J. Physiol. (Paris) 94 (2000) 473–488

PII: S0928-4257(00)01098-6/REV

Modelling studies on the computational function of fast temporal structure in cortical circuit activity

Friedrich T. Sommer[a]*, Thomas Wennekers[b]

[a] *University of Ulm, Department of Neural Information Processing, 89069 Ulm, Germany*
[b] *Max-Planck-Institute for Mathematics in the Sciences, Inselstraße 22-26, 04103 Leipzig, Germany*

Abstract – The interplay between modelling and experimental studies can support the exploration of the function of neuronal circuits in the cortex. We exemplify such an approach with a study on the role of spike timing and gamma-oscillations in associative memory in strongly connected circuits of cortical neurones. It is demonstrated how associative memory studies on different levels of abstraction can specify the functionality to be expected in real cortical neuronal circuits. In our model overlapping random configurations of sparse cell populations correspond to memory items that are stored by simple Hebbian coincidence learning. This associative memory task will be implemented with biophysically well tested compartmental neurones developed by Pinsky and Rinzel [58]. We ran simulation experiments to study memory recall in two network architectures: one interconnected pool of cells, and two reciprocally connected pools. When recalling a memory by stimulating a spatially overlapping set of cells, the completed pattern is coded by an event of synchronized single spikes occurring after 25–60 ms. These fast associations are performed even at a memory load corresponding to the memory capacity of optimally tuned formal associative networks (>0.1 bit/synapse). With tonic stimulation or feedback loops in the network the neurones fire periodically in the gamma-frequency range (20–80 Hz). With fast changing inputs memory recall can be switched between items within a single gamma cycle. Thus, oscillation is not a primary coding feature necessary for associative memory. However, it accompanies reverberatory feedback providing an improved iterative memory recall completed after a few gamma cycles (60–260 ms). In the bidirectional architecture reverberations do not express in a rigid phase locking between the pools. For small stimulation sets bursting occurred in these cells acting as a supportive mechanism for associative memory. © 2000 Elsevier Science Ltd. Published by Éditions scientifiques et médicales Elsevier SAS

1. Introduction

It is often argued that the creative interaction between experimenters and theorists is an important factor in exploring the working principles of the brain [2, 21, 31, 32, 55, 63, 87, 88]. Computational models can serve as interfaces between biophysical findings and their functional interpretation. Here we will present such an approach to understand the role of temporal structure in neuronal responses with respect to associative memory in cortical circuits.

The nature of spatio-temporal coding in the cortex is still one of the most basic and still unresolved questions in brain research (see e.g. [1] for an overview). There have been suggestions beyond rate coding, stating that information is at least in part represented in the exact timing of single spikes relative to others. Classical examples are the 'temporal correlation hypothesis' by C. von der Malsburg [80] and the 'synfire chain' concept by M. Abeles. Synfire chains are directly based on experimental evidence [2], and also stimulus-related synchronized oscillations in the beta and gamma range (20–80 Hz) have been found in experiments in many cortical areas of various species [19, 20, 29, 30, 64, 65, 86]. Moreover, recent EEG-studies suggest that synchronized gamma-oscillations are involved in cognitive processes, like associative learning and perception [50, 61].

The aim of this paper is to explore the possible function of spike synchronicity, bursting, and oscillations for synaptic associative memory in strongly connected networks of cortical cells. We will develop and study an associative memory model with biophysical compartmental neurones in simulation experiments. The development of the simulation model will be based on results from the literature about more abstracted associative memory models. Associative memory is one of the best understood functions of neural networks.

We first review the basic features of associative neural networks and explain how and under what assumptions the analysis of abstracted associative networks pin down associative memory function in more realistic neuronal networks. In chapter 2 the derived computational function is implemented in a biophysically well tested network of two-compartment neurones. We discuss relations and differences to other models in the literature and formulate the basic questions to be addressed in our model. The simulation experiments are de-

* Correspondence and reprints.

scribed in chapter 3. In chapter 4, we summarize the main observations and results of our simulation experiments and resume with the more general conclusions in chapter 5.

1.1. Neural associative memories

The associative memory theory of the cortex has first been coherently described in informal language by D.O. Hebb [31]. Mathematical models of neural associative networks were first proposed at the turn of the fifties to the sixties to account for an important aspect which perception models of the brain did not reflect, namely the fact that human memory is associative as well as distributed. The working principle of an associative network has three basic features: a) a distributed self-sustaining representation of information as claimed in Rashewsky's reverberation circuit hypothesis [59]; b) synaptic associative learning; and c) associative recall [37]. a) An information entity is represented by a persistent network state, i.e. a spatially distributed activity pattern. b) To store or 'learn' a new information entity, the network activity has to be externally driven to the corresponding activity pattern. Synaptic plasticity, essential to the learning phase, will increase synapses between activated neurones. c) After learning the synaptic structure will favour the appearance of learned activity patterns, which Hebb called 'cell assemblies', and which in the context of associative memory models are often referred to as memory patterns. To recall or retrieve a particular memory pattern one has to stimulate some of its active neurones. Hebb regarded such a pattern completion process as a mechanism for a mental association process.

The first formal associative memory models by Taylor and Steinbuch focused on modelling Pavlovian conditioning, but did already lay out the possibility of an artificial implementation of Hebb's much more comprehensive theory of mental processes [72–74]. Subsequent associative memory studies can be characterized by two different ways how a pattern of dimension n is stored in a synaptic trace. Inspired by holography some works suggested the synaptic trace to be a vector of the same dimension n as the patterns themselves [9, 10, 24, 78]. However, the biologically and technically more impactful correlation matrix models use synaptic traces of dimension n^2 as in the original 'Steinbuch Lernmatrix' [4, 40, 41, 54, 91]. They were the basis of early computational theories of the cerebellum [3, 47], for the hippocampus [48] and for cortical networks [90].

1.2. Results from binary associative network studies

The most extreme abstraction of the behaviour of nerve cells are binary threshold neurones. They sum up the synaptic input and determine their binary output (on or off) by comparing the input sum with a threshold. The binary neurone model was broadly introduced by the work of McCulloch and Pitts [49] and is employed in the most prominent associative memory models, the Willshaw model [91] and the Little/Hopfield model [35, 45]. In the technical literature the computational functions of binary associative networks are often denoted as content addressable memory or matched filter operations. To assess the efficiency of a particular implementation of one of these functions, information theory can be applied on the information channel comprising the storage and retrieval procedure. The capacity of this channel normalized by the number of synapses in the memory network is called the memory capacity [55, 57].

In the Willshaw model, the memory trace of a binary memory pattern is the outer-product matrix and memory traces are superimposed by the logical OR operation. Thus, the resulting synaptic strength structure is binary. Synapses are either potentiated if the coincidence condition was fulfilled in at least one activity configuration, or non-potentiated otherwise. Willshaw's analysis yields a maximum capacity of 0.69 bit per synapse [91]. The analysis is interesting in two regards, see also [56]. First the superimposed storage of distributed representations in the model exploits a substantial part, but not the complete synaptic information content of 1 bit per synapse. Second the capacity maximum is achieved only for very sparse patterns, i.e. if a stored configuration prescribes only activity in a very small portion of the network neurones.

As a biophysical model the retrieval and learning in the Willshaw net is not satisfactory. First, the activity flow during retrieval in the Willshaw model is feed-forward, just as in a logical gate. In contrast, the massive feedback in the wiring schemes known from neuroanatomy suggests iterative update processes during retrieval. Second, the binary synapses in the Willshaw net appear on the first sight as very rough descriptions of real synapses.

Both aspects seem to be better reflected by the Little/Hopfield model with iterative retrieval dynamics and linearly superimposed memory traces leading to integer valued synaptic values. The iter-

ative dynamics results in trajectories in the binary state space that can be characterized by their asymptotic attractor states, which may be fixed point attractors, or periodic as well as chaotic trajectories. Little's study [45] showed that persistent states corresponding to Hebbian assemblies are possible depending on symmetry properties of the interneuronal connectivity. In fact, the coincidence learning rule prescribed by Hebb leads to a symmetric synaptic matrix for the Hopfield and for the Willshaw model. In the Hopfield model, the retrieval dynamics minimizes a simple energy function where under certain conditions the memory states are minima. The capacity analysis of the Hopfield-model yields 0.15 bit per synapse, much less than the Willshaw model [8]. However, as it turned out, this low efficiency has to do with the choice of unbiased memory patterns in the traditional Hopfield model comprising 50% active neurones and 50% inactive ones. In the range of sparse memory patterns, the performance becomes very similar to that of the Willshaw model: The maximum capacity in that limit rises to the same magnitude (0.72 bit per synapse, see [77]). With sparseness also the synaptic structure becomes quite similar to that of the Willshaw model, i.e. most of the synapses just assume the values 0 or 1, and only a few take higher values.

A sparse associative memory provides not only high information efficiency and employs biologically more plausible representations, it also exhibits accelerated recall. The Hopfield model with unbiased patterns takes hundreds of update cycles for memory recall [15], a biologically unrealistic number, given the known time constants in neurones and reaction times in behaviour. The advantage of sparse associative memories in this regard was first not recognized. It cannot be investigated by analysis of infinite sized models since finite size effects become important. For instance, in the original infinite Willshaw model iterations are useless. They do not improve at all the result of the first step [62]. Finite size studies showed that provided some threshold mechanism controls the number of active neurones, iterations improve the pattern completion [26, 28, 33]. With the sparseness of the memories the number of required iterations drops drastically and with sparseness for optimal memory capacity less than three iteration steps suffice [62].

Another objection against the biological realism of the network models discussed so far is their full synaptic connectivity, since in the cortex by far not every neurone pair has a direct connection. However, it was shown even for strongly diluted networks that sparse memory patterns can still be stored and recalled with high information capacity [13]. Also for associative networks employing physiological estimates of neuronal excitability and anatomical estimates of network size and connectivity of a cortical column, sparse memories are still processed with high memory capacity [66, 89].

Thus, the results referred so far suggest the (sparse) feedback Willshaw model as the most appropriate under the abstract associative memory models to describe real neuronal circuits. No matter of the detailed mechanism of superposition of the memory traces from Hebbian coincidence learning, sparse memory patterns provide an efficient and therefore robust use of synaptic memory. Feedback activity flow can improve the recall of stored patterns, though, the number of helpful iteration cycles is low. Synaptic dilution preserves qualitatively the functional properties found in fully connected associative networks.

But what conclusions can be drawn from such a model neglecting the temporal response features of real neurones, and in particular any temporal structure as found experimentally in neuronal responses? In this context, argumentation often was based on a universality hypothesis, 'the' credo of statistical physics proven as justifiable in many different domains. It assumes that in many-particle systems microscopic details have only small effects on the collective behaviour and can therefore be neglected. A basic factor here are statistical limit laws valid in large random systems. There have been attempts to compare results from related theoretical studies with experimental data, see for instance [6, 7]. The correspondence, however, is vague for several reasons. First, cortical neural associative networks might simply not be large and homogeneous enough to be subject to statistical limit laws. Second, electrophysiological measurements at a time are limited to few neurones and cannot access distributed spatial coding. Not even the overall activity in a neuronal network has necessarily to correlate with the activity in single memory patterns. For instance, it has been shown that assuming a realistic connectivity, several memory patterns can be activated and recalled in a cortical column at the same time [66]. Finally, statistical physics often assumes statistical independence between micro states of a system. This, however, becomes questionable for neural systems as soon as one adopts the hypothesis of spike time coding.

In the following we will use the results summarized in this section as a guideline for more realistic associative memory models.

1.3. Associative networks with refined neuronal models

A first step towards more biophysical realism of computational models are neurones with graded responses as they already had been proposed in the earliest works on associative nets by Taylor and Steinbuch [72–74]. One can interpret the continuous output variable as the momentary or short-time averaged spike-firing rate of the neurone. Associative networks employing neurones with sigmoid shaped transfer-function have been extensively analysed. It was shown for auto-associative nets [18, 36, 79] and for bidirectional associative nets [42] that sigmoid neurones allow the same collective computations like two-state threshold neurones. Qualitatively, in terms of the associative memory task this is true, since the attractors for continuous-time updating are still (approximately binary) fixed points. However, it should be noted, that the freedom of setting the slope of the sigmoid can be used to improve the access of stored patterns quantitatively. For a particular retrieval situation the optimal slope can be derived from a Bayesian analysis [67].

The next step towards biophysical faithfulness are neurones producing stereotyped spike-like output pulses. In rate-modulated (RM) neurones the spikes are generated stochastically by a Poisson process with firing probability depending on the input spiking rate within a short past interval. In refractory integrate-and-fire (RIF) neurones the spike generation is also influenced by the input, but in addition by the spiking history of the neurone. A spike is produced if the membrane potential of a cell crosses a threshold, the so-called 'integrate and fire' mechanism. After a spike has been emitted further firing is suppressed within a certain 'refractory' time period. To model jitter of action potential generation as it appears in real neurones, in most RIF models some noise is added to the spike generation process [27, 43, 71, 83].

RM and RIF neurones lead to qualitatively different collective behaviour in otherwise identical network architectures. In large randomly connected networks of RM neurones, the ensemble averaged network activity closely follows that of an equivalent network of graded sigmoid neurones. Up to fluctuations due to the stochastic nature of the spike generation, both networks reveal the same ensemble averaged collective behaviour [5]. In principle also associative networks with RM neurones can be designed to behave virtually identical to the corresponding networks of graded or binary threshold neurones. This can be done in the simplest way by replacing each cell in a network of graded response neurones by a sufficiently large pool of RM neurones (cf. [5]). If the network connectivity is symmetric as usual in auto-associative memories and in bidirectional associative memories, the dynamics then converges to steady states. In contrast, the identical network with RIF neurones typically shows collective oscillations at least below a certain noise level. This is, because these neurones themselves are already intrinsic oscillators, which become synchronized by the mutual connections (e.g. [51]). The result directly generalizes to associative memory networks consisting of RIF-neurones [16, 17, 83]. At high noise levels their behaviour can be essentially reduced to graded response neurone associative memories, but at lower noise they show a more complex spatio-temporal spiking behaviour that critically depends upon the synaptic time delays and the neurone intrinsical refractory mechanism [27, 92].

Because they provide a level of description that at the same time is biologically quite plausible but nonetheless simple enough to allow for large scale simulations of neural systems, refractory integrate-and-fire neurones have been employed frequently in associative network models addressing information processing mechanisms in the cortex as well as the tentative role of spike timing, synchronization and network oscillations (review in [83]). For instance, in primary sensory areas synchronization can support coding of global stimulus features (e.g. [81]). Beside that, it also increases signal-to-noise ratios and thereby detection probabilities. Furthermore, interactions of cell assemblies in and between cortical areas have been addressed in compound networks of one or several associative memories [69, 84, 85]. Here, it turned out that rhythmic associative retrieval is faster than asynchronious retrieval, robust against noise, and provides a high memory capacity. Applied to higher associative areas synchronization further supports the build up of local a well as global cell assemblies (i.e. assemblies distributed over several cortical areas), because the excess of spike coincidences drives Hebbian learning mechanisms more efficiently than asynchronious spike trains. This has been shown in network studies modelling the cortico-hippocampal interplay, in especially, ongoing

learning processes, generalization over stimulus situations, context learning and memory consolidation [11, 12].

The results summarized in the previous sections indicate that associative memory is a possible operational mode in networks where simple two-state neurones are exchanged by spiking neurones. Such networks reveal all computational properties that also appear in associative networks of graded response or RM neurones. With respect to retrieval speed and efficiency, however, they outperform their simpler counterparts, and they show further properties that that should be useful for cortical information processing (i.e. binding by synchronization, increased learning rates and SN-rations due to spike correlations, etc.). Clearly, the role of spike synchronization and mass oscillations in associative memory function can only be studied with RIF networks in the oscillatory mode. In the referred works this was not the central point, or it has not been done systematically. Instead of addressing these questions in RIF neurone associative network, we will go on to networks employing more elaborated and biophysically well tested compartmental neurones.

2. Associative networks with compartmental neurones

2.1. Biophysical modelling

Hodgkin and Huxley introduced a biophysical model for the generation of action potentials based on the voltage dependent ion channels in nerve cell membranes [34]. Models on this level of description have been developed much further during the last years. For example, Traub et al. [76] designed a biophysically faithful nineteen-compartment cable model of a hippocampal pyramidal cell in area CA3 of the guinea pig. Each iso-potential compartment contained up to six ionic conductances that were controlled by ten channel-gating variables. The kinetics of these variables had been fitted to voltage clamp data from CA3 slices and other slice preparations. Conductance densities where chosen consistent with current clamp recordings from soma and dendrites of whole neurones, and from isolated apical dendrites. Meanwhile, very similar models have also been proposed for neocortical cells, although there, anatomical and physiological cell parameters are, of course, slightly different [60]. Nonetheless, the principal dynamical modes of such elaborated neurone models are comparable (cf. e.g. the simulations in [58, 60, 76] for hippocampal and neocortical cells).

Compared to refractory integrate-and-fire neurones, compartmental neurone models clearly show a much richer dynamic behaviour. In Traub et al. [76] and Rhodes and Gray [60], complex types of low-frequency bursting are investigated which are not observable in simple spiking neurone models. The complex types of bursting, however, appear already in simpler neurone models: Pinsky and Rinzel [58] reduced Traub's model heuristically to only two galvanically coupled phenomenological compartments, modelling the soma and the dendrites of a neurone, see *figure 1*. This reduced model reproduces quite faithfully the stimulus-response properties of Traub's model cells, to be more specific, those responses induced by steady somatic and steady dendritic current injection (other input modes were not tested). The model includes realistic synaptic transmission characteristics, i.e. synaptic time constants, reversal potentials and other properties of AMPA and NMDA synapses. The reduced model furthermore has the practical advantage that it is computationally simple enough to be employed in network simulations. In Pinsky and Rinzel [58], a biophysical study was performed using a simulated network of 100 cells, randomly connected with a synaptic density of 20% (2 000 of the $100(100-1)$ connections possible between cell pairs were randomly chosen and assigned to the same positive value). All cells further received a constant inhibitory soma current. If the soma of a single neurone was stimulated by a brief excitatory current, the network responded with synchronized population bursting in the gamma range that persisted for 400 ms or longer, depending on the maximal NMDA conductance. The NMDA synapses hence provided a persistent excitation, but the phase coupling between the bursting cells was provided by fast AMPA mediated currents. Blockade of the AMPA synapses lead to a desynchronization of the bursts. Thus, the simplified Pinsky-Rinzel network reflects a property that has already been demonstrated in slice experiments by Traub et al. [75]: the fast AMPA synapses provide the coupling mechanism responsible for burst and spike synchronization.

2.2. Functional modelling and questions to be addressed

Computational models of associative memories employing compartment neurones and a descrip-

tion of the active membrane properties à la Hodgkin-Huxley have been proposed in several previous studies (for instance, [38, 39, 44]). In these works, the computational function was only defined on the basis of rate coded patterns and it was not asked how the additional repertoire of time coding present in associative networks of this complexity contributes to associative memory function. As already discussed, rate coding might be appropriate if the neural units in the model simulate not single cells but whole cell groups, like cortical columns as proposed by Fransén and Lansner [23]. These studies can barely address questions about the functional role of the temporal fine structure of single cell activity.

The simulation studies presented in the sequel differ to previous studies with respect to the design of the computational model and with respect to the questions they address. We use the biophysically very well confirmed neuronal model developed by Pinsky and Rinzel [58] but assume that their connection weights are formed in a Hebbian learning process. The underlying associative memory task and the simplistic learning procedure are specified using the results from studies on more abstract associative networks revisited in the Section 1. Thus, the model combines much of the known biophysics of single neurones with the functional hypothesis of associative memory. The questions that can be addressed by such a model are in particular:

- What is the function of spike synchronization, bursting and oscillations?
- What is the role of structured feedback connectivity?
- How does the information theoretical performance compare with formal models?

3. Simulation experiments

In this section, we present simulation experiments of small cortical networks comprising bio-

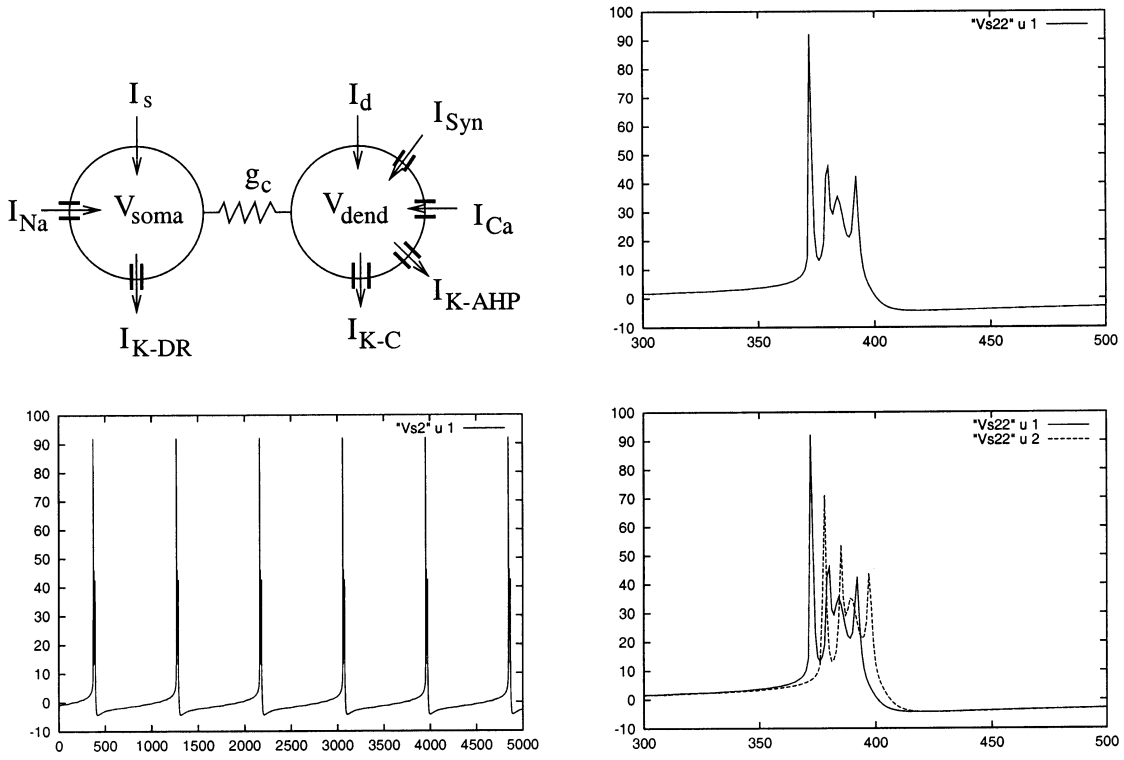

Figure 1. Scheme of a Pinsky-Rinzel neurone (cf. [58]) and plots of its qualitative behaviour. The upper left plot depicts the dendritic and somatic compartments with the different active ion-currents and synaptic inputs. A single neurone is capable of firing repetitive complex bursts (lower left and upper right plot). In a network of synaptically connected cells these bursts synchronize, but not the individual spikes constituting the respective bursts: the lower right plot shows soma potentials of two cells out of at total of 100 neurones. (Scales: time is in 0.1 ms, potentials are in mV).

physical conductance based compartment neurones developed by Pinsky and Rinzel [58]. The computational capabilities of networks with two different architectures are tested: an auto-associative and a bidirectional network.

3.1. The simulation model

The associative memory model used in the following employs the two-compartment neurone model of Pinsky and Rinzel (PR neurone) [58] where the exact dynamic equations and all parameter settings can be found. We employ a network of 100 excitatory cells with parameter settings exactly as in Pinsky and Rinzel [58] but extend it to an associative network by the following modifications: a) the network connectivity is structured assuming common cortical wiring schemes and Hebbian learning; b) the inhibition is not constant but moderates the network activity; and c) we apply external stimulation by current injection to subsets of the network cells.

3.1.1. Structured network connectivity

In the auto-associative wiring scheme described in detail in Section 3.2, the cells were completely interconnected. This reflects a configuration of cells in close vicinity of each other, i.e. within a cortical column. Neuroanatomical studies have estimated a mean density of synaptic contacts of 0.1 between cells with distances lower than the radius of a cortical column [14]. Full connectivity is used since among the roughly 10^5 pyramidal cells in a column, subnetworks will exist with connectivity much higher than the mean. These subnetworks are the most probable candidates to co-operate in a computational function.

In Section 3.3, we examine a bidirectional wiring scheme. Apart from cortical circuits formed by local connectivity, anatomical studies suggest another common cortical wiring scheme providing a strong internal coupling within a set of excitatory cells: a pair of cell groups that project on each other reciprocally. Such wiring schemes can be expected between cell groups of different cortical layers, distant patches in the same area, or between cell groups located in different cortical areas. The latter expectation is motivated by the following anatomical findings [70]: i) the majority of cortico-cortical connections have been reported to be reciprocal [22]; and ii) projections are patchy with patch sizes comparable to the size of cortical columns [46]. We modelled reciprocal connectivity by a wiring scheme where the cells were divided into two groups A and B of fifty cells each that mutually projected onto each other. Within the groups there were no excitatory connections. Each of the cell groups had its independent inhibitory components, acting as in the network of the auto-associative wiring scheme.

For both wiring schemes we assume that connected cell pairs have synapses in both directions. In a local network the spatial vicinity of the cell bodies makes multiple synaptic contacts probable. For the reciprocal circuit between two cell group such a property could be provided by mechanisms like activity dependent guidance of axonal growth during early development. However, the patchwise correspondence of reciprocal connections has not yet been investigated experimentally.

The transmission efficacy of synapses are formed in a Hebbian learning phase preceding the retrieval trials. We used the clipped synaptic modification of the Willshaw model, driven by a set of on/off activity configurations (memory patterns) presented to the network. The configurations were random and overlapping, i.e. each contained a fixed number of active cells and each cell could be active in more than one memory pattern. In the auto-associative scheme we used memory patterns with $k = 10$ active neurones, in the bidirectional scheme the memory pattern pairs had ten active neurones in each of the two cell groups. Excitatory synapses terminated on the dendrite-like compartment and activated AMPA- and NMDA-mediated currents as in Pinsky and Rinzel [58]. Since only ten cells constituted a memory pattern synaptic strengths had to be scaled appropriately such that a single spike evoked a maximum postsynaptic conductance change of 0.09 $mS \cdot cm^{-2}$ for AMPA currents inducing EPSP-amplitudes of roughly 5 mV. NMDA conductances quickly saturated under retrieval conditions [58]. The simple learning in our model allowed the direct comparison of the retrieval efficiency with the theory of the simple and the feedback Willshaw model [57, 62].

3.1.2. Inhibitory system

The role of inhibition to modulate excitatory network activity is based on the assumption that in a local network, disynaptic loops from principal cells to interneurones and back should provide an amount of inhibition that depends roughly proportionally on the total network activity. Such threshold control keeps the network activity from unphysiological states where more than a sparse fraction of cells fire at high rates. As explained in the Section 1 it furthermore improves the efficiency

of associative memory [33, 57, 62, 81]. Technically, in the simulation model, we do not need to implement interneurones individually, but instead assume that action potentials of principal cells evoke not only EPSPs on their target cells, but also — via inhibitory loops — IPSPs on all cells in the network. Accordingly, any spike of a principal cell evokes equally weighted IPSCs into all principal cells. These inhibitory synapses employed a fast GABA-ergic conductance change with reversal potential $V_{CI} = -75$ mV and a shape resembling an alpha function with a maximum conductance change of 0.17 $mS \cdot cm^{-2}$.

3.1.3. External network stimulation

During a retrieval trial a subset of stimulated cells with varying overlap to one of the memory patterns was selected. Since different stored patterns could overlap the stimulation subset had also random overlaps to other memories. For a defined period of time the cells in the stimulation subset received depolarizing dendritic input (modelled by Poisson processes) strong enough to evoke steady firing. To judge the recall of a stored activity configuration, we calculated its transinformation to instantaneous network states (spike/no spike). We normalized by the information content of a stored activity configuration (in our case 36 bits) and called this quantity retrieval quality or simply 'quality' in the remainder. In the bidirectional network we stimulated cells only within one of the cell groups.

3.2. Results for the auto-associative network

We first present simulation results for a single pool of neurones with Hebbian auto-associative connectivity. A set of patterns is stored prior to the shown simulations by means of coincidence learning. During a retrieval experiment a subset of cells with a defined overlap l to one memory pattern received depolarizing dendritic input.

Figure 2 displays spike trains of two cells in the network for $l = 5$ out of a total of $k = 10$ ones per pattern. The upper cell receives direct afferent input, i.e. it is one of the five directly addressed neurones. The second cell receives only input from internal connections. Because it belongs to the addressed memory pattern its synapses to the directly stimulated neurones are potentiated and if these neurones fire the internal synaptic input into the second neurone is strong.

First, note that the spiking rates are in the gamma range and that the cells are synchronized within a time-window of a few milliseconds. This is merely a consequence of the synaptic time-constants of both, the excitatory and inhibitory PSPs. The spikes are phase-locked but the spike of the second cell is delayed due to synaptic integration on the cell membranes. Here, essentially the time-constants of excitatory PSPs on the soma matter. Afterwards cell firing is suppressed as long as the inhibition build up by the excitatory burst itself keeps the neurones below firing threshold. So, the frequency of the collective oscillation in this simulation is basically determined by the relaxation time of inhibitory PSPs and the recurrent excitatory transmission steps (cf. also [86]).

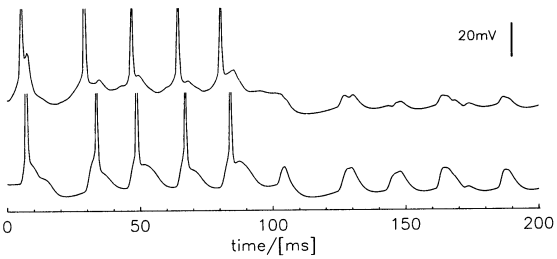

Figure 2. Typical soma-potentials (spikes truncated) for $P = 50$ and $l = 5$. The upper cell receives direct external addressing input, the lower cell belongs to the rest of the addressed memory pattern. At time $t = 95$ ms, stimulation switches to another address.

Half time in the sweep displayed in *figure 2*, at time 100 ms, the external stimulation switches to another stimulus pattern. The new stimulus addresses five neurones of a second, randomly generated memory pattern. The two neurones shown in *figure 2* are not part of this pattern. Notably, the cell responses follow the stimulus withdrawal immediately, that is, there is no afteractivation of the first memory pattern, which would be expressed, for instance, in an ongoing spiking of the neurones (cf. e.g. [38]). Instead, only sub-threshold membrane-oscillations remain, which are caused by cross-talk from the new activated memory pattern: Since the memory patterns have mutual overlap there are potentiated synapses even between neurones not belonging to the same pattern. The membrane potential increase by cross-talk EPSPs is again followed by inhibitory phases. Thus, the rhythmic potential fluctuations in *figure 2* for times larger than 100 ms consist of superimposed excitatory and inhibitory PSPs. During the first 100 ms, these fluctuations are stronger because the neurones belong to the addressed memory pattern; in fact, there they are strong enough to evoke spiking activity in the respective assembly of memory neurones.

This can be seen more clearly in *figure 3*. The lower frame of *figure 3* shows a raster-plot of the soma potentials for all neurones in the network during the same stimulation event as in *figure 2*. Time runs from left to right and the y-axis counts the neurones. Soma potentials are grey-value coded where white denotes high potentials during spikes and black denotes hyperpolarization. Neurones 1, ..., 5 constitute the first address pattern, neurones 1, ..., 10 the corresponding complete memory pattern. Rows 1 and 6 in *figure 3* correspond with the potential traces shown in *figure 2*. Again one can see that directly stimulated cells fire first and the internally driven cells belonging to the same pattern come up a few milliseconds delayed. The time synchronization among the internally driven cells is tighter since the internal connection convergence and divergence averages over the time dispersion within the set of directly stimulated cells.

The retrieval quality is displayed in the upper frame of *figure 3*. The quality plot quantifies a fact already obvious in the raster-plot. The repeated retrieval events yield higher quality than the first completion process just after stimulus onset (at times around $t = 10$ ms and $t = 110$ ms). Presumably this is due to the slower neuronal variables that become better aligned after the first population burst. These first bursts after stimulus switch contain considerably more than the ten ones of the addressed memory patterns, which lowers the retrieval quality. During later retrieval phases adaptation processes in the neurones improve the quality, but also during these periods spurious ones appear occasionally. Thus, the pattern recall is cleaned up with respect of spurious activity with the second and further retrieval phases. Furthermore, one observes that the quality often assumes a sharp maximum in the first part of individual retrieval periods. This means that the correct cells respond somewhat earlier than spurious cells and could therefore, in principle, be segregated in further processing stages (for instance by coincidence detection).

Figure 4 compares the memory capacity of the model achieved with different addresses and varying memory load P. For high P and low numbers of address bits (stimulated neurones), the capacity drops because the few input bits do not suffice to determine the addressed memory pattern uniquely. Hence, the retrieval quality is impaired.

The longer the plateau in the capacity curve, the better is the input fault tolerance of the recall process. Up to a load of $P = 50$, we find a pronounced input fault tolerance and the corresponding memory capacity is close to 0.1 bit per

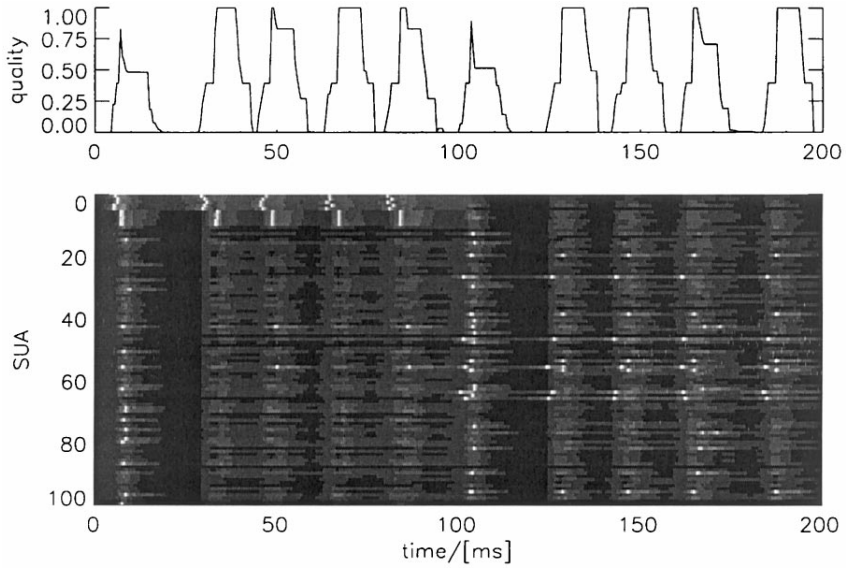

Figure 3. Raster-plot of soma-potentials of all cells for $P = 50$, $k = 10$, and $l = 5$. Neurones 1, ..., 5 constitute the first address pattern, neurones 1, ..., 10 the corresponding memory pattern. Note that action potentials (white dots) are not perfectly synchronized. The spikes scatter over several milliseconds mainly caused by excitatory synaptic transmission: address neurones 1, ..., 5 always fire first and trigger the cells 6, ..., 10. Since for $P = 50$, the memory load is high, retrieval is impaired by cross-talk, i.e. 'quality' < 1. The quality is measured continuously for spikes within the previous 7 ms.

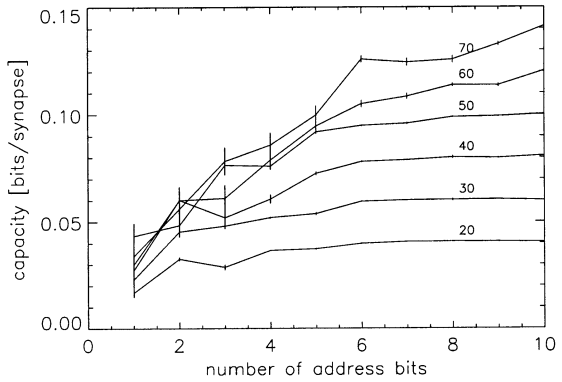

Figure 4. Memory capacities for different numbers of active address neurones and increasing memory load; $P = 20, 30, \ldots, 70$; $l = 1, 2, \ldots, 10$; $N = 100$; $k = 10$. The capacity is derived from the transinformation in spike patterns, averaged over ten retrieval periods for ten randomly selected addresses.

synapse. This is a striking result since for $P = 50$ the synaptic storage has reached the theoretically optimum where 50% of the synapses have been potentiated. The theoretical optimum could even be approached more closely in our model with an optimized memory pattern size k which is lower than $k = 10$ for a network size of $N = 100$ neurones. (Note, that for finite size networks, the theoretical optimum is below the earlier mentioned asymptotic limit of 0.69 bit per synapse. For instance, with a size of 512 cells the optimum is 0.4 bit per synapses, see [57].)

3.3. Results for the bidirectional network

In a second series of simulation experiments, we used a bidirectional network architecture connecting two cell populations A and B and a somewhat different stimulation scheme. We stimulated cells only in cell population A. Unlike the previous experiments in Section 3.2, the stimulation was not permanent but was restricted to a brief period of 25 ms. After that the input was turned off. To test robustness of the retrieval against spurious input we used not only subsets of memory patterns for stimulation but also patterns containing more neurones than the memory pattern (i.e. additional random bits). This test is critical, because in the Willshaw model the retrieval quality is considerably more sensitive against additional activity than against missing activity [62]. To assess retrieval performance we compared only the activity state in the non-addressed cell group B with the respective stored configuration. High quality retrieval in this cell group is obviously impossible with bad retrieval in the directly addressed cell group, since the only input to B consists of the activity in A. The model was symmetrical with respect to the two cell groups. All neurone and network parameters were set to identical values in both groups with the exception of the afferent input, which was only supplied to cell group A.

We conducted experiments using stimulation patterns of different sizes and recorded the network activity over a 500-ms interval after stimulus onset. Typical time courses of soma potentials induced from different stimulation patterns are displayed in *figure 5*.

About 25–40 ms after stimulus onset, the first synchronized wave of induced activity arrived in cell group B. Subsequently, the activity propagated back and forth through the reciprocal connections, influencing the activity configurations of the rhythmic synchronized spike events in both cell groups. The spike frequencies were in the gamma band (30–90 Hz) just as in the experiments in Section 3.2. This is not surprising, since network parameters in the bidirectional model were virtually the same than those chosen for the single pool in the previous section. We observed spike synchronization within each cell group but we did not find a phase locking between the groups. Neurones that did not experience external input showed regular spiking. In contrast, neurones receiving afferent input showed a tendency to burst. This tendency increased with decreasing size of the stimulated population mainly caused by a reduced inhibition. In *figure 5* with small stimulated input sets, populations *5a* and *5b* produce bursting in the first gamma cycles, while larger stimulated populations in *5c* and *5d* do not. This provides a mechanism of activity balance in the secondary layer. In a wide range of stimulation pattern size (we tested stimulations comprising between two and twenty cells receiving direct current injections) the recall quality was high without any adjustments of network parameters. Thus, bursting can enhance the input fault tolerance of association processes by balancing the network activity induced by different stimulation pattern size.

To judge the performance of the fastest possible response (we call this one-step retrieval since only monosynaptic activity propagation to cell group B is involved), we determined the quality maximum in the first wave of activity. To monitor the results provided by iterative activity flow between the cell groups (the bidirectional retrieval mode), we also detected the quality maximum over the whole recording sequence. The following figure shows the

experimental results resolved for different stimulation conditions.

Diagrams a and c in *figure 6* display the measured quality values. A quality equal to one corresponds to perfect retrieval. At the memory load examined, it translates to a synaptic storage capacity of $0.295/2 \approx 0.148$ bit/synapse. The divisor of two is due to the fact that bidirectional transmission requires two biological synapses.

Diagram a compares the first quality maximum with the performance of the Willshaw model (a feed-forward network with binary synapses) at the same memory load. The curve is computed for constant activity threshold setting using the theory from Sommer and Palm [68]. The fact that the quality curves stay somewhat below one indicates that the load in both networks is near the maximum, cross-talk is already setting in, even in associations with perfect address. By the additional noise in the biological model its maximum capacity is decreased by 10% to $0.27/2 = 0.135$ bit/synapse. Also the fault tolerant association capability is impaired, only slightly for lower, but substantially for higher input activities. The question underlying the measurements displayed in diagram b) is whether bidirectional activation cycles in the network can improve retrieval quality. In fact, for very small as well as in the entire range of high input activity the quality maximum is always achieved by iterative retrieval. For very small ($l = 2.3$) and for large input activities ($12 < l \leq 20$) iterative retrieval improved the retrieval result in between 70 and 80% of the simulation runs.

The diagrams c) and d) compare the quality and retrieval time of one-step and iterative retrieval in the Pinsky-Rinzel neurone network. The iterative retrieval performs better in all cases. Most significantly, the fault tolerance with respect to higher input activity can be significantly improved by iteration. Of course, the required retrieval time increases from 25–60 to 60–260 ms depending on the input condition.

4. Summary

In our model, in cells with strong postsynaptic input the AMPA-mediated part of the EPSPs causes the synchronized spike events via selective associative connections. Thus, if the set of cells

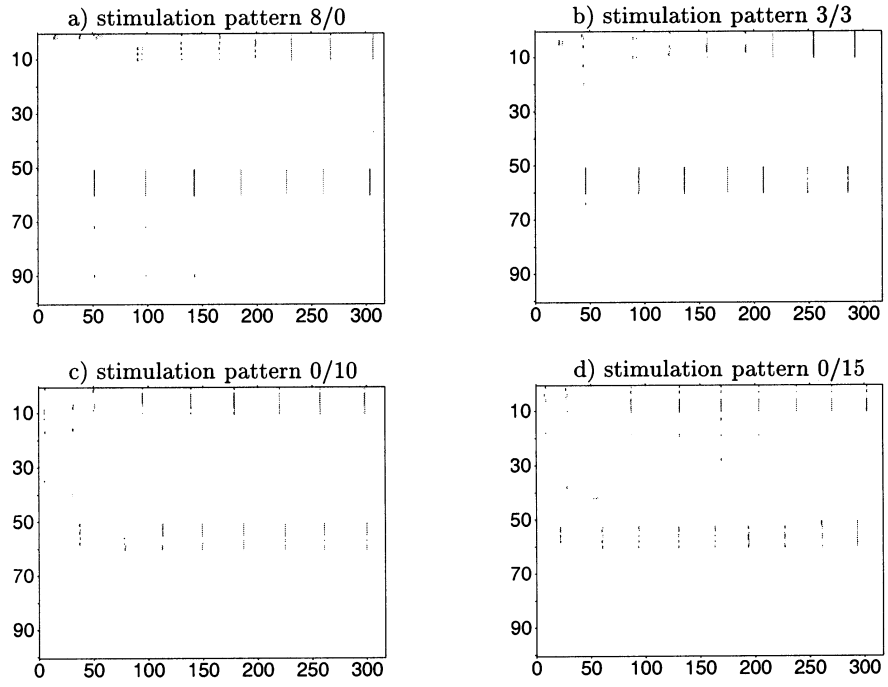

Figure 5. Raster-plots of soma potentials during single retrieval trials using different stimulation patterns. The y-axes display the cell numbers, the x-axes the time, as in *figure 3*. Here, cells 1 to 50 form pool A and cells 51 to 100 pool B. The different stimulation patterns are described by a tuple x/y, where x is the number of cells in the memory pattern that received no stimulation and y the number of stimulated cells outside the memory pattern.

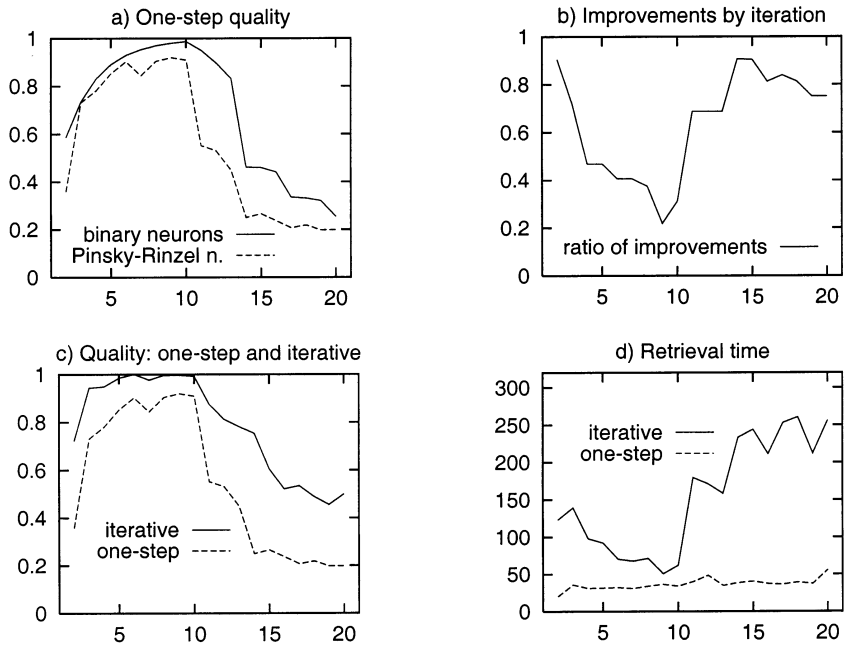

Figure 6. Experimental results. The x-axes of the diagrams label the input activity, i.e. the number of cells that receive stimulation currents. At an input activity of ten the stimulation pattern was identical to one of the stored patterns, at lower or higher activities it deviated by miss or add errors, respectively. For every input condition we tested all of the memory patterns and averaged the measured quantities. In addition we sampled over 32 runs with different sets of memory patterns.

externally stimulated has enough overlap with a stored memory, the pattern of induced synchronized spike events resembles the memory pattern quite closely. One finds only a few milliseconds of time lag between spike events in different cells belonging to one memory. This suggests to consider such a synchronized population spike as an elementary association or retrieval process. The larger the set of spiking cells in the network the stronger inhibition builds up until it finally suppresses further spiking. Nonetheless, if afferent excitation persists or if there are selective excitatory feedback connections, after decay of the inhibition another population burst appears due to the slow dynamic mechanisms. We observe this rhythmic activity caused by the interaction between excitatory and inhibitory cells to fall in the gamma-frequency range (20–80 Hz), just as in the random connectivity network where inhibition was constant [58]. All our experiments demonstrate that biologically realistic neural associative networks store and retrieve sparse patterns with almost the same memory capacity as formal associative memories. We chose an auto-associative and a bidirectional network architecture for closer inspection by simulation experiments.

The auto-associative scheme was chosen to study a local cortical circuit receiving tonic afferent stimulation. A variable number of cells in a memory receive steady but noisy current injections (supplied by Poissonian input spike processes). The first synchronized spike event provides already a reasonable estimation of a memory pattern, but contains some erroneous responses of non-addressed cells. The second synchronized spike event, i.e. the second gamma cycle after stimulus onset, provides the memory with increased quality. The improvement is due to the alignment of the slow neuronal variables (neuronal adaptation) that requires more than one gamma cycle. With the input strength used, the quality in the cycles after the second stays almost constant and the time lag between primary and secondary driven cells is almost the same in all cycles. This implies that the internal wiring provides a feed-forward like completion of the addressed memory pattern starting from the directly driven cells. The internal feedback plays no other significant role. If the stimulus is changed to another spatial pattern the network switches within one gamma cycle to the new pattern. This suggests that tonic stimulation over more than two gamma cycles cannot evoke addi-

tional internal computational mechanisms. On the other hand, the maximum readout speed is just limited by the gamma rhythm: with fast input switching one could reach one memory pattern every cycle, though, not with maximum quality.

The bidirectional wiring scheme addressed the possible computational processes in reciprocally connected cortical structures. Cells only in one group received noisy current injections. Unlike in the previous experiment, the current injections stopped after a short time period (25 ms), and the set of stimulated cells was not a subset of one memory pattern. The latter was to test the robustness of the associative memory function not only against missing but also against spurious activity. Since the stimulation was switched off, the spike events following the first response reflect feedback over the reciprocal connections. We evaluated retrieval quality in the first response finished after 25–40 ms, and by iterative recall. The first response realizes unidirectional retrieval quite similar to the Willshaw model. Unsurprisingly, the additional noise sources impair the performance of the biological model and the performance drops below that of the Willshaw model. This impairment is mild for error-free addressing and small stimulation sets. In the latter case bursting of directly stimulated cells turned out as a mechanism to enhance the performance. A small number of stimulated cells evokes only a relatively low net inhibition that allow the cells to burst. This compensates for the small number of stimulated cells. The performance of the first response drops more drastically for retrieval trials with spurious input activity. This is a biologically relevant case since spontaneous stochastic activity of cortical cells, as well as interferences from other associations in the same network, increase the network activity. Nonetheless, the simulations show that the retrieval is robust against a considerable amount of background noise provided this way. Iterated retrieval turned out as an effective means to improve first response retrieval, in particular, in the regime of imperfect stimulation. In this regime the quality of first response retrieval was suboptimal in 70 to 80% of the performed retrieval trials but iterated retrieval in an individual trial was able to correct most of the appearing errors. In these simulations, we observed no rigid phase coupling between the pools. Apparently, the AMPA mediated fast PSP component was not strong enough for a phase coupling of the pools, the NMDA component was too slow, and the separate inhibitory systems of the two pools also did not provide means for synchronization. Overall, our experiments suggest that two different modes of retrieval are possible: a quick unidirectional, and a more precise iterative mode. Experimentally they should be discernible by latency measurements: In our model, the first response required from 25–60 ms and iterative retrieval lasted 60–260 ms, up to ten gamma cycles.

5. Discussion and conclusions

This paper presents a novel modelling approach to reveal the function of the spatial and temporal structure of neuronal activity, in particular, spikes, synchronization and oscillations in the context of cortical associative memory. We propose associative network models for small groups of cortical cells employing the quite faithful biophysical description of neurones and synapses by Pinsky and Rinzel [58] and the rather simplified version of Hebbian synaptic learning of the Willshaw model [91]. This model choice as well as the specification of the computational task with sparse memory patterns are established on results from associative memory studies using neurones with stereotyped outputs summarized in the Section 1.

The aims of this associative memory study are to understand the functional role of spike synchronization and gamma-oscillations, and to compare the efficiency of a biologically realistic network model with a simple formal model. It has to be emphasized that associative memory and gamma rhythms observed in the simulations are not a surprise but a result of the functional and biophysical ingredients in our model. The biophysical ingredients, however, are borrowed from well established models of real neurones.

In the light of our model, a synchronized spike event indicates a fast associative retrieval process. Tonic input evoked sequences of repetitive associative processes in the gamma-frequency band. Neither the frequency of the repetitions nor the periodicity of the processes at all are crucial for a proper functioning of the network. Nonetheless, fast repetition times in principle allow for fast switches between different memory patterns. By fast switching between stimuli the readout speed can be increased up to the point where every one or two gamma cycles a new pattern is elicited. In this case spike trains of individual cells show no or only very short phases of gamma-activity.

Short input flashes followed by silence evoked gamma spike trains in single cells with a functional

meaning: They correspond to reverberation processes that can improve the retrieval quality, in particular, for noisy input patterns. This is consistent with Amit [7], where reverberations in local networks in IT have been used to explain electrophysiological data recorded during delayed matched to sample tasks.

Our experiments further revealed that bursting of stimulated cells might play a role in activity control. When only relatively few cells in the network are externally stimulated the overall level of depolarisation is low and cells show an increased tendency to burst. These bursts can in part account for the reduced input activity such that the addressed cells that do not receive external input can still reach firing threshold. In some sense this normalizes the total activity in the set of externally addressed neurones which enables a proper network response for a large range of address pattern sizes.

This study compares quantitatively the information theoretical efficiency between a formal neural network, the finite Willshaw model, and a same sized neural network employing neurones with detailed biophysical properties. As it turns out the efficiencies are of comparable order of magnitude. With iterative retrieval over a few gamma cycles the biological associative network outperforms the Willshaw model. Our study demonstrates that sparse associative memory tasks can be efficiently and fast performed in circuits of biological realistic neurones. Of course, our study cannot not exclude, that also associative memories more efficient than the simple feedback Willshaw model could be realized biologically, for instance, crosswise bidirectional retrieval [68], or even efficient non-sparse associative memories like those with non-local learning [25] or with non-monotonous response neurones [52, 53].

The behaviour of the studied model turned out to be robust against different kinds of noise, too. The simulation experiments included noise in the external current injections, variations in the properties of individual pyramidal cells (cf. [58]), crosstalk due to overlapping memory patterns, and spurious external input (i.e. missing and spurious bits in address patterns). Clearly, the stronger these noise sources, the smaller the obtainable memory capacity will be, but this applies to any associative memory model. If noise influences are increased to an extent that synchronization is lost we are back in the simpler regime that is already well described by graded neuronal associative memories. Such networks lack the ability of fast retrieval described in the present work.

The synchronous single-spike event coding of the retrieved patterns in our model is in contrast to coding by adjusting phases of periodically firing neurones as proposed to interpret gamma-oscillations by feature coding [19, 65, 86].

The interpretation of gamma-oscillations as sequences of fast individual retrieval processes carried by associative excitatory connections and rhythmically interrupted by inhibitory interneurones has been already proposed based on simulation experiments in simpler models [81–83, 85]. Nevertheless, the phases of gamma-rhythmic reverberations of memory patterns in different cortical networks could be influenced by additional cortical mechanisms to code binding or separation of memory items.

Acknowledgements

The authors want to express their gratefulness for the severe criticism of one referee leading to a substantial improvement and straightening of the line of discussion in this manuscript.

References

[1] Abbott L., in: Abbott L., Sejnowski T. (Eds.), Neural Codes and Distributed Representations, MIT Press, Cambridge, MA, 1999.

[2] Abeles M., Corticonics: Neural Circuits of the Cerebral Cortex, Cambridge University Press, Cambridge, UK, 1991.

[3] Albus J.S., A theory of cerebellar function, Math. Biosci. 10 (1971) 25–61.

[4] Amari S.-I., Learning patterns and pattern sequences by self-organizations of threshold elements, IEEE Trans, Comput. C-21 (1972) 1197–1206.

[5] Amari S.I., A mathematical foundation for statistical neurodynamics, SIAM J. Appl. Math. 33 (1977) 95–126.

[6] Amit D.J., Modelling Brain Function, Cambridge University Press, Cambridge, 1989.

[7] Amit D.J., The Hebbian paradigm reintegrated: local reverberation internal representations, Behav. Brain Sci. 18 (1995) 617–657.

[8] Amit D.J., Gutfreund H., Sompolinsky H., Statistical mechanics of neural networks near saturation, Ann. Phys. 173 (1987) 30–67.

[9] Anderson J.A., A memory model utilizing spatial correlation functions, Kybernetik 5 (1968) 113–119.

[10] Anderson J.A., A simple neural network generating an interactive memory, Math. Biosci. 14 (1972) 197–220.

[11] Bibbig A., Wennekers T., Palm G., A neural network model of the cortico-hippocampal interplay and the representation of contexts, Behav. Brain Res. 66 (1995) 169–175.

[12] Bibbig A., Wennekers T., Palm G., A neural network model of the cortico-hippocampal interplay: contexts and generalization, Eng. Applicat. Artif. Intelligence 9 (1996) 145–151.

[13] Bosch H., Kurfess F.J., Information storage capacity of incompletely connected associative memories, Neural Networks 11 (1998) 869–876.

[14] Braitenberg V., Schüz A., The Anatomy of the Cortex, Statistics and Geometry, Springer, Berlin, Heidelberg, New York, 1991.

[15] Bruck J., On the convergence properties of the Hopfield model, IEEE Trans. Neural Networks 7 (1990) 1579–1585.

[16] Buhmann J., Schulten K., Associative recognition and storage in a model network of physiological neurons, Biol. Cybern. 54 (1986) 319–335.

[17] Choi M.Y., Dynamic model of neural networks, Phys. Rev. Lett. 61 (1988) 2809–2812.

[18] Cohen M.A., Grossberg S., Absolute stability of global pattern formation and parallel memory storage by competitive neural networks, IEEE Trans. Sys. Man Cybern. 13 (1983) 815–826.

[19] Eckhorn R., Neural mechanisms of scene segmentation: recordings from the visual cortex suggest basic circuits for linking field models, JEEE Trans. Neural Networks 10 (1999) 464–479.

[20] Eckhorn R., Bauer R., Jordan W., Brosch M., Kruse W., Munk M., Reitboeck H.J., Coherent oscillations: a mechanism of feature linking in the visual cortex?, Biol. Cybern. 60 (1988) 121–130.

[21] Edelman G.M., Bright Air, Brilliant Fire: On the Matter of the Mind, Basic Books, New York, 1992.

[22] Felleman D.J., van Essen D.C., Distributed hierarchical processing in the primate cerebral cortex, Cerebral Cortex 1 (1991) 1–47.

[23] Fransén E., Lansner A., A model of cortical associative memory based on a horizontal network of connected columns, Network 9 (1998) 235–264.

[24] Gabor D., Associative holographic memories, IBM J. Res. Dev. 13 (1969) 156–159.

[25] Gardner E., Maximum storage capacity in neural networks, Europhys. Lett. 4 (1987) 481–485.

[26] Gardner-Medwin A., The recall of events through the learning of associations between their parts, Proc. R. Soc. Lond. B 194 (1976) 375–402.

[27] Gerstner W., van Hemmen J.L., Associative memory in a network of 'spiking' neurons, Network 3 (1992) 139–164.

[28] Gibson W.G., Robinson J., Statistical analysis of the dynamics of a sparse associative memory, Neural Networks 5 (1992) 645–661.

[29] Gray C.M., Synchronous oscillations in neuronal systems: mechanisms and functions, J. Comput. Neurosci. 1 (1994) 11–38.

[30] Gray C.M., König P., Engel A.K., Singer W., Oscillatory responses in cat visual cortex exhibit intercolumnar synchronization which reflects global stimulus properties, Nature 338 (1989) 334–337.

[31] Hebb D.O., The Organization of Behaviour, Wiley, New York, 1949.

[32] Hebb D.O., Essay on Mind, Erlbaum, New Jersey, 1980.

[33] Hirase H., Recce M., A search for the optimal thresholding sequence in an associative memory, Network 7 (1996) 741–756.

[34] Hodgkin A.L., Huxley A.F., A quantitative description of membrane current and its application to conduction and excitation in nerve, J. Physiol. 117 (1996) 500–544.

[35] Hopfield J.J., Neural networks and physical systems with emergent collective computational abilities, Proc. Natl. Acad. Sci. USA 79 (1982) 2554–2558.

[36] Hopfield J.J., Neural networks with graded responses have collective computational properties like those of two-state neurons, Proc. Natl. Acad. Sci. USA 81 (1984) 3088–3092.

[37] James W., Briefer Psychology, Harvard University Press, Cambridge, MA, 1890.

[38] Jensen O., Idiart A.P., Lisman J.E., Physiologically realistic formation of autoassociative memory in networks with theta ma oscillations: role of fast NMDA channels, Learning Mem. 3 (1996) 243–256.

[39] Jensen O., Lisman J.E., Theta/gamma networks with slow NMDA channels learn sequences and encode episodic memory: role of NMDA channels in recall, Learning Memory 3 (1996) 264–278.

[40] Kohonen T., Correlation matrix memories, IEEE Trans, Comput. C-21 (1972) 353–359.

[41] Kohonen T., An adaptive associative memory principle, IEEE Trans. Complex Syst. C-23 (1974) 444–445.

[42] Kosko B., Bidirectional associative memory, IEEE Trans. SMC 18 (1988) 49–60.

[43] Lankheet M., Molenaar J., van de Grind W., The spike generating mechanism of retinal ganglion cells, Vision Res. 29 (1989) 505–517.

[44] Lansner A., Fransén E., Modelling Hebbian cell assemblies comprised of cortical neurons, Network 3 (1992) 105–119.

[45] Little W.A., The existence of persistent states in the brain, Math. Biosci. 19 (1974) 101–120.

[46] Malach R., Cortical columns as devices for maximizing neuronal diversity, Trends Neurosci. 3 (1994) 101–104.

[47] Marr D., A theory of cerebellar cortex, J. Physiol. 202 (1969) 437–470.

[48] Marr D., Simple memory: a theory for archicortex, Phil. Trans. R. Soc. Lond. B 262 (1971) 23–81.

[49] McCulloch W.S., Pitts W., A logical calculus of the ideas immanent in nervous activity, Bull. Mathe. Biophys. 5 (1943) 115–137.

[50] Miltner W., Braun C., Arnold M., Witte H., Taube E., Coherence of gamma-band eeg activity as a basis for associative learning, Nature 397 (1999) 434–436.

[51] Mirollo R.E., Strogatz S.H., Synchronization of pulse-coupled biological oscillators, Nature 397 (1999) 434–436.

[52] Morita M., Associative memory with nonmonotone neural networks, Neural Networks 6 (1993) 115–126.

[53] Morita M., Memory and learning of sequential patterns by nonmonotone neural networks, Neural Networks 9 (1996) 1477–1489.

[54] Nakano K., Associatron-a model of associative memory, IEEE Trans. Syst. Man Cybern. SMC 2 (1972) 380–388.

[55] Palm G., Neural Assemblies, Springer, Berlin, 1982.

[56] Palm G., On the information storage capacity of local learning rules, Neural Comput. (1992).

[57] Palm G., Sommer F.T., Associative data storage and retrieval in neural networks, in: Domany E., van Hemmen J.L. (Eds.), Models of Neural Networks III, Springer, New York, 1995, pp. 79–118.

[58] Pinsky P.F., Rinzel J., Intrinsic and network rhythmogenesis in a reduced Traub model for CA3 neurons, J. Comput. Neurosci. 1 (1994) 39–60.

[59] Rashewsky N., Mathematical Biophysics, University of Chicago Press, Chicago, 1938.

[60] Rhodes P.A., Gray C.M., Simulations of intrinsically bursting neocortical pyramidal neurons, Neural Comput. 6 (1994) 1086–1110.

[61] Rodriguez E., George N., Lachaux J.P., Martinerie J., Renault B., Varela F.J., Perception's shadow: long-distance synchronisation of human brain activity, Nature 397 (1999) 430–433.

[62] Schwenker F., Sommer F.T., Palm G., Iterative retrieval of sparsely coded associative memory patterns, Neural Networks 9 (1996) 445–455.

[63] Sejnowski T.J., Koch C., Churchland P.S., Computational neuroscience, Science 241 (1988) 1299–1306.

[64] Singer W., Synchronization of cortical activity and its putative role in information processing and learning, Ann. Rev. Physiol. 55 (1993) 349–374.

[65] Singer W., Gray C.M., Visual feature integration and the temporal correlation hypotheses, Annu. Rev. Neurosci. 18 (1995) 555–568.

[66] Sommer F.T., On cell assemblies in a cortical column, Neurocomputating 32-33 (2000) 517–522.

[67] Sommer F.T., Dayan P., Bayesian retrieval in associative memories with storage errors, IEEE Trans. Neural Networks 9 (1998) 705–713.

[68] Sommer F.T., Palm G., Improved bidirectional retrieval of sparse patterns stored by Hebbian learning, Neural Networks 12 (1999) 281–297.

[69] Sommer F.T., Wennekers T., Associative memory in a pair of cortical cell groups with reciprocal connections, Neurocomputating (2000) (in press).

[70] Sommer F.T., Wennekers T., Palm G., Bidirectional completion of cell assemblies in the cortex, in: Computational Neuroscience: Trends in Research, Plenum Press, 1998.

[71] Stein R., Some models of neuronal variability, Biophys. J. 7 (1967) 37–68.

[72] Steinbuch K., Die Lernmatrix, Kybernetik (Biol. Cybern.) 1 (1961) 36–45.

[73] Taylor W.K., Electrical simulation of some nervous system functional activities, in: Information Theory, vol. 3, Butterworths, 1956, pp. 314–328.

[74] Taylor W.K., Cortico-thalamic organization and memory, Proc. R. Soc. B 159 (1964) 466–478.

[75] Traub R.D., Wong R.K.S., Miles R., Synaptic and intrinsic conductances shape picrotoxin-induced synchronized after discharges in the guinea-pig hippocampal slice, J. Physiol. 93 (1993) 525–547.

[76] Traub R.D., Wong R.K.S., Miles R., Michelson H., A model of a CA3 hippocampal pyramidal neuron incorporating voltage-clamp data on intrinsic conductances, J. Neurophysiol. 66 (1991) 635–650.

[77] Tsodyks M.V., Feigelman M.V., The enhanced storage capacity in neural networks with low activity level, Europhys. Lett. 6 (1988) 101–105.

[78] van Heerden P.J., A new optical method of storing and retrieving information, Appl. Optics 2 (1963) 387–392.

[79] van Hemmen J.L., Kühn R., Nonlinear neural networks, Phys. Rev. Lett. 57 (1986) 563–580.

[80] von der Malsburg C., The correlation theory of brain function, Internal Report 81-2, Max-Planck Institut für Biophysikalische Chemie, Göttingen, 1981.

[81] Wennekers T., Palm G., On the relation between neural modeling and experimental neuroscience, Theory Biosciences 116 (1997) 273–289.

[82] Wennekers T., Palm G., How imprecise is neuronal synchronization?, Neurocomputing 26-27 (1999) 579–585.

[83] Wennekers T., Palm G., Cell assemblies, associative memory and temporal structure in brain signals, in: Miller R. (Ed.), Time and the Brain. Conceptual Advances in Brain Research, vol. 2, Harwood Academic Publishers, 2000.

[84] Wennekers T., Sommer F.T., Gamma-oscillations support optimal retrieval in associative memories of two-compartment neurons, Neurocomputing 26-27 (1999) 573–578.

[85] Wennekers T., Sommer F.T., Palm G., Iterative retrieval in associative memories by threshold control of different neural models, in: Supercomputers in Brain Research, World Scientific, 1995, pp. 301–319.

[86] Whittington M.A., Traub R.D., Jefferys J.G.R., Synchronized oscillations in interneuron networks driven by metabotropic glutamate receptor activation, Nature 373 (1995) 612–615.

[87] Wickelgren W.A., Context-Sensitive Coding, Associative Memory, and Serial Order in (Speech) Behavior, Prentice-Hall, Englewood Cliffs, NJ, 1977.

[88] Wickelgren W.A., Webs, cell assemblies, and chunking in neural nets, Concepts Neurosci. (1992) 1-53.

[89] Wickens J.R., Miller R., A formalism of the neural assembly concept. 1. Constraints on neural assembly size, Biol. Cybern. 77 (1997) 351–358.

[90] Wigström H., A neuron model with learning capability and its relation to mechanisms of association, Biol. Cybern. 12 (1973) 204–215.

[91] Willshaw D.J., Buneman O.P., Longuet-Higgins H.C., Non-holographic associative memory, Nature 222 (1969) 960–962.

[92] Yoshioka M., Shiino M., Associative memory based on synchronized firing of spiking neurons with time-delayed interactions, Phys. Rev. E 58 (1998) 3628–3639.