Biol. Cybern. 68, 363–374 (1993)

Biological Cybernetics

A biologically motivated and analytically soluble model of collective oscillations in the cortex

I. Theory of weak locking

Wulfram Gerstner, Raphael Ritz, J. Leo van Hemmen

Institut für Physik der Technischen Universität München, W-8046 Garching bei München, Germany

Received: 15 June 1992/Accepted: 31 August 1992

Abstract. A model of an associative network of *spiking* neurons with stationary states, globally locked oscillations, and weakly locked oscillatory states is presented and analyzed. The network is close to biology in the following sense. First, the neurons spike and our model includes an absolute refractory period after each spike. Second, we consider a distribution of axonal delay times. Finally, we describe synaptic signal transmission by excitatory and inhibitory potentials (EPSP and IPSP) with a realistic shape, that is, through a response kernel. During retrieval of a pattern, all active neurons exhibit periodic spike bursts which may or may not be synchronized ('locked') into a coherent oscillation. We derive an analytical condition of locking and calculate the period of collective activity during oscillatory retrieval. In a stationary retrieval state, the overlap assumes a constant value proportional to the mean firing rate of the neurons. It is argued that in a biological network an intermediate scenario of '*weak locking*' is most likely.

1 Introduction

Whereas associative retrieval of stationary patterns has attracted a lot of attention during the last ten years (Hopfield 1982, 1984; Amit et al. 1985; 1987; for a review see Amit 1989 and Domany et al. 1991), emphasis has now shifted considerably to the problem of collective oscillations after evidence of coherent activity has been found in the cortex (Eckhorn et al. 1988; Gray and Singer 1989; Gray et al. 1989; Engel et al. 1991). This shift of interest is mainly due to the fact that synchronicity of firing might be used by the brain as a code to link features that belong to the same pattern and to separate them from the rest (von der Malsburg and Schneider 1986; Eckhorn et al. 1988). Coherence could thus solve two old questions of information processing in the brain which are known as the problems of feature-linking and pattern segmentation.

In most network models of collective oscillations, the applications to image processing have been in the center of interest (Wang et al. 1990; Schillen and König 1991; König and Schillen 1991; Sompolinsky et al. 1991; Schuster and Wagner 1990b; Horn et al. 1991) and the biology of the experimental systems has been incorporated at a rather crude phenomenological level. In particular, the basic unit in most model networks is a set of ad hoc differential equations for amplitudes or phases of the excitation of an *ensemble* of neurons, i.e., one column (Wilson and Cowan 1972; Baird 1986; Wang et al. 1990; König and Schillen 1991; Schuster and Wagner 1990a). This is to be contrasted with traditional associative networks where the basic unit is taken to describe the mean firing rate of a *single* neuron (Hopfield 1982, 1984). It is thus an interesting question how both approaches – the single neuron Ansatz used in associative networks and the ensemble Ansatz used in oscillation networks – can be combined.

To unify the two approaches we return to biology and include some details of neuronal signal transmission which are usually neglected. We focus on a *single spike* as the important phenomenon and describe its generation at the soma, transmission along the axon, and transfer across the synapse by plausible model assumptions. Within this model, we derive exact conditions under which stationary or oscillatory activity occurs.

In the present paper, we concentrate on general properties of a fully connected network during retrieval of a single pattern. To give a specific example, the system can be considered as a model of a single column in the visual cortex, but such an interpretation is somewhat arbitrary. Our aim is not a detailed description of a specific system but rather an explanation of general phenomena from a unifying point of view. The generalization of our model to a columnar structure with distance-dependent connectivity and the applications of our results to completion and separation of simultaneously presented patterns is discussed in a companion paper (Ritz et al. 1993). A short account (van Hemmen

Correspondence to: J. L. van Hemmen

et al. 1992) of our basic results has been given during the conference "Complex dynamics in neural networks" (Vietri, 17–21 June, 1991).

There have been, of course, several other models that focus on spikes and biological realism. Buhmann and Schulten (1986) and Horn and Usher (1989) discuss associative networks of spiking units similar to our model neuron but concentrate on stationary retrieval. Results on collective oscillations in large networks with spiking neurons are reported by Buhmann (1989), and by Hansel and Sompolinsky (1992), Kurrer et al. (1990), and Horn et al. (1991). On the other hand, Bush and Douglas (1991) consider a cortical 'network' of ten bursting pyramidal neurons and one basket cell and show in simulations with a set of realistic neuronal parameters and connections that a burst synchronization is possible. A similar, but larger network of compartmental model neurons with local connectivity which is intended to describe an area of the visual cortex is considered by Wilson and Bower (1990). A rather complex but interesting model that includes columnar organization of a large number of neurons has been studied by Sporns et al. (1989, 1991); in particular, its application to the problem of figure-ground segregation is discussed in detail.

In all cases, however, the results are mainly based on computer simulations of the respective model. In contrast to this, our model of a 'biological' network is simple enough to allow analytical solutions which are exact in the limit of a large number of noiseless neurons and realistic enough to provide insight in the behavior of biological systems. Furthermore, we are able to derive conditions for the existence and stability of a collective oscillation and to determine its period. The analytical results can then be compared to computer simulations of a finite, but large system at a finite noise level. The present network (Ritz 1991) which is organized in two layers, i.e., inhibitory and 'Hebbian' neurons, is a generalization of an earlier homogeneous model (Gerstner and van Hemmen 1992a).

In Sect. 2, we define the model network based on a simplified description of neurobiological signal transmission. In Sect. 3, we present an analytical solution of the macroscopic network states in terms of the dynamic evolution of so-called overlaps. We calculate the retrieval overlap in a stationary state, the oscillation period of an oscillatory state, and derive a condition of 'locking' into a collective oscillation. If the locking condition is not fulfilled, weak locking may result. Section 4 is devoted to computer simulations of a network consisting of two layers of 4000 neurons. The simulations visualize typical network states in three different scenarios. Classification of the network states yields a phase diagram in parameter space. We close with an extensive discussion of the capabilities and limitations of our model.

2 Definition of the model network

2.1 Basic model assumptions

Before a network model can be defined, a couple of basic decisions concerning the desired level of description and the treatment of biological details have to be made. Some of the most important and intriguing problems of theoretical neurobiology concern the coding procedure through which a biological system maps data from the outside world onto the network states. This includes the question whether the information concerning the environment is contained in the mean firing rates of the neurons or rather in complex temporal spiking patterns. Or is it even the exact voltage trace and shape of a single spike which is important? Furthermore, are there ensembles of neurons (e.g., columns) which are involved in the same information processing tasks, or should we take the spiking of each single neuron into account? It is our opinion that a model network should impose as few assumptions as possible concerning the above questions. We therefore take a single spiking neuron as the basic unit of our network. Averaging over time (to get the mean firing rate) or over space (to define the ensemble average or 'global' activity) can then, if appropriate, be done at a later stage during the theoretical analysis of the network – but not beforehand. Averaging should not be an underlying assumption of the model definitions either.

It is, of course, impossible to include all known details of a specific neural system, e.g., the visual cortex of the cat, into a general artificial network. Our approach neglects all details of processes at the level of neurotransmitters and ion channels as well as the branching structure of axons and dendritic trees. A microscopic Ansatz including these effects would yield the exact shape and velocity of spikes moving along the axon to the synapses (Hodgkin and Huxley 1952; Ekeberg et al. 1991) and of the postsynaptic potentials spreading over the dendritic tree (Rall 1964; Jack et al. 1975; Abbott 1991). It is, however, our assumption that the exact shape of the spikes and dendritic signals conveys no information in addition to that of the spiking *event* itself which is already contained in much simpler models. It is thus convenient to adopt a more phenomenological approach and consider *formal* spikes that are generated by a threshold process and transmitted along the axon to the synapses. The signal arrives there after some delay Δ^{ax} and evokes an excitatory or inhibitory postsynaptic potential (EPSP or IPSP). The EPSP and IPSP which change on a time scale much longer than a single spike are modelled with a realistic time course. Finally, the postsynaptic contributions of all active neurons are added in a linear fashion and compared to the firing threshold so as to determine the firing probability during the next time step.

As to the connection topology, it is known that the pyramidal neurons make both long-ranged and short-ranged connections whereas inhibition is primarily local. Here we take the point of view of a theoretician and model the extreme and most transparent case, i.e., full connectivity between pyramidal neurons and local inhibition by inhibitory partner neurons. An exact definition of the above ideas is given in the next subsection.

2.2 Elements of the network: Neurons and synapses

A phenomenological model of a single neuron i can be built on two measurable parameters, both amenable to experiment, viz., the firing threshold θ and the refractory time τ_{ref}. Since we do not want to describe the shape of a single spike, we model spikes by a formal variable $S_i(t) = \{0, 1\}$ and take a typical spike width of 1 ms as the basic time step of our model. The spiking dynamics of neuron i is defined by the probability of firing during one time step, given a membrane potential h_i,

$$P_F[S_i(t+1) = 1 \mid h_i(t)] = (1/2)\{1 + \tanh[\beta(h_i(t) - \theta)]\}, \quad (1)$$

where β is a parameter that takes care of the internal noise of a neuron. The membrane potential $h_i(t)$ consists of three components

$$h_i(t) = h_i^{\text{syn}}(t) + h_i^{\text{ext}}(t) + h_i^{\text{ref}}(t), \quad (2)$$

where $h_i^{\text{syn}}(t)$ is the sum of the synaptic inputs from all other neurons in the network (see below), $h_i^{\text{ext}}(t)$ is some external input, and $h_i^{\text{ref}}(t)$ is a formal contribution to describe the refractoriness of the neuron. If we take

$$h_i^{\text{ref}}(t) = \begin{cases} -R & \text{for } t_F \leqslant t \leqslant t_F + \tau_{\text{ref}} \\ 0 & \text{otherwise} \end{cases} \quad (3)$$

and $R \gg 1$, then firing is prevented during a time τ_{ref} after emission of a spike at $t = t_F$. In all simulations we take the absolute refractory time τ_{ref} equal to 1 ms, a value that is commonly reported (see any textbook on neurobiology, e.g., Kuffler et al. 1984). This choice of τ_{ref} limits the maximum spiking frequency to 500 Hz which is a reasonable value during short activity bursts. Relative refractoriness and adaptation are excluded in our present model, but the Ansatz (3) for $h_i^{\text{ref}}(t)$ can be generalized easily so as to include both effects (Gerstner and van Hemmen 1992a).

A single neuron, separated from the rest of the network $[h_i^{\text{syn}}(t) = 0]$ and driven by a constant input $h_i^{\text{ext}}(t) = \gamma$, has the input/output characteristics shown in Fig. 1a. The graph exhibits the typical sigmoidal dependence of the mean firing rate $f = n_F/T$ (number of spikes during a measurement interval T) upon the input γ. With a simple argument based on the spiking probability per time step (1) and the duration of absolute refractoriness (3) it can be shown (Gerstner and van Hemmen 1992a) that $f(\gamma) = P_F(\gamma)/[1 + \tau_{\text{ref}} P_F(\gamma)]$ where $P_F(\gamma)$ is the firing probability for the potential $h_i = \gamma$. This is the function that is plotted in Fig. 1a. The set of parameters ($\tau_{\text{ref}} = 1$ ms, $\theta = 0.12$, $\beta = 15$) is the same as in the simulations of a network of neurons in Sect. 4. The maximum spiking rate of a single neuron equipped with these parameters is 500 Hz. If, however, the model neuron is combined with an inhibitory partner neuron – as in the network introduced below – then the mean firing rate at high input levels is reduced to less than 150 Hz (Fig. 1b). Let us therefore turn now to the structure of the network.

The neurons are connected in a scheme of N elements (Fig. 2), each element consisting of a 'Hebbian' neuron (top layer) and an inhibitory partner neuron (bottom layer). The 'Hebbian' neurons form a fully connected network which is able to store q patterns by adjusting the synaptic weights according to a 'Hebbian' learning rule (Hebb 1949; Herz et al. 1988). A pattern μ is defined as a set of random variables ξ_i^μ ($1 \leqslant \mu \leqslant q$) and ($1 \leqslant i \leqslant N$) which are distributed stochastically with mean activity a, i.e., $\xi_i^\mu = \pm 1$ with probability $(1 \pm a)/2$. A more realistic set of elementary patterns is to be considered by Ritz et al. (1993). Learning the patterns yields the synaptic efficacies J_{ij} of signal transmission from neuron j to neuron i (van Hemmen et al. 1990),

$$J_{ij} = \frac{2}{N(1-a^2)} \sum_{\mu=1}^{q} \xi_i^\mu (\xi_j^\mu - a). \quad (4)$$

The neurons in the network communicate via the exchange of spikes which are generated according to the threshold-firing process (1). If a neuron j in the Hebbian layer fires, the signal of the spike is transmitted to all neurons of the Hebbian layer as well as to the partner neuron in the inhibitory layer. Let us now analyze the effect on a neuron i in the Hebbian layer first (Fig. 2, inset top right).

A postsynaptic neuron i that is connected to neuron j through a synapse of efficacy J_{ij} will receive the signal

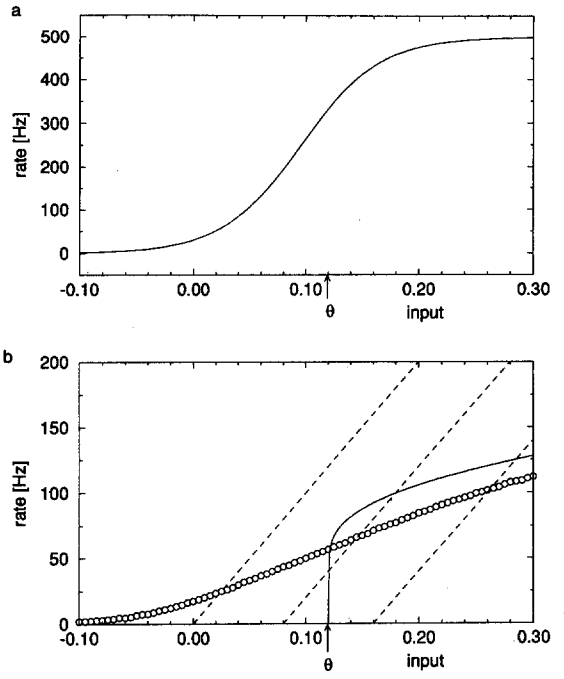

Fig. 1. a Gain function of a single neuron as predicted by the model. The mean firing rate (y-axis) displays a sigmoid dependence upon the applied input (x-axis, arbitrary units). All parameters (noise $\beta = 15$, threshold $\theta = 0.12$, refractory time $\tau_{\text{ref}} = 1$ ms) are the same as used later on during the simulations of a large network of model neurons; cf. Figs. 6–8. **b** Gain function of the very same neuron, after it has been connected to an inhibitory partner neuron (*circles*: $\beta = 15$; *solid line*: $\beta = \infty$). The mean firing rate of the neuron pair is much lower than that of a single neuron. Note the different scale in **a** and **b**. The intersection of the gain function with the dashed lines yields the stationary mean firing rate at different input levels, if the neuron pair is part of a large network; see Sect. 3.2 for details

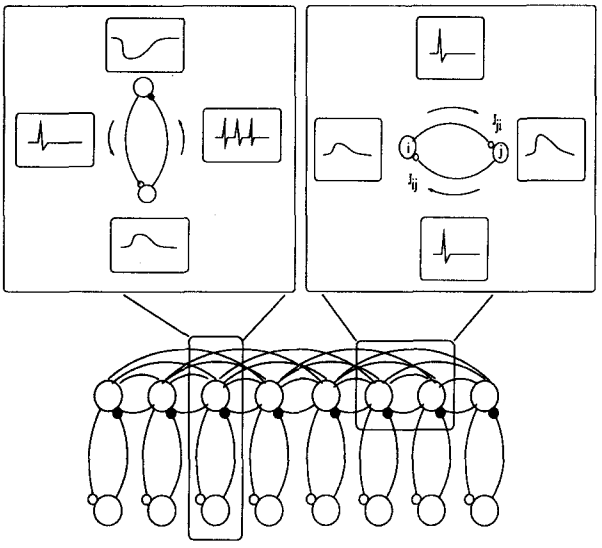

Fig. 2. Network structure. The network consists of two layers of 4000 neurons (only 8 are shown) which communicate via the exchange of spikes. The neurons in the top layer ('Hebbian' neurons) are fully connected by Hebbian synapses J_{ij} that store a fixed set of patterns. Each 'Hebbian' neuron is also connected to an inhibitory partner neuron (*bottom*). If one of the neurons fires, a spike is transmitted along the axon and, after some delay, evokes an excitatory (EPSP) or inhibitory (IPSP) postsynaptic potential at the receiving neuron (*inset top left* and *top right*)

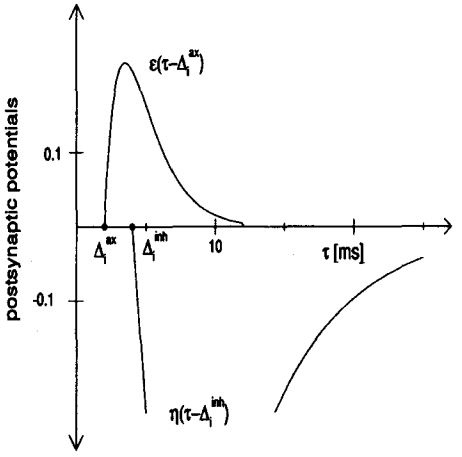

Fig. 3. Excitatory (EPSP) and inhibitory (IPSP) postsynaptic potential. If a 'Hebbian' neuron i fires at $\tau = 0$, a spike is transmitted to all other 'Hebbian' neurons to which it is connected. There it evokes – after some transmission delay Δ_i^{ax} – an EPSP. At the same time a signal is transmitted to the inhibitory partner neuron, which in turn sends a spike back so as to induce – after a delay Δ_i^{inh} – a strong IPSP; cf. Fig. 2. The time course of an EPSP is modeled by the function $\epsilon(t) = (t/\tau_\epsilon^2)\exp(-t/\tau_\epsilon)$ with $\tau_\epsilon = 2$ ms (*top*). Inhibition is described by a sharp rise and an exponential decay with a time constant $\tau_\eta = 6$ ms (*bottom*)

of the spiking of neuron j only after some axonal transmission delay Δ_i^{ax} which is taken to vary stochastically in a range of $\Delta_{min}^{ax} \leqslant \Delta_i^{ax} \leqslant \Delta_{max}^{ax}$. After this delay, an excitatory (or inhibitory) potential is induced across the membrane of the postsynaptic neuron. Its time-course is described by a response function $\epsilon(\tau)$. Its amplitude, however, is determined by the synaptic efficacy J_{ij} (4). The response function $\epsilon(\tau)$ of an excitatory synapse is simply the excitatory postsynaptic potential (EPSP) which has been determined experimentally (for a review see, e.g. McCormick 1990). We approximate this function by a discrete version of the α-function (Jack et al. 1975)

$$\epsilon(t) = (t/\tau_\epsilon^2)\exp(-t/\tau_\epsilon)\,, \tag{5}$$

which is shown in Fig. 3.

Summing the contributions of all neurons which send signals to neuron i yields the synaptic part of the membrane potential (2),

$$h_i^{syn}(t) = \sum_{j=1}^{N} J_{ij} \sum_{\tau=0}^{\infty} \epsilon(\tau) S_j(t - \tau - \Delta_i^{ax}) + h_i^{inh}(t)\,, \tag{6}$$

where $h_i^{inh}(t)$ is the contribution of the inhibitory partner neuron of neuron i.

The inhibitory contribution can be determined as follows (Fig. 2, top left). If neuron i starts firing at time t_F, a couple of spikes are transmitted to the inhibitory partner neuron where they evoke – after a delay δ_1 – an EPSP. This excites the inhibitory neuron to send a series of spikes back to neuron i where they generate – after another delay δ_2 – an inhibitory signal, i.e., an IPSP. The net effect is a strong inhibitory feedback to the active neuron i, $\eta(\tau)$, which sets in after a total delay of $\Delta_i^{inh} = \delta_1 + \delta_2$, climbs within 2 ms to a saturation value η_{max} and decays exponentially with a time constant $\tau_\eta = 6$ ms afterwards; see Fig. 3. The delay Δ_i^{inh} is assumed to vary for different neurons in a range of $\Delta_{min}^{inh} \leqslant \Delta_i^{inh} \leqslant \Delta_{max}^{inh}$ with equal probability. In view of the above considerations we obtain

$$h_i^{inh}(t) = \sum_{\tau=0}^{\tau_{max}} \eta(\tau) S_i(t - \tau - \Delta_i^{inh})\,. \tag{7}$$

To include saturation effects at strong inhibition levels, we take the upper bound τ_{max} in (7) as a flexible limit and stop the summation after the first non-vanishing summation term. That is, inhibitory potentials which arise after a bursting episode of neuron i do not add up linearly, but only the most recent contribution matters.

3 Macroscopic states of the network: theory

The network as defined in the preceding section combines the features of association networks with the properties of oscillation networks (see also Baird 1986 and Buhmann 1989). In particular, we find retrieval states that have a macroscopic overlap with a single pattern. Depending on the time structure of excitation and inhibition, the overlap is either stationary or oscillatory. In contrast to most oscillation models where a neuron – or a whole set of neurons – is modelled by a mere nonlinear oscillator with some limit cycle, our model of a spiking neuron allows to determine the period and stability of a collective oscillation in terms of biological parameters, notably, the shape of EPSP and IPSP. This can be done in spite of the fact that we have a *distribution* of delay times Δ_i^{ax} and Δ_i^{inh}.

The analysis of a large network ($N \to \infty$) in the low-noise limit ($\beta \to \infty$) is done in three steps. First we derive the dynamic equation for the evolution of the network states (Sect. 3.1). Then we calculate the overlap in a stationary retrieval state (Sect. 3.2) and the oscillation period in an oscillatory retrieval state (Sect. 3.3). Finally, we present a condition for locking of neuron activity into a collective oscillation (Sect. 3.4), a stability analysis.

3.1 Equation of motion

To simplify notation we introduce the overlap $m_\mu(t)$ as a measure of the correlations in the firing pattern of the neurons with pattern μ

$$m_\mu(t) = \frac{2}{N(1-a^2)} \sum_{j=1}^{N} (\xi_j^\mu - a) S_j . \tag{8}$$

Here $a = \langle \xi_j^\mu \rangle$ is the mean of random pattern μ. The overlap $m_\mu(t)$ takes its maximum value 1, if all neurons i in the 'foreground' of pattern μ ($\xi_i^\mu = +1$) fire synchronously during one time step while all 'background' neurons j ($\xi_j^\mu = -1$) stay quiescent. It vanishes, if the firing of 'foreground' as well as 'background' neurons occurs stochastically and N is large ($N \to \infty$).

Substituting (4), (7), and (8) into Eq. (6) yields the postsynaptic potential

$$h_i^{\text{syn}}(t) = \sum_{\mu=1}^{q} \xi_i^\mu \sum_{\tau=0}^{\infty} \epsilon(\tau) m_\mu(t - \tau - \Delta_i^{\text{ax}}) + \sum_{\tau=0}^{\tau_{\max}} \eta(\tau) S_i(t - \tau - \Delta_i^{\text{inh}}) . \tag{9}$$

At this point, it is convenient to group the neurons into ensembles of those neurons that have identical 'properties'. To this end we introduce *sublattices* (van Hemmen et al. 1986, 1988; van Hemmen and Kühn 1986)

$$L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho) = \{ i \mid \xi_i^\mu = \mathbf{x}, \Delta_i^{\text{ax}} = D^{\text{ax}}, h_i^{\text{inh}}(t) = -\eta, h_i^{\text{ref}}(t) = -\varrho \} , \tag{10}$$

that gather neurons with the same storage vector ($\xi_i^\mu, 1 \leqslant \mu \leqslant q$), with an identical axonal delay Δ_i^{ax}, the same (momentary) inhibition strength $h_i^{\text{inh}}(t)$ and refractory field $h_i^{\text{ref}}(t)$ into a common class. If $p(\mathbf{x}, D^{\text{ax}}, \eta, \varrho, t)$ is the portion of neurons that belong at time t to the sublattice $L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho)$, then the overlap can be written (Herz et al. 1988, 1989)

$$m_\mu(t+1) = [N(1-a^2)]^{-1} \sum_{\mathbf{x}} \sum_{D^{\text{ax}}=\Delta_{\min}^{\text{ax}}}^{\Delta_{\max}^{\text{ax}}} \sum_{\eta=0}^{\eta^{\max}} \sum_{\varrho=0}^{R} \times p(\mathbf{x}, D^{\text{ax}}, \eta, \varrho, t) x^\mu \times \{1 + \tanh[\beta(h(\mathbf{x}, D^{\text{ax}}, \eta, \varrho, t) - \theta)]\} , \tag{11}$$

with

$$h(\mathbf{x}, D^{\text{ax}}, \eta, \varrho, t) = \sum_{\mu=1}^{q} x^\mu \sum_{\tau=0}^{\infty} \epsilon(\tau) m_\mu(t - \tau - D^{\text{ax}}) - \eta - \varrho + h^{\text{ext}}(\mathbf{x}) . \tag{12}$$

For the sake of simplicity we have assumed that the external signal depends on the storage vector $\mathbf{x}$ only.

Equations (11) and (12) describe the dynamic evolution of the macroscopic network states and contain all possible solutions of our model network. The derivation of these results is based on a time-dependent mean-field-theory which is exact in the case of a finite number of patterns in a large network ($N \to \infty$). See Gerstner and van Hemmen (1992a) for details of the derivation.

Two basic types of solution – stationary and oscillatory overlaps – may occur, which we study in the limit of noiseless threshold neurons ($\beta \to \infty$). Intermediate between both cases is a regime of weak locking which is discussed in Sect. 3.5.

3.2 Stationary states

As stationary state is defined by the condition $m_\mu(t) \equiv m_\mu$ for all patterns $1 \leqslant \mu \leqslant q$. In this case (12) can be simplified

$$h(\mathbf{x}, D^{\text{ax}}, \eta, \varrho, t) = \sum_{\mu=1}^{q} x^\mu m_\mu - \eta - \varrho + h^{\text{ext}}(\mathbf{x}) , \tag{13}$$

where we have used the normalization condition $\sum_{\tau=0}^{\infty} \epsilon(\tau) = 1$. Equation (13) shows that the interaction of neuron $i \in L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho)$ with all of the 'Hebbian' neurons and the external signal yields a *constant* input $h_{\text{const}}(\mathbf{x}) = \sum_{\mu=1}^{q} x^\mu m_\mu + h^{\text{ext}}(\mathbf{x})$. The only time-dependent part in the postsynaptic potential of a neuron i belonging to $L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho)$ stems from the interaction with its inhibitory partner neuron and its own refractory field. The effect of inhibition and refractoriness can be understood easily.

If $h_{\text{const}}(\mathbf{x}) < \theta$, a neuron $i \in L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho)$ stays quiescent and fires no spikes ($n_B = 0$). If, however, $h_{\text{const}}(\mathbf{x}) > \theta$, then neuron i fires with maximum frequency and emits a burst of spikes until the inhibitory feedback becomes strong enough to end the spiking. Only after another time τ^0 when the inhibitory signal has declined sufficiently, i.e., $\eta(\tau^0 - 1) < \theta - h_{\text{const}}(\mathbf{x})$ and $\eta(\tau^0) \geqslant \theta - h_{\text{const}}(\mathbf{x})$, the firing can start again.

This result can be used to simplify (11). Let us assume that the IPSP $\eta(\tau)$ is a sharply rising function and blocks firing immediately when it sets in. Then a burst of neuron i that has started at $t = 0$ is ended at $t = \Delta_i^{\text{inh}}$ when the inhibitory feedback due to the first spike arrives. This leaves the neuron time to fire a total of $n_B(\Delta_i^{\text{inh}}) = 1 + \text{Int}[\Delta_i^{\text{inh}}/(\tau_{\text{ref}} + 1)]$ spikes where $(\tau_{\text{ref}} + 1)^{-1}$ is the firing frequency during the burst and Int[·] denotes the integer part of the term in square brackets. Thus spiking occurs at times $t_i = 0, (\tau_{\text{ref}} + 1), \cdots, [n_B(\Delta_i^{\text{inh}}) - 1](\tau_{\text{ref}} + 1)$ and stops afterwards due to the inhibitory feedback. Only if the inhibition that is due to the last spike of the burst has declined sufficiently the neuron can fire again and start the next burst. The bursting period T_B is therefore given by

$$T_B(\Delta_i^{\text{inh}}) = [n_B(\Delta_i^{\text{inh}}) - 1](\tau_{\text{ref}} + 1) + \tau^0 . \tag{14}$$

We see that the result (14) only depends on the inhibitory delay loop Δ_i^{inh}, so is independent of the axonal delays times Δ_i^{ax}. Instead of a classification of the neurons by sublattices $L(\mathbf{x}, D^{\text{ax}}, \eta, \varrho)$ it is now more

convenient to introduce new sublattices defined by $L(\mathbf{x}, D^{\text{inh}}) = \{i \mid \xi_i^\mu = \mathbf{x}, \Delta_i^{\text{inh}} = D^{\text{inh}}\}$. If $p(\mathbf{x}, D^{\text{inh}})$ is the portion of neurons belonging to $L(\mathbf{x}, D^{\text{inh}})$ we find for the overlap

$$m_\mu = \frac{2}{N(1-a^2)} \sum_{\mathbf{x}} \sum_{D^{\text{inh}}=\Delta_{\min}^{\text{inh}}}^{\Delta_{\max}^{\text{inh}}} x^\mu p(\mathbf{x}, D^{\text{inh}}) \frac{n_B(D^{\text{inh}})}{T_B(D^{\text{inh}})}. \quad (15)$$

The quantity $[n_B(D^{\text{inh}})/T_B(D^{\text{inh}})]$ is the mean firing rate of neurons with an inhibitory delay loop $\Delta_i^{\text{inh}} = D^{\text{inh}}$. This result, Eq. (15), is in accordance with a much more general theorem that in a stationary state of a fully connected network storing a finite number of patterns the *mean firing rate* of the neurons is the only important quantity (Gestner and van Hemmen 1992b).

The Eqs. (13)–(15) allow a straightforward numerical solution for the stationary overlap m_μ. In particular, it is possible to find the retrieval states $m_\mu = m\delta_{\mu\nu}$ as a function of the external signal $h^{\text{ext}}(\mathbf{x}) = \gamma(x^\nu + 1)/2$. To simplify the analysis, we now consider time as a continuous variable and calculate τ_0 from the exact threshold equation $\eta(\tau_0) = \theta - \gamma$. In Fig. 4 the theoretical predictions for a large and noiseless network are compared with simulation results of a network consisting of two layers of 4000 neurons. Whereas at $T = 0$ the transition is sharp and occurs exactly at $\gamma = 0.12$ (open diamonds), it is smoothened to a continuous transition at a finite noise level ($\beta = 15$, filled circles). These results can be understood with the help of Fig. 1b. According to (13)–(15), the stationary retrieval overlap is given by $m = \bar{f}(m + \gamma)$ where $\bar{f}$ is the mean firing rate [in kHz] averaged over all pairs of neuron and inhibitory partner neuron. Thus the intersection of a typical gain function of a neuron pair (Fig. 1b, solid line – noiseless; circles – $\beta = 15$) with a straight line of slope one (dashed) yields the stationary retrieval overlap. The external signal γ shifts the dashed line parallel to the x-axis. Using this graphical construction we can explain the nature of the solution in Fig. 4. In the noiseless case we have the trivial solution for small γ. With increasing external signal, we suddenly obtain a nonzero firing rate, move through a regime of bistability, and then arrive at a regime of large retrieval overlap only. In the case of finite but high enough temperature the gain function does not show a steep increase at threshold and the overlap comes up continually with the external input.

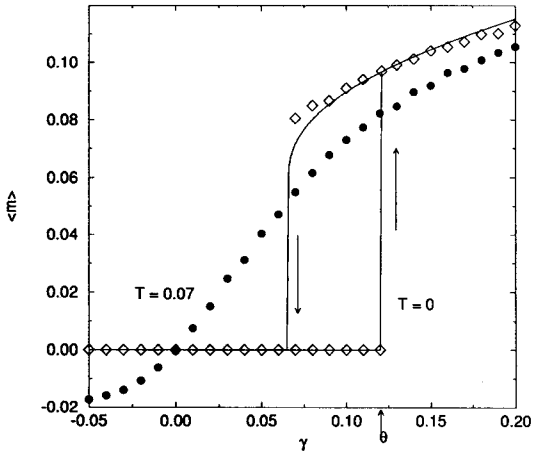

Fig. 4. Stationary retrieval $\langle m \rangle$ as a function of the external stimulus. Theoretical results of a large ($N \to \infty$) and noiseless ($\beta \to \infty$) network are shown by the *solid lines*. Simulation results for a network of $N = 4000$ neuron pairs are plotted as *open diamonds* ($\beta = \infty$) and *filled circles* ($\beta = 15$). If the external stimulus γ is increased, the noiseless system passes a regime of bistability which ends at $\gamma = \theta = 0.12$. There the overlap jumps to the upper branch (pattern retrieval). The hysteresis loop has been indicated by arrows. At finite temperature ($\beta = 15$) the transition to the retrieval state is continuous. These results can be understood with the aid of a graphical solution, indicated in Fig. 1b, using the above gain function of a neuron pair

The remaining difference in Fig. 4 between theory and simulation in the noiseless case is apart from finite-size effects also due to a small oscillation in the overlap. The stationarity condition is thus a good approximation, but it is not exactly fulfilled. Oscillations with a large amplitude are considered in the next subsection.

3.3 Oscillatory states

In addition to stationary retrieval states with constant overlap $m_\mu(t) \equiv m\delta_{\mu\nu}$ there are also collective oscillation states

$$m_\mu(t) = \delta_{\mu\nu} f(t + nT_{\text{osc}}), \qquad n \in \mathbb{Z} \quad (16)$$

where T_{osc} is the period of a collective oscillation. While f is a T_{osc}-periodic function, $\mathbb{Z}$ denotes the integers.

We have seen in the preceding paragraph that neurons with different delay loops Δ_i^{inh} have a different intrinsic bursting period $T_B(\Delta_i^{\text{inh}})$, if the neurons are subject to a constant input. The above Ansatz (16) presupposes that a collective oscillation with a *common* period T_{osc} is nevertheless possible. In other words, Eq. (16) implies 'locking' of oscillatory elements with variable intrinsic frequencies. In this subsection we show that these 'locked' oscillations indeed exist. The next subsection is devoted to the questions under which condition they are *stable* solutions of the system.

In order to check whether oscillatory solutions are possible, we assume that all neurons in the foreground of a specific pattern ($\xi_i^\nu = 1$) have fired regularly in a coherent oscillation for all times in the past ($t < 0$). To be more explicit, we assume that there have been collective bursts at times $T_F = -nT_{\text{osc}}$ where $n \in \mathbb{N}$ denotes the positive integers and T_{osc} is the unknown bursting period which we are going to determine self-consistently. Despite the common period T_{osc} groups of neurons with different delay times will not burst exactly at the same time since neurons with short delays start a little too early in relation to the collective firing time T_F and neurons with long delays fire a little too late. We denote the delay of the burst start for a neuron with axonal and inhibitory delays $(D^{\text{inh}}, D^{\text{ax}})$ compared to the collective firing time T_F by the quantity $\delta(D^{\text{inh}}, D^{\text{ax}})$. If we now remember from the preceding subsection that each burst contains $n_B(\Delta_i^{\text{inh}})$ spikes fired with a frequency $(\tau_{\text{ref}} + 1)^{-1}$, then we can write the spiking times for a neuron $i \in L(D^{\text{inh}}, D^{\text{ax}})$ where

$L(D^{\text{inh}}, D^{\text{ax}}) = \{i \mid \Delta_i^{\text{inh}} = D^{\text{inh}}, \Delta_i^{\text{ax}} = D^{\text{ax}}\}$ and $(\xi_i^\nu = 1)$ in the form

$$t_i = -nT_{\text{osc}} + \delta(D^{\text{inh}}, D^{\text{ax}}) + (k-1)(\tau_{\text{ref}} + 1)\,, \qquad (17)$$

where $T_F = -nT_{\text{osc}}$ with $n \in \mathbb{N}$ is the formal collective firing time, $\delta(D^{\text{inh}}, D^{\text{ax}})$ is the shift in the burst start of a neuron $i \in L(D^{\text{inh}}, D^{\text{ax}})$, and k with $1 \leqslant k \leqslant n_B(D^{\text{inh}})$ denotes the k^{th} spike in the burst. The origin of time is chosen in such a way that the next formal collective burst ($n = 0$) occurs at $t = T_F = 0$. In the following we are going to determine the unknown quantities T_{osc} and $\delta(D^{\text{inh}}, D^{\text{ax}})$.

To simplify notation we define a summation of all past EPSP's

$$\tilde{\epsilon}(D^{\text{inh}}; T, t) = \sum_{n=1}^{\infty} \sum_{k=1}^{n_B} \epsilon[t + nT - (k-1)(\tau_{\text{ref}} + 1)]\,. \qquad (18)$$

T and t are considered as free variables, whereas D^{inh} is a parameter. The quantity $\tilde{\epsilon}$ as a function of T can be used to determine the oscillation period T_{osc} in the following way. Because of the regular firing in the past, viz. Eq. (17), the next bursting of a neuron $i \in L(D^{\text{inh}}, D^{\text{ax}})$ is due at time $\delta(D^{\text{inh}}, D^{\text{ax}})$. This implies that the threshold condition $h_i^{\text{syn}}(t) + h_i^{\text{ext}}(t) \geqslant \theta$ is fulfilled at time $t = \delta(D^{\text{inh}}, D^{\text{ax}})$, but not yet at $t = \delta(D^{\text{inh}}, D^{\text{ax}}) - 1$. If we replace the inequality by an equality and insert the explicit expression for $h_i^{\text{syn}}(t)$, we can write the threshold condition

$$\theta = \sum_{D_0^{\text{inh}} = \Delta_{\min}^{\text{inh}}}^{\Delta_{\max}^{\text{inh}}} \sum_{D_0^{\text{ax}} = \Delta_{\min}^{\text{ax}}}^{\Delta_{\max}^{\text{ax}}} \times \tilde{\epsilon}[D_0^{\text{inh}}; T, \delta(D^{\text{inh}}, D^{\text{ax}}) - D^{\text{ax}} - \delta(D_0^{\text{inh}}, D_0^{\text{ax}})] + \eta\{T - [n_B(D^{\text{inh}}) - 1](\tau_{\text{ref}} + 1) - D^{\text{inh}}\} + h^{\text{ext}}(\mathbf{x})\,. \qquad (19)$$

As before we take $h^{\text{ext}}(\mathbf{x}) = \gamma(x^\nu + 1)/2$. Simultaneous solution of (19) for all groups $L(D^{\text{inh}}, D^{\text{ax}})$ yields the oscillation period $T_{\text{osc}} = \text{Int}[T + 1]$ and the shift in the bursts start $\delta(D^{\text{inh}}, D^{\text{ax}})$.

To simplify expression (19) we now assume that $\tilde{\epsilon}$ and η vary only slowly during one time step and that differences in the delays D^{inh} and D^{ax} are small and result also in a spread of firing times $\delta(D^{\text{inh}}, D^{\text{ax}})$. In this case, $\tilde{\epsilon}$ and η can be taken continuous and differentiable. Expansion around the mean $\bar{D}^{\text{inh}}$ and $\bar{D}^{\text{ax}}$ yields after some algebra the approximate formula

$$\theta \approx \eta(T - 2\bar{D}^{\text{inh}}) + \tilde{\epsilon}(\bar{D}^{\text{inh}}; T, -\bar{D}^{\text{ax}}) + h^{\text{ext}}(\mathbf{x})\,, \qquad (20)$$

where $\bar{D}^{\text{inh}} \approx [n_B(\bar{D}^{\text{inh}}) - 1](\tau_{\text{ref}} + 1)$ has been used. This allows a solution for the oscillation period T_{osc}.

Within the same approximation, the starting times of the bursts depend on the inhibitory delay through

$$\delta(D^{\text{inh}}, D^{\text{ax}}) = 2\,\frac{\frac{\mathrm{d}}{\mathrm{d}t}\eta(T_{\text{osc}} - 2\bar{D}^{\text{inh}})}{\frac{\mathrm{d}}{\mathrm{d}t}\tilde{\epsilon}(\bar{D}^{\text{inh}}; T_{\text{osc}}, -\bar{D}^{\text{ax}})}\,(D^{\text{inh}} - \bar{D}^{\text{inh}})\,. \qquad (21)$$

If we assume realistic parameters for the shape of the EPSP, the response function $\epsilon(\tau)$ shows a pronounced maximum followed by a fast decay. Then $\tilde{\epsilon}(\bar{D}^{\text{inh}}; T, -\bar{D}^{\text{ax}})$ can be approximated by

$$\tilde{\epsilon}(\bar{D}^{\text{inh}}; T, -\bar{D}^{\text{ax}}) \approx \epsilon(T - \bar{D}^{\text{ax}}) n_B(\bar{D}^{\text{inh}})\,. \qquad (22)$$

This allows a simple graphical interpretation of the above result; see Fig. 5. The firing period T_{osc} is given by the intersection of the appropriately scaled and delayed graphs of $\eta(\tau)$ and $\epsilon(\tau)$; cf. Eq. (20). The spread of the starting times of the bursts is determined by the slope of the graphs at the intersection point; see Eq. (21).

In a reasonable oscillation scenario, we expect that the fast neurons with $\Delta_i^{\text{inh}} < \bar{D}^{\text{inh}}$ come a bit too early ($\delta_i < 0$) in relation to the collective firing, whereas slow neurons $\Delta_i^{\text{inh}} > \bar{D}^{\text{inh}}$ are a bit too late ($\delta_i > 0$). Equation (21) tells us that this is true only if $\mathrm{d}\tilde{\epsilon}/\mathrm{d}t$ has the same sign as $\mathrm{d}\eta/\mathrm{d}t$. Since $(\mathrm{d}\eta/\mathrm{d}t)(T_{\text{osc}} - 2\bar{D}^{\text{inh}}) > 0$, it follows that $\tilde{\epsilon}$ must have a *positive slope* at the firing point. We will see in the following paragraph that this is also the condition of a stable oscillation.

3.4 A condition of locking

So far we have assumed that a coherent oscillation exists and – based on the assumption that the collective bursting has been stable for a long time in the past [cf. Eq. (17)] – we have calculated the period T_{osc} of the oscillation and the delay $\delta(D^{\text{inh}}, D^{\text{ax}})$ of the burst's start for the various groups of neurons.

In this subsection, we derive a condition that must be fulfilled to ensure that an oscillation is indeed stable. To this end, we assume that one of the neurons, let us say neuron $j \in L(D^{\text{inh}}, D^{\text{ax}})$, is not perfectly in time with its group, but starts bursting a little too early or too late, in other words, its firing is shifted by a short time Δt. The oscillation is stable, if the neuron is 'drawn back' into its group, i.e., if the time shift $\tilde{\Delta t}$ is reduced during the next cycle.

The synaptic part of the membrane potential has two contributions

$$h_j^{\text{syn}}(t) = h_j^{\epsilon}(t) + h_j^{\eta}(t)\,. \qquad (23)$$

The first term

$$h_j^{\epsilon}(t) = \sum_{D_0^{\text{inh}}, D_0^{\text{ax}}} \tilde{\epsilon}[D_0^{\text{inh}}; T, \delta(D^{\text{inh}}, D^{\text{ax}}) - D^{\text{ax}} - \delta(D_0^{\text{inh}}, D_0^{\text{ax}})]\,, \qquad (24)$$

is induced by the signals of the other neurons in the net and is unchanged compared to the unperturbed system. The inhibitory term, however, it shifted due to the perturbation [cf. (19)].

$$h_j^{\eta}(t) = \eta\{t - [n_B(D^{\text{inh}}) - 1](\tau_{\text{ref}} + 1) - D^{\text{inh}} - \Delta t\} = h_0^{\eta}(t - \Delta t)\,, \qquad (25)$$

where the subscript 0 indicates the unperturbed system. The next firing occurs at $t_0 - \tilde{\Delta t}$ where t_0 is the firing time of the unperturbed network. In the limit of contin-

uous time, the threshold condition is

$$h_j^\epsilon(t_0+\tilde{\Delta t})+h_0^\eta(t_0+\tilde{\Delta t}-\Delta t)=\theta-h_j^{\mathrm{ext}}. \tag{26}$$

Expansion around t_0 and using the fact that $h_j^\epsilon(t_0)+h_0^\eta(t_0)=\theta-h_i^{\mathrm{ext}}(t)$ yields

$$\tilde{\Delta t}=\frac{\mathrm{d}}{\mathrm{d}t}h_0^\eta(t_0)\left[\frac{\mathrm{d}}{\mathrm{d}t}h_0^\eta(t_0)+\frac{\mathrm{d}}{\mathrm{d}t}h_j^\epsilon(t_0)\right]^{-1}\Delta t. \tag{27}$$

Since $(\mathrm{d}h_0^\eta/\mathrm{d}t)(t_0)>0$, a condition for locking is $(\mathrm{d}h_j^\epsilon/\mathrm{d}t)(t_0)>0$. In other words, a global oscillation is stable only if a burst starts while excitation is still growing.

3.5 Weak locking

Equation (27) gives a condition for a *globally* locked oscillation. But what happens if some of the neurons, let us say all neurons in $L(D^{\mathrm{inh}}, D^{\mathrm{ax}})$, do not fulfill (27)? If so, they will remain out of phase, once they have dropped out of the collective oscillation. This does not imply that the oscillation is destroyed completely. It is also possible that only a small group of neurons with very long or very short axonal delay Δ_i^{ax} drop out of the collective oscillation whereas all other neurons remain in an oscillatory state. In this case, the macroscopic overlap is – as before – a T-periodic function of time, but not all neurons fire exactly T-periodic. Some of the neurons (i.e., neurons in $L(D^{\mathrm{inh}}, D^{\mathrm{ax}})$) occasionally slip through and 'miss' the collective firing. We call this a state of *weak locking*.

To find a lower bound of the regime of 'weakly locked' solutions we can adopt an iterative approach. First, we calculate the globally locked solution, and determine the stability of locking (27) for all neurons of all sublattices $L(D^{\mathrm{inh}}, D^{\mathrm{ax}})$. If (27) is not fulfilled for some neurons, we *assume* that they are completely out of phase and give – on the average – no contribution to the T-periodic Ansatz (16), providing only a stationary background. Based on this assumption we repeat the procedure, calculate a new period, and check whether all remaining neurons are stably locked, and so on. If the iterative procedure converges to a state with 'non-participating' neurons, then a 'weakly locked' solution is possible. This criterion, however, is only a necessary condition, since simulations show that the neurons which have dropped out of the collective oscillation are not completely out of phase, but only slip through occasionally. Due to the remaining oscillations in the overlap which acts as a periodic stimulus on those neurons, they have a tendency to spike in phase with the rest of the network. Thus they are, in a true sense, *weakly locked.*

The above arguments are valid in the noiseless case ($T=0$). The regimes of stationary states, global oscillations and weakly locked oscillatory states are determined only by the width and position of the distributions of the delays Δ_i^{inh} and Δ_i^{ax}. If noise is added, each neuron fires stochastically, and the spikes may come a little too early or too late in comparison with the noiseless case. It is therefore to be expected that adding noise lowers the oscillation amplitude and drives the system from global oscillation to weak locking or stationarity. Simulations of a network at a finite noise level which show this behavior are presented in the next section.

4 Macroscopic states of the network: Simulations

In the preceding section we have shown that two basic types of solution are possible: Oscillatory and stationary retrieval of patterns. Oscillatory solutions can be divided in globally and weakly locked oscillations. The theoretical arguments have demonstrated that it is the timing of the EPSP in relation to the IPSP which determines the solution type. In this section, we test the validity of the theoretical results in simulations of a network consisting of two layers of 4000 neurons at a *finite* noise level ($\beta=15$) with $q=5$ patterns and $a=-0.8$. In passing we note that extensively many patterns ($q=200$ in a system of $N=4000$) will do as well. The EPSP and IPSP are modelled with a biological shape as shown in Fig. 3. The delay in the inhibitory loop of a specific neuron pair is chosen once and for all from a block-shaped distribution that varies between $\Delta_{\mathrm{min}}^{\mathrm{inh}}=3$ ms and $\Delta_{\mathrm{max}}^{\mathrm{inh}}=6$ ms. The only remaining free parameter then is the duration of the axonal transmission delay Δ_i^{ax}. To test its influence we have considered three different scenarios: see Fig. 5.

4.1 Three scenarios

In all scenarios we start in a randomly chosen state of mean activity a. For the first 200 ms no external signal is applied, then a weak signal is switched on and kept on a constant level of $h_i^{\mathrm{ext}}(t)=\gamma(\xi_i^\mu+1)/2$ with $\gamma=0.2$,

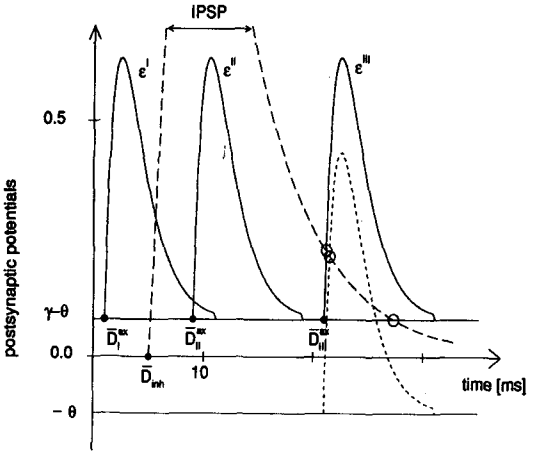

Fig. 5. Relative position of EPSP (*solid line*) and IPSP (*long dashes*) in three different scenarios. Scenario I: Short axonal delays between 0 and 2 ms; mean value $\bar{D}_I^{\mathrm{ax}}=1$ ms. Scenario II: medium axonal delays (8–10 ms, $\bar{D}_{II}^{\mathrm{ax}}=9$ ms). Scenario III: long axonal delays (20–22 ms, $\bar{D}_{III}^{\mathrm{ax}}=21$ ms). The intersection of $\epsilon^{I,III}=n_B\epsilon(\tau-\bar{D}_{I,III}^{\mathrm{ax}})+(\gamma-\theta)$ with the function $-\eta(\tau-2\bar{D}^{\mathrm{inh}})$ yields the oscillation period in scenario I ($T_{\mathrm{osc}}^I=27$ ms) and scenario III ($T_{\mathrm{osc}}=23$ ms); see text for details. In scenario III an oscillation is possible even after the external signal has been turned off ($\gamma=0$, *dashed line*). Scenario II does not allow oscillatory solutions

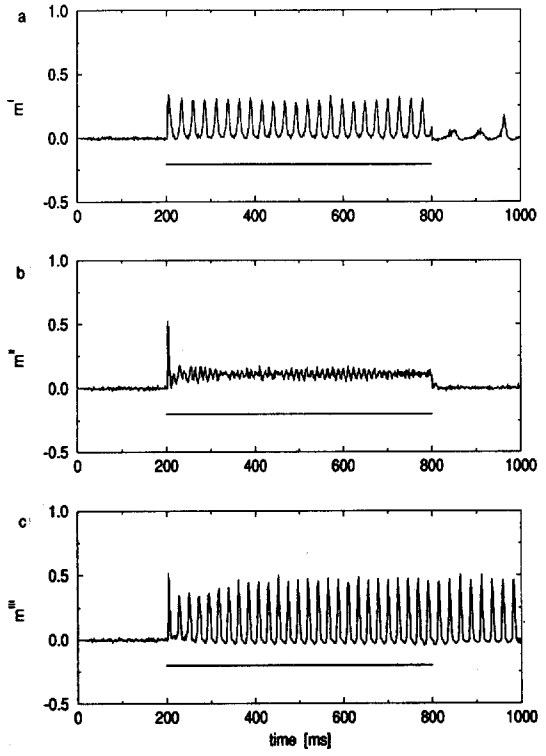

Fig. 6. Response of a network to stimulation. Overlaps with a specific pattern are shown as a function of time for the three different scenarios. The pattern is supported by a weak external signal for a time between 200 and 800 time steps (ms) as indicated by the horizontal bar. *Top:* scenario I, oscillations occur only *while* an external stimulus is applied. *Middle:* scenario II, transient behaviour to a stationary retrieval state. *Bottom:* scenario III, oscillations remain even after the stimulus has been turned off

which is only slightly above threshold. After another 600 ms the signal is switched off again.

In scenario I (Fig. 6a), we have assumed that axonal delays Δ_i^{ax} are short and distributed in a range between 0 and 2 ms with equal probability. With this delay distribution, an oscillation starts, if the signal is switched on, and it continues *as long as the signal is active*. It stops immediately after the signal is turned off. This is consistent with the theoretical results (19) and (27).

In scenario II, the axonal delays are slightly longer, distributed in a range $8\text{ ms} \leqslant \Delta_i^{ax} \leqslant 10\text{ ms}$. In this case, excitation is growing while inhibition is still dominant (see Fig. 5, curve 'II') and an oscillation cannot be stable; cf. Eq. (27). This is confirmed in the simulation (Fig. 6b) where a constant overlap develops after initialization of the signal. The magnitude of the overlap is consistent with the theoretical result (15). If the signal is turned off, the overlap vanishes and the network returns to the quiescent state.

All this is different in scenario III where we have long axonal delays ($20\text{ ms} \leqslant \Delta_i^{ax} \leqslant 22\text{ ms}$). Figure 6c shows that a collective oscillation develops which is stable even *after* the signal has been turned off. We can understand this result by analyzing the graphical interpretation of (20), (22), and (27) as shown in Fig. 5. The intersection of the graph of $-\eta(\tau)$ with the scaled version of $\epsilon(\tau)$ (curve 'III') yields an oscillation period $T_{osc} \approx 23$ ms that matches well with the simulation result, Fig. 6c. In both cases – with or without signal – a collective oscillation is stable, since bursts start while excitation is still growing; cf., Eq. (27).

Scenarios I–III are typical examples of a network behaviour that varies continuously from stationarity to full locking. The transition between the different regimes can be seen in the 'phase diagram' (Fig. 7) where the amplitude of the oscillation is shown by a scale of grey values. Dark areas indicate large oscillations, white area negligible oscillations. Depending on the minimal duration of axonal transmission delays (y-axis) and the width of their distribution (x-axis) the amplitude varies from below 0.1 (stationary) to above 0.3 (perfect locking). Whereas a broader distribution (larger x-values) always lowers the amplitude, a change in the y-values may switch the system from one regime to the next and either increase or decrease the amplitude, as discussed above.

4.2 Interpretation

The most interesting behaviour is seen in scenario I where the network oscillates only *as long as* the external signal is applied. If the signal is removed, the system returns to the stationary resting state. This behaviour in scenario I compares favorably to experiments in the visual cortex of the cat where oscillations are seen only during stimulation. Otherwise a stationary background activity is found.

Scenario I is also the most realistic one, at least for the cat. It is known that axonal delays within one area of the cortex are rather short. Delay times distributed in a range between 0 and 2 ms seem to be a reasonable assumption (Kuffler et al. 1984). The other parameters (rise time of EPSP $\tau_\epsilon \approx 3$ ms, rise time of IPSP ≈ 2 ms, decay time $\tau_\eta \approx 6$ ms, inhibition loop delay 3–6 ms) are also taken in a biologically plausible range. With this set of parameters we find a period of oscillation of approximately 20–25 ms which is consistent with experiments where oscillations with 40–50 Hz have been found (Eckhorn et al. 1988; Gray and Singer 1989).

The theoretical argument (Eq. 27) shows that the oscillation is only weakly stable since $(\mathrm{d}/\mathrm{d}t)h^\epsilon(t_0) \approx 0$ for the 'fast' neurons. This may be a reason that in the noisy environment of real systems oscillations are barely visible (Pawelzik 1991). Simulations with scenario I within a more realistic neurobiological setting and at a higher noise level are presented in a companion paper.

4.3 Spike raster

In contrast to most other models of oscillatory neural networks our approach yields not only global activities and time-averaged firing rates but also *spike trains of single neurons* and their correlations. The full information on the network behaviour is contained in the spike raster which shows all spikes of an arbitrary set of neurons in a time-resolved plot. A spike raster is thus equivalent to experimental multi-electrode-recordings where the activity of several neurons is measured at the same time. The spike raster of Fig. 8 shows the spikes of 10 neurons in scenarios I to III. All neurons from

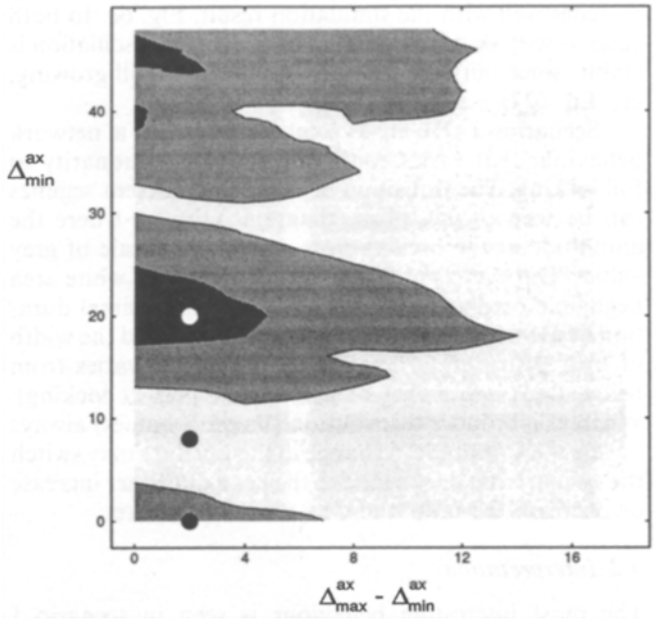

Fig. 7. Phase diagram. The amplitude of an oscillation $m_{osc} = \langle m_{max} - m_{min} \rangle$ is plotted as a function of both minimal axonal delay Δ^{ax}_{min} (y-axis) and width of the delay distribution $\Delta^{ax}_{max} - \Delta^{ax}_{min}$ (x-axis). Dark areas indicate a large-amplitude oscillation ($m_{osc} > 0.3$), white areas indicate a stationary overlap ($m_{osc} < 0.1$) while gray-shaded areas define the region of weak locking ($0.1 < m_{osc} < 0.3$). The three scenarios lie along the line ($x = \Delta^{ax}_{max} - \Delta^{ax}_{min} = 2$ ms) parallel to the y-axis and are marked by *black* and *white circles*. Whereas for a sharp distribution of delay times ($x = 0$), the oscillation amplitude depends critically on the transmission delay, a broad distribution ($x > 16$) always leads to a stationary solution. Note that scenario I lies in a regime intermediate between strong oscillations and stationarity (*grey-shaded area*)

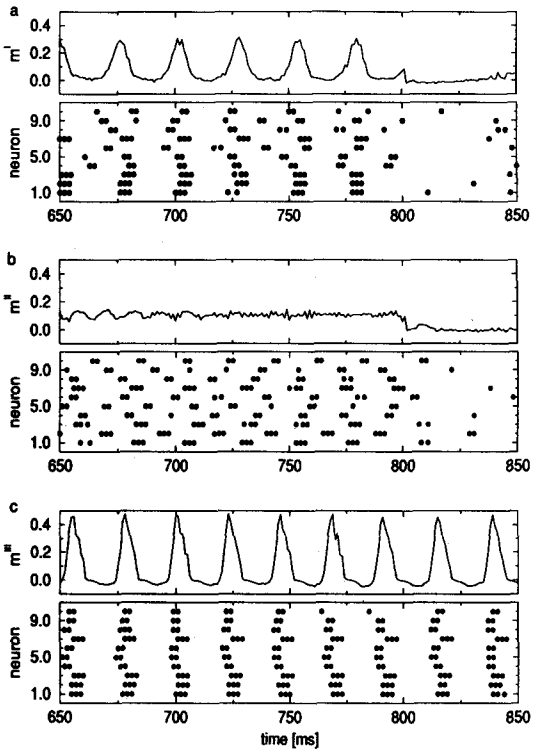

Fig. 8. Spike raster in the three scenarios. The upper curve in (a)–(c) shows a small protion of the overlap function of Fig. 6 with higher time resolution. The signal is active up to $t = 800$ ms and is turned off afterwards. The spike raster below shows the spikes of 10 active neurons. Neurons are numbered and plotted along the y-axis, time is plotted along the x-axis. Each spike of a neuron is marked by a dot. (a) In scenario I, coherent activity – visible as vertical columns of spikes – is dominant but some of the neurons (e.g., numbers 6 and 9) occasionally slip through: 'weak locking'. (b) In scenario II, all neurons burst asynchronously as is confirmed by the stationary overlap. (c) In scenario III, locking is perfect. Note that neurons with short delays (e.g., numbers 4–6) tend to come earlier than neurons with long delays (e.g., number 3 and 7), as predicted by the theory

which recordings are made are numbered and plotted along the y-axis. Time is plotted in x-direction and the spikes of each neuron are marked as a dot along a horizontal line parallel to the x-axis. Coherent activity can then be visualized as vertical columns of dots; see e.g., at $t = 700$ ms in scenario I (Fig. 8a) and III (Fig. 8c). The burst structure of any single neuron (e.g., bursts of two or three spikes) is clearly visible if we follow the spike dots along a horizontal line. A closer look at the spike raster of scenario III reveals that, while bursting is globally synchronized for all active neurons, the burst start of different neurons is not exactly at the same time. Neurons with a short inhibitory delay loop (e.g., #4–6) start firing earlier than those with a long delay loop (e.g., #3 and 7), as predicted by (21). Both groups, however, 'fast' neurons as well as 'slow' neurons, have the same period of global collective oscillation ($T_{osc} \approx 23$ ms). In scenario II, each neuron exhibits bursting activity, but the bursts occur stochastically and are not synchronized. An intermediate case is shown in the spike raster of scenario I where some of the neurons (e.g. #6 and 9) slip through the 'oscillation maze' occasionally, that is, are *'weakly locked'*. Nevertheless, global coherence is still dominant as indicated by the oscillation of the overlap m. The spike raster, however, enables us to reveal additional information that is not obvious, if we look at the overlap only.

5 Discussion and conclusions

We have incorporated a couple of often neglected biological details into formal associative networks and shown their relevance. In particular, we have included an absolute refractory period of the model neuron, a distribution of axonal transmission delays, and, finally, a realistically shaped postsynaptic response (EPSP and IPSP). Our biological approach allows to model network behaviour in more detail and reveals the spiking behaviour of single neurons and correlations between different neurons as well as the global activities.

We have shown that apart from the quiescent state at least two basic types of solution are possible: stationary and oscillatory retrieval of the learnt patterns. It depends on the exact timing of the EPSP in relation to the IPSP whether the system goes into an oscillatory or stationary retrieval state. In the *stationary* state, all neurons in the 'foreground' of a pattern are active, but the activity bursts of different neurons are not synchronized. The overlap – which is a global measure of the retrieval quality of a pattern – takes a constant value

proportional to the mean firing rate of the neurons. Stationary retrieval occurs for medium delays in the axonal transmission (scenario II).

For short (scenario I) or long (scenario III) transmission delays *oscillatory* retrieval is dominant. The period of collective oscillations can be calculated, if transmission delays and the shape of EPSP and IPSP are known. An important conclusion of our analysis is that 'locking' of different neurons into a collective oscillation is possible *only if the total excitatory potential is still growing at the time of firing*. If it is declining, the collective oscillation is unstable and the neurons go into an unsynchronized firing state. In view of the *distribution* of axonal and inhibitory delays the occurrence of locking might seem surprising. We could show both analytically and in the simulations of scenario III that a collective oscillation with a common period is nevertheless possible. Our analysis also reveals that neurons with short delays spike always slightly earlier than those with long delays.

In scenario I where the neurons have axonal delay times in a biologically plausible range, coherent oscillations occur but locking is only weak. Neurons with short delays occasionally slip through and miss the collective firing times. It can thus be speculated that biological networks operate in a regime intermediate between strict locking and unsynchronized firing – in agreement with available biological data (Eckhorn et al. 1988; Kreiter and Singer 1992).

Locking as well as the phase shift between fast and slow units are a much more general phenomenon and occur in many other circumstances (Yamaguchi and Shimizu 1984; Kuramoto and Nishikawa 1987). But while most other models of collective oscillations are based on abstract differential equations, our approach is based on a specific model of neuronal signal transmission. Despite the fact that many biological phenomena have been included in the description, an analytical solution of our model in the limit of a large and noiseless network is nevertheless possible.

There are, of course, a couple of biological details which are still neglected in our approach. As we have explained in Sect. 2.1, we disregard all microscopic phenomena on the level of ion channels and the structure of dendritic trees. Furthermore, we assume throughout the paper that dendritic summation is linear and we neglect all effects of adaptation and relative refractoriness. It should be checked in future work how an incorporation of the above phenomena changes the network behaviour. Preliminary investigations suggest that it does not.

In our model of synaptic transmission, we have assumed that a mathematical description can be separated into two parts: A response function which models the time course of the synaptic response and a synaptic efficacy factor which determines the amplitude of the response. Experiments on hippocampal brain slices suggest that such a separation might be possible. Indeed, it is the amplitude of the postsynaptic response which changes during long-term potentiation while the time course remains unaffected (e.g., Malinow and Miller 1986; Larson and Lynch 1986) We have made no attempt to model the electrical and chemical processes that lead to long-term-potentiation by correlation of pre-and postsynaptic activity (Hebb 1949; see Brown et al. 1989; Bindman et al. 1991 for a review on experiments, Herz et al. 1988; Lisman 1989; Kitajima and Hara 1990 on modeling). Instead, we have taken an effective Ansatz (4) of 'Hebbian' efficacies for random patterns. This allows us to compare our results with the retrieval properties of traditional formal networks (Hopfield 1982). We do not claim, however, that the visual cortex is an association network that stores random patterns by the above rule (4). In fact this rule is rather implausible, since the 'Hebbian' model neurons make excitatory as well as inhibitory connections. In principle, the inhibitory synapses in the 'Hebbian' layer can be removed, if the threshold is adjusted at the same time. For technical reasons, however, we prefer to take the rule (4), which makes analysis more transparent.

On a more global level, the connection topology of our model may be criticised. The 'extreme locality' of inhibition by partner neurons is certainly not a realistic model of biological systems. In the visual cortex of the cat, for example, it is known that only a small fraction of the neurons are inhibitory stellate cells and each cell makes local connections to a group of excitatory cells. Simulations of a more realistic model that preserves the characteristics of the present one but allows a reduced number of inhibitory neurons and local connections to a group of nearby 'Hebbian' neurons show that the basic network behaviour is unchanged (Trefz 1991). In fact, it can be argued that the 'relaxed locality' condition tends to synchronize firing and to stabilize oscillations. Our approach of 'extreme locality' is thus not only the most transparent, but also the most difficult, in a sense the 'worst' case, if we aim at collective oscillations. Regarding the 'Hebbian' neurons, we have taken the other extreme and assumed full connectivity. This would be a bad assumption, if we wanted to model the cortex as a whole, but as a model of a single column or a very small area in the visual cortex of a cat or monkey it is a fair approximation. In a companion paper (Ritz et al. 1993) we show how in a model of a larger area of the cortex a connection topology across several columns can be introduced by distance-dependent axonal delays. Such a more involved model allows to simulate complex network behaviour which can be compared with experimental measurements on the cortex of the cat, and thus to narrow the gap between theory and biology.

References

Abbott LF (1991) Realistic synaptic inputs for model neural networks. Network 2:245–258

Amit DJ (1989) Modeling brain function. Cambridge University Press, Cambridge

Amit DJ, Gutfreund H, Sompolinsky H (1985) Spin-glass models of neural networks. Phys Rev A 32:1007–1032

Amit DJ, Gutfreund H, Sompolinsky H (1987) Statistical mechanics of neural networks near saturation. Ann Phys (NY) 173:30–67

Baird B (1986) Nonlinear dynamics of pattern formation and pattern recognition in the rabbit olfactory bulb. Physica D 22:150–175

Bindman L, Christofi G, Murphy K, Nowicky A (1991) Long-term potentiation (LTP) and depression (LTD) in the neocortex and

hippocampus: an overview. In: Stone TW (ed), Aspects of synaptic transmission, vol 1. Taylor & Francis, London
Brown TH, Ganong AH, Kairiss EW, Keenan CL, Kelso SR (1989) Long-term potentation in two synaptic systems of the hippocampal brain slice. In: Byrne JH, Berry WO (eds) Neural models of plasticity. Academic Press, San Diego, pp 266–306
Buhmann J, Schulten K (1986) Association, recognition and storage in a model network with physiological neurons. Biol Cybern 54:319–335
Buhmann J (1989) Oscillations and low firing rates in associative memory neural networks. Phys Rev A 40:4145–4148
Bush PC, Douglas RJ (1991) Synchronization of bursting action potential discharge in a model network of neocortical neurons. Neural Comput 3:19–30
Domany E, van Hemmen JL, Schulten K (eds) (1991) Models of neural networks. Springer, Berlin Heidelberg New York
Eckhorn R, Bauer R, Jordan W, Brosch M, Kruse W, Munk M, Reitboeck HJ (1988) Coherent oscillations: A mechanism of feature linking in the visual cortex? Biol Cybern 60:121–130
Ekeberg Ö, Wallen P, Lansner A, Traven H, Brodin L, Grillner S (1991) A computer based model for realistic simulations of neural networks. Biol Cybern 65:81–90
Engel AK, König P, Singer W (1991) Direct physiological evidence for scene segmentation by temporal coding. Proc Natl Acad Sci USA 88:9136–9140
Gerstner W, van Hemmen JL (1992a) Associative memory in a network of 'spiking' neurons. Network 3:139–164
Gerstner W, van Hemmen JL (1992b) Universality in neural networks: The importance of the mean firing rate. Biol Cybern 67:195–205
Gray CM, Singer W (1989) Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc Natl Acad Sci USA 86:1698–1702
Gray CM, König P, Engel AK, Singer W (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar syncronization which reflects global stimulus properties. Nature 338:334–337
Hansel D, Sompolinski H (1992) Synchronization and computation in a chaotic neural network. Phys Rev Lett 68:718–721
Hebb DO (1949) The organization behavior. Wiley, New York
Hemmen JL van, Kühn R (1986) Nonlinear neural networks. Phys Rev Lett 57:913–916
Hemmen JL van, Grensing D, Huber A, Kühn R (1986) Elementary solution of classical spin glass models. Z Phys B-Condensed Matter 65:53–63
(1988) Nonlinear neural networks I and II. J Stat Phys 50:231–257 and 259–293
Hemmen JL van, Gerstner W, Herz AVM, Kühn R, Sulzer and B, Vaas M (1990) Encoding and decoding of patterns which are correlated in space and time. In: Dorffner G (ed) Konnektionismus in Artificial Intelligence und Kognitionsforschung, Springer, Berlin Heidelberg New York
Hemmen JL van, Gerstner W, Ritz R (1992) A 'microscopic' model of collective oscillations in the cortex. In: Taylor JG, Caianiello EK, Cotterill RNJ, Clark JW (eds) Neural network dynamics. Springer, Berlin Heidelberg New York, pp 250–257
Herz A, Sulzer B, Kühn R, van Hemmen JL (1988) The Hebb rule: Representation of static and dynamic objects in neural nets. Europhys Lett 7:663–669
(1989) Hebbian learning reconsidered: Representation of static and dynamic objects in associative neural nets. Biol Cybern 60:457–467
Hodgkin AL, Huxley AF (1952) A quantitative description of ion currents and its applications to conduction and excitation in nerve membranes. J Physiol (London) 117:500–544
Hopfield JJ (1982) Neural networks and physical systems with emergent collective computational abilities. Proc Natl Acad Sci USA 79:2554–2558
Hopfield JJ (1984) Neurons with graded response have computational properties like those of two-state neurons. Proc Natl Acad Sci USA 81:3088–3092
Horn D, Usher M (1989) Neural networks with dynamical thresholds. Phys Rev A 40:1036–1040
Horn D, Sagi D, Usher M (1991) Segmentation, binding and illusory conjunctions. Neural Comput 3:510–525
Jack JJB, Noble D, Tsien RW (1975) Electric current flow in excitable cells, Clarendon Press, Oxford
Kitajima T, Hara K (1990) A model of the mechanisms of long-term potentiation in the hippocampus. Biol Cybern 64:33–39
König P, Schillen TB (1991) Stimulus-dependent assembly formation of oscillatory responses: I. Synchronization. Neural Comput 3:155–166
Kreiter AK, Singer W (1992) Oscillatory neuronal response in the visual cortex of the awake macaque monkey. Eur J Neurosci 4:369–375
Kuffler SW, Nicholls JG, Martin AR (1984) From neuron to brain, 2nd Ed. Sinauer, Sunderland
Kuramoto Y, Nishikawa I (1987) Statistical macrodynamics of large dynamical systems. Case of a phase transition in oscillatory communities. J Stat Phys 49:569–605
Kurrer C, Nieswand B, Schulten K (1990) A model for synchroneous activity in the visual cortex. In: Babloyantz A (ed) Self-organization, emerging properties and learning. Plenum Press, New York
Larson J, Lynch G (1986) Induction of synaptic potentiation in Hippocampus by patterned stimulation involves two events. Science 232:985–988
Lisman J (1989) A mechanism for Hebb and anti-Hebb processes underlying learning and memory. Proc Natl Acad Sci USA 86:9574–9578
Malinow R, Miller JP (1986) Synaptic hyperpolarization during conditioning reversibly blocks induction of long-term potentiation. Nature 320:529–530
Malsburg C von der, Schneider W (1986) A neural cocktail-party processor. Biol Cybern 54:29–40
McCormick DA (1990) Membrane properties and neurotransmitter actions. In: Sheperd GM (ed) The synaptic organization of the brain, 3rd Ed. Oxford University Press, Oxford
Pawelzik K (1991) Nichtlineare Dynamik und Hirnaktivität. Verlag Harri Deutsch, Frankfurt
Rall W (1964) Theoretical significance of dendritic trees for neuronal input-output relations. In: Reiss RF (ed) Neural theory and modeling. Stanford University Press, Stanford, pp 73–97
Ritz R (1991) Kollektive Oszillationen in neuronalen Netzen. Diplomarbeit, Technische Universität München
Ritz R, Gerstner W, Hemmen JL van (1993) A biologically motivated and analytically soluble model of collective oscillations in the cortex: II. Association, segmentation, and binding. IV. Columnar organization (in preparation)
Sompolinsky H, Golomb D, Kleinfeld D (1990) Global processing of visual stimuli in a neural network of coupled oscillators. Proc Natl Acad Sci USA 87:7200–7204
Schuster HG, Wagner P (1990a) A model for neuronal oscillations in the visual cortex 1. Mean-field theory and derivation of the phase equations. Biol Cybern 64:77–82
Schuster HG, Wagner P (1990b) A model for neuronal oscillations in the visual cortex 2. Phase description and feature dependent synchronization. Biol Cybern 64:83–85
Sporns O, Gally JA, Reeke GN, Edelman GM (1989) Reentrant signaling among simulated neuronal groups leads to coherency in their oscillatory activity. Proc Natl Acad Sci USA 86:7265–7269
Sporns O, Tononi G, Edelman GM (1991) Modeling perceptual grouping and figure-ground segregation by means of active reentrant connections. Proc Natl Acad Sci USA 88:129–133
Trefz T (1991) Oszillationen im Cortex. Diplomarbeit, Technische Universität München
Wang D, Buhmann J, von der Malsburg C (1990) Pattern segmentation in associative memory. Neural Comput 2:94–106
Wilson AM, Bower JM (1991) A computer simulations of oscillatory behavior in primary visual cortex. Neural Comput 3:498–509
Wilson HR, Cowan JD (1972) Excitatory and inhibitory interactions in localized populations of model neurons. Biophys J 12:1–24
Yamaguchi Y, Shimizu H (1984) Theory of selfsynchronization in the presence of native frequency distribution and external noises. Physica D 11:212–226