitts 1943) as well as in the study of neural networks (Little 1974; Hopfield 1982). The gain function is that of a simple threshold device with a step at $I = 0$. Noise can be added, if we assume that spiking occurs stochastically with conditional probability

$$\text{Prob}\{S = +1|I\} = \tfrac{1}{2}\{1 + \tanh[\beta(I - \theta)]\}\,. \tag{29}$$

This is the so-called Glauber dynamics. Averaging over time we find the mean firing rate

$$f(I) = \tfrac{1}{2}\{1 + \tanh[\beta(I - \theta)]\}\,, \tag{30}$$

which is also plotted in Fig. 5 (bottom left, dotted).

The gain functions of the models presented above should be understood as typical examples. The models have been selected to show a variety of qualitatively different results. Other models yield gain functions that are similar to one of the three types or lie somewhere in between. Experimental results show that real neurons exhibit a similar variety of gain functions (Hodgkin 1948; Llinas and Sugimori 1980; Jahnsen and Llinas 1984; Connors and Gutnick 1990).

In the right column of Fig. 5 we have plotted the function

$$g(I) := f(I) - f(-I)\,. \tag{31}$$

According to (22) and (23) the intersection of this function with a straight line of slope $1/J_0$ yields the stationary overlaps with one of the patterns. The solution $g(I_0) = I_0/J_0$ is unstable, if $(\partial g/\partial I)(I_0) > J_0^{-1}$. The other solutions may be unstable with respect to oscillatory solutions, see Sect. 5 for details. If the feedback parameter J_0 is strong enough, all models have a retrieval solution with finite stationary overlap. If we start to reduce the synaptic feedback J_0, the value of the overlap gets smaller and smaller. If one reduces the feedback even further the overlap either jumps to zero discontinually at a critical value J_c (Fig. 5, top and middle, right column), or it decays continually as in the Hopfield model (Fig. 5, bottom right).

It has sometimes been argued that an obvious flaw of associative network models is the high firing rate in the stationary states of the system as compared to the low spiking rates found in experiments (Amit and Treves 1989; Amit and Tsodyks 1991). From the graphical solutions in Fig. 5, however, we immediately see that for a gain function of the Hopfield type (bottom), solutions with arbitrary low firing frequencies are possible. For integrate-and-fire neurons there is a lower bound, but it is low enough to allow reasonably low firing rates (Amit and Tsodyks 1991). This is also true for other realistic models that show a rather linear and flat (steady state) gain function (e.g., Ekeberg 1991). It is only for gain functions with a sharp threshold as it is seen in the Hodgkin–Huxley model (Fig. 5, top) and similar models that solutions with low firing rates are not possible, at least not in the noise-free case. So what is the effect of noise on the system?

In the Hopfield model with formal two-state neurons and Glauber dynamics, the slope of the gain function is directly correlated with the magnitude of the noise in the stochastic dynamics. In the more refined models like the integrate-and-fire model or the variable threshold model, the slope is set by internal parameters of the models, i.e., the time constant of the membrane and the decay time of the refractory period. If we add noise to the system, the gain function changes slightly and the threshold is rather blurred (Fig. 5, top and middle, dotted), but the main difference is in the interval distribution which can take different forms depending on the kind of noise we choose (Gaussian, Poisson and other). The derivation of Eq. (15) in Sect. 3, however, shows that the form and the variance of the interval distribution do not matter. Noise matters only *in as far as it decreases or increases the mean firing rate.*

An explicit way to introduce noise into the above models is to take the input current as a parameter that varies stochastically around the nominal current I_0, i.e.

$$I = I_0 + \delta I \tag{32}$$

where δI is chosen from a probability distribution $P(\delta I)$. Besides the distribution of amplitudes $P(\delta I)$ (which may be a Gaussian) the frequency distribution of the noise is also an important parameter that determines the effective noise level. As an example of a noisy dynamics we have simulated the Hodgkin–Huxley and the 'integrate-and-fire' model neurons with an input current that changed abruptly every *ms*. The noise amplitude δI has been chosen from a block-shaped distribution $P(\delta I) = 1/(2\Delta I)$ for $|\delta I| \leqslant \Delta I = 0.3 I_\theta$ and 0 otherwise. The mean firing rate has been determined according to the procedure outlined in Sect. 2. We then calculated the function $g(I)$ that is plotted in the right column of Fig. 5 (dotted curves). As before, we find the overlap value from a graphical solution of (22) (Fig. 5, intersection of the dotted curves with the straight line). Thus, the qualitative behaviour of the system remains unchanged.

The gain functions of Fig. 5 are based on some models that are commonly used in neurobiology and neural network theory. We have taken them only as convenient examples to explain the various types of solution, but the arguments of Sect. 3 do not rely on any model at all. Instead of one of the model gain functions it is also possible to take an experimentally measured gain function of neurons in the association area of the cortex and some typical values of the synaptic feedback. A graphical solution analogous to Fig. 5 shows whether the retrieval of a stationary patterns is possible.

5 Discussion and generalization

5.1 Patterns of arbitrary activity

There are two obvious objections to our network analysis. They concern the symmetry of the connections and the high spatial activity in the retrieval states. Both objections can be overcome by a generalization of the model network to patterns of arbitrary activity a, i.e. $\xi_i^\mu = \pm 1$ with probability $(1 \pm a)/2$. To code the patterns we change the synaptic connection matrix and allow it to assume the asymmetric form (van Hemmen et al. 1990)

$$J_{ij} = \frac{2J_0}{N(1-a^2)} \sum_{\mu=1}^{q} \xi_i^\mu (\xi_j^\mu - a) . \tag{33}$$

Despite the asymmetry of the connections, an exact solution is still possible. With the new definition of the overlap

$$\begin{aligned} m^\mu &= \frac{2}{N(1-a^2)} \sum_{j=1}^{N} (\xi_j^\mu - a) S_j \\ &= \frac{2}{1-a^2} \sum_{\mathbf{x}} p(\mathbf{x})(x^\mu - a) A(\mathbf{x}) \end{aligned} \tag{34}$$

we find instead of (15)

$$I(\mathbf{x}) = (2J_0/1-a^2) \sum_{\mu=1}^{q} x^\mu \sum_{\mathbf{y}} p(\mathbf{y})(y^\mu - a) f[I(\mathbf{y})] , \tag{35}$$

whereas (22) remains unchanged. Thus the retrieval solution does not change either.

5.2 Non-identical neurons and synapses

As we have pointed out in Sect 3.1, the model has been defined under the assumption that all neurons and synapses have identical characteristics. In neurobiology,

however, several distinct types of neuron, e.g., pyramidal and stellate cells, and also several types of synapse, characterized by some neurotransmitter substance and receptor mechanism, are known. Furthermore, the neurons of one type may have different size and the synapses may have different positions on the dendritic tree. To take these effects into account, we group the neurons into several classes $\lambda = 1, \ldots l$ and the synapses into distinct types $\kappa = 1, \ldots k$. The elements of each class are assumed to show identical characteristics. It is impossible to give an analysis of the most general case of synaptic connections between the various neurons. But as long as each postsynaptic neuron of class λ has the same distribution of synapses along the dendrites, an extension of the above considerations and results is straightforward. In this case, the postsynaptic response to a single presynaptic spike arriving via a synapse of type κ can be described by a generalization of (9),

$$J_0(\kappa, \lambda)\varepsilon(\kappa, \tau) \tag{36}$$

where $\varepsilon(\kappa, \tau)$ describes the time dependence of the response of a synapse of type κ and $J_0(\kappa, \lambda)$ is the effective strength due to the dendritic distance between the synapse and the soma of a neuron of type λ. We define sublattices $L(\mathbf{x}, \lambda)$ of neurons i that belong to class λ and have stored the data $\xi_i = \mathbf{x}$. All neurons in $L(\mathbf{x}, \lambda)$ experience the same current

$$I(\mathbf{x}, \lambda, t) = 2 \sum_{\mu=1}^{q} x^{\mu} \sum_{\kappa=1}^{k} J_0(\kappa, \lambda) \int_0^{\infty} \mathrm{d}\tau \varepsilon(\kappa, \tau) \times \sum_{\mathbf{y}, \lambda'} p(\mathbf{y}, \lambda') y^{\mu} A(\mathbf{y}, \lambda', t - \tau) . \tag{37}$$

If we solve the system of equations in a way analogous to (13)–(15), then we find instead of (22) for the overlap

$$m = \sum_{\lambda} p(\lambda)[f_{\lambda}(I_{\lambda}) - f_{\lambda}(-I_{\lambda})] . \tag{38}$$

Here $p(\lambda)$ is the portion of neurons of class λ in the net, f_{λ} the gain function of these neurons, and I_{λ} the driving current $I_{\lambda} = \sum_{\kappa=1}^{k} J_0(\kappa, \lambda) m$. Thus the result is basically the same as (22), except that the contributions of the various neurons and synapses have to be weighted with the prefactor $p(\lambda)$ and $J_0(\kappa, \lambda)$, respectively.

5.3 Mean-field conditions and how to get rid of them

The main result of the preceding sections is that it is possible to construct the stationary solutions of a network in terms of overlaps in a manner which is *independent of any specific model of the neurons*. The only object we use are spike trains, may they be produced by real neurons or some formal neuron. Using the spike trains we calculate the mean firing rate defined as the inverse of the mean interval length. The only parameters which we need to determine the existence and quality of the retrieval solutions are the gain function of the neurons, i.e., the mean firing rate in dependence upon the input current, and the synaptic efficacy factor J_0 as a measure of the feedback of the network onto the neurons. From our considerations it follows that second and higher-order moments of the spiking statistics which determine the shape and the variance of the inverval distribution do not matter. Thus, the results are *independent of the nature of the noise*, be it Poisson, Gaussian or other. Only the mean of the interval distribution counts.

In fact, our results are identical to a naive mean-field solution of the network. If we assume two populations of neurons, those that should be 'on' for pattern ν and those that should be 'off', both will see the same input current, but with different signs. Performing the 'sloppy' *mean-field* argument (Wilson and Cowan 1972) we immediately get (22).

The present argument, however, shows that (22) is exact and not based on any additional mean-field assumption. Such a result, however, should be no surprise, since it is known that for a fully connected system with a finite number of different connection weights (i.e., q finite) mean-field theory becomes exact in the bulk limit $N \to \infty$. This is due to the fact that in a fully connected network each neuron receives inputs from extensively many others. If these inputs are independent, the law of large numbers states that only the mean of the distribution matters. With the assumption of stationarity we could neglect correlations of the synaptic contributions in time, while the sublattice argument showed that there are no correlations in space. Thus the conditions for the application of the law of large numbers and the exact validity of mean-field-results are the *full connectivity* and the *assumption of stationarity* plus the restriction to a *finite number of patterns*. What happens if we loosen one – or more – of these conditions?

i) Finite number of patterns. The crucial step of the analysis is from (13) to (14) where we have to use the fact that each sublattice contains in the limit $N \to \infty$ an extensive number of neurons. For N large, but finite, we have to restrict the number of patterns so as to assure that the number of sublattices 2^q is much smaller than N, or $q \ll \log N$. If, however, the number of patterns q is of the same order than the number of neurons, i.e., $q = \alpha N$ with finite α, then the coupling matrix (11) introduces an additional source of noise into the system. In analogy to earlier results (Amit et al. 1987, Kühn et al. 1991) we expect that (22) must be changed so as to include the extra noise term. A careful interpretation of the results of Kühn et al. for a network of graded-response neurons shows that the gain function alone is not enough to determine the state of the system and that the variance of spiking frequencies at fixed input is also important. The qualitative nature of the phase diagram, however, remains the same, i.e., in the limiting cases of high and low loading, models with identical gain function yield the same result.

ii) Full connectivity. Let us now consider the condition of full connectivity. If we remove synapses stochastically, we achieve sparse, but long-ranged connections. As long as each neuron has connections to a large

number of neurons in arbitrary distance, hardly anything changes and the same type of analysis as in Sect. 3 can be carried out. In the cortex, it is believed that every neuron is connected to thousands of other neurons. There are many local connections, but there is also a large portion of axons that extend over long distances. A model with full connectivity may thus be an excellent approximation to the high connectivity found in some areas of the cortex.

In the case of predominantly local connections, however, the situation changes and a completely different mathematical description becomes necessary. In this case, correlations between neighbouring neurons can lead to local clusters that behave in a way different from the bulk of the neurons. Mean-field solutions are then no longer valid. In a biological context, local connections are of extreme importance for the understanding of columnar structures in the visual cortex and the formation of neural maps (Hubel and Weisel 1977). Nevertheless we have good arguments to speculate that in the association areas of the cortex uniformly ranged connections are a fair approximation. In those areas our approach may be applied.

iii) Stationarity. The third important condition in the argument of Sect. 3 has been the assumption of a stationary solution of the collective dynamics of the system. This assumption has been possible since we were interested in the retrieval of stationary patterns only. From a biological point of view, time-dependent solutions are certainly important; we only have to think of the fast reaction of the nervous system to changing external input conditions as for example during the flight of a fly or a bat. It is also known that single neurons as well as neural systems produce a variety of adaptation effects in a changing environment. Furthermore, there is evidence that the brain reacts with *coherent oscillations* of the spiking activity even during fairly constant input conditions as for example in the odor system of the rat (Freeman 1975) or in the visual cortex of the cat (Eckhorn et al. 1988; Gray and Singer 1989). With respect to the possibility of oscillations in a neural net we have to consider the conditions under which the assumption of a stationary solution is no longer justified.

The theoretical conditions for the stability of the stationary solutions are a long postsynaptic integration time as compared to the inverse of the mean firing rate, or, if the integration time is short, a timing of delay and response that leads to a negative feedback. Even if a stationary solution is locally stable it might be of minor importance, if its domain of attraction is small compared to oscillatory solutions. It has been shown that in a network with realistic neurons *both* collective oscillations *and* stationary solutions can occur. It depends on the exact timing of the synaptic transmission delays and the postsynaptic response whether the stationary solution is stable or the system is driven into the oscillatory state (Ritz 1991; Gerstner and van Hemmen 1992). Thus the stability of the stationary solution is achieved by a complex combination of delay time and postsynaptic response on the one hand, refractory period, mean firing rate, and spiking noise on the other hand. For this reason, time-dependent effects require a specific model of the neurons that takes all these elements of neuronal spiking into account. For time-dependent effects, the mean firing rate can no longer take a dominant role, since the correlation among the firing of different neurons becomes important. Thus, any approach that uses mean firing rate assumptions in the context of collective oscillations and other time-dependent problems seems questionable.

6 Conclusions

Using the concepts of statistical spike train analysis, we have shown that there is a controlled way to go from realistic neuronal spike trains to a description by gain functions (mean firing rate versus input current). It turns out that in a recurrent network with high connectivity the mean firing rate is the only relevant variable of neuronal spiking in a stationary state. This conclusion is independent of any specific model of a neuron. Hence it is possible to predict the existence and quality of retrieval solutions, provided the gain function and the efficacy of the synaptic transmission are known. Thus, as long as we are interested in stationary solutions only, there is no need to include more and more refined models of a neuron into associative neural network models – the gain function is the only object that matters.

It is however, important to realize that these results are exact only for a *stationary state of a highly connected* network. If we are interested in locally connected nets or in time-dependent solutions, then the mean firing rate is no longer the only relevant parameter. In this case, detailed models of a neuron and its firing behaviour are required. It is necessary to keep this in mind, if a description of collective oscillations in the cortex, adaptation phenomena, or other time dependent problems of neuronal spiking like is attempted.

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