Biol. Cybern.	54.	319-335	(1986)
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Associative Recognition and Storage in a Model Network of Physiological Neurons

J. Buhmann and K. Schulten

Physikdepartment, Technische Universität München, James-Franck-Strasse, D-8046 Garching, Federal Republic of Germany

Abstract. We consider a neural network model in which the single neurons are chosen to closely resemble known physiological properties. The neurons are assumed to be linked by synapses which change their strength according to Hebbian rules on a short time scale (100 ms). The dynamics of the network - the time evolution of the cell potentials and the synapses - is investigated by computer simulation. As in more abstract network models (Cooper 1973; Hopfield 1982; Kohonen 1984) it is found that the local dynamics of the cell potentials and the synaptic strengths result in global cooperative properties of the network and enable the network to process an incoming flux of information and to learn and store patterns associatively. A trained net can associate missing details of a pattern, can correct wrong details and can suppress noise in a pattern. The network can further abstract the prototype from a series of patterns with variations. A suitable coupling constant connecting the dynamics of the cell potentials with the synaptic strengths is derived by a mean field approximation. This coupling constant controls the neural sensitivity and thereby avoids both extremes of the network state, the state of permanent inactivity and the state of epileptic hyperactivity.

1 Introduction

Recent research in brain modelling and neural network theory follows two main paths. The followers of one path seek to describe the circuits of small neural assemblies and their system theoretical tasks. Stent and coworkers (Stent et al. 1978) have simulated the swimming movement of the leech identifying the oscillatory and feedback mechanisms in the leech neural network. Kandel and Schwartz (1982) have investigated the changes of the plastic synapses in Aplysia, a marine mollusk, and interprete this epcial mechanism as learning.

The followers of the second path describe the information processing properties of neural networks by simulating a large number of formal neurons which do not resemble closely physiological neurons but rather physical spins with two or more internal states. Models of this kind introduced by Little (1974) and by Hopfield (1982, 1984) and the more involved model of Edelman (1982) propose simplifications of the single neuron behavior arguing that the essential properties of information processing systems which lie in their global behavior are reproduced well. Details of the neuroanatomy and neuronal functions, e.g. the synaptic connectivity and the cell potentials, are described in a rather abstract fashion. The simple behavior of the network constituents allows to simulate neural assemblies containing several thousands of formal neurons. If also symmetric synaptic interactions are chosen this formal neuronal system is equivalent to a spin glass and the concepts of statistical mechanics can be applied to understand the macroscopic behavior of such systems (Peretto 1984).

The question arises however, in how far this behavior of formal neuronal systems also arises if the constituents and their dynamic properties are described in a more realistic fashion. We want to investigate in this article the information processing properties of a neural network composed of "physiological" neurons. The neurons are interconnected by synapses, communicate among each other and obtain input from a primary set of neurons, the receptors. Physiological properties like the firing rate of a single neuron are reproduced by the millisecond dynamics of the cells. The synapses between pairs of neurons possess plasticity and change their strengths on a time scale of 300 ms according to the synchronicity of the activity of the two neurons involved. This approach as advocated by v. d. Malsburg (v. d. Malsburg 1981, 1985; v. d. Malsburg and Bienenstock 1985) allows to account for the formation of short term memory traces.

Initially, the synaptic strengths are chosen at random resembling a completely uninstructed state of the network. With this model based as closely as possible on physiological laws we hope to avoid model artefacts. We like to show that such physiological neural networks are capable of autoassociative memory and recognition.

The self-instruction (learning) of the network is simulated in three stages. In a first stage the receptors activate the network with the pattern to be learned. Synchronous activities of pre- and postsynaptic neurons in the network strengthen the excitatory synapses according to the Hebbian rule (Hebb 1949; Palm 1982) and promote a cooperative interaction between neurons belonging to those features of the pattern which are simultaneously excited in the receptors. This stage of initial learning lasts for 300 ms. Afterwards, the receptors are quiescent for 20 ms and the network relaxes to the unexcited state. This second stage is then followed by a third period which examines the success of the self-instruction of the network by requiring different associative tasks. For this purpose the receptors are made to present to the network, for example, the input of the first learning stage, albeit with some features missing. The self-instructed network has to demonstrate that it can restore these features.

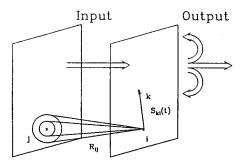
In another course of self-instruction the receptors present to the network an initial input pattern with strong noise of asynchronous activity superimposed. The network has to demonstrate its ability to filter out this noise and to restore the pattern.

In a third course of self-instruction the network abstracts a prototype pattern from a set of patterns differing in details from one another.

2 Initial Synapses in the Model Network

The neural system discussed in this paper is a 2-dimensional network of "physiological" neurons arranged on a rectangular lattice. Each nerve cell receives input from a primary set of receptors. These receptors may be interpreted either as sensory cells which receive information about the physical world or as a more peripheral set of neurons in the cerebral cortex which collects and processes patterns and projects them onto another area, our network, for further processing. In the latter case the receptors may also change the strengths R_{ij} of their synaptic connections to the network, but in our model we neglect this possibility. The receptors are the input devices which present the learning and test patterns without changing their own properties.

Figure 1 schematically represents our model and the flux of information leading from the receptors to



Receptors Neurons

Fig. 1. Schematic presentation of the model investigated: Receptors send spikes to a network of neurons. The connectivity between the receptors (labelled by j) and neurons (labelled by i, k) is given by the matrix R_{ij} , the connectivity between the neurons is given by $S_{ik}(t)$. The resulting activity of the neural network is affected by an activity-dependent alteration of $S_{ik}(t)$, i.e. the network experiences a feed-back as indicated. As a result, the relationship between the input of the neural network — the receptor activity — and the output — the neural activity — is highly non-linear

the neuronal net. The activity of the neurons is interpreted as the output of the network. The backward bended arrows indicate the feed-back due to the effect of the neuron activity on the synaptic strengths $S_{\mathbf{n}}(t)$ between neuron k and i in the neural network.

The connections between the receptors and the "physiological" neurons have a local center-surroundorganization with peak value R. Receptors j which are lying in the neighbourhood of the receptor i are connected with the neuron i by excitatory synapses, whereas receptors arranged in the immediate surrounding of this excitatory center have an inhibitory effect on neuron i. Indices labelling the neurons and the receptors shall always be read as double indices for the cell position (x, y). The area on the receptor set which affects the neuron i is much smaller than the size of the network. Therefore, the connections between the receptors and the neurons constitute a continuous projection of the input pattern onto the neural net, the projection being locally convoluted with the centersurround function.

In Fig. 2 the connection strengths between the receptors j and the neuron i are shown. The connections between receptors and neurons support activity areas with a diameter L_e and suppress the longer ranged noise which is part of the background.

The synapses $S_{ik}(t)$ which carry action potentials from cell k to cell i are initially chosen at random, i.e. $S_{ik}(0) = \pm S$ with equal probability for an excitatory or

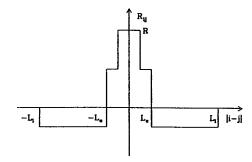


Fig. 2. The strengths of the receptor-neuron connections have a center-surround organisation. The synaptic strength R_{ij} between receptor j and neuron i depends, as indicated, on the distance between the position of i and j, i.e. on |i-j|, in the case of a 1-dim.

an inhibitory synapse. Hence, the network is initially completely uninstructed, and no memory traces can be found in the network at the beginning of the learning stage. The neurons in our model possess excitatory and inhibitory synapses. This feature differs from the properties of physiological neurons which can either excite or inhibit other neurons, not both. We decided to offend Dale's law (Dale 1935) for the sake of computational simplification. We expect that for large populations of neurons the error introduced is not significant.

The ratio R/S between the peak value of the initial receptor-neuron connection and the absolute value of the neuron-neuron synapses determines the coupling of the network to the set of receptors. If $R/S \gg 1$ the network is very strongly coupled to the receptors, whereas in the case $R/S \approx 1$ the network dynamics is dominated by the synaptic communication between the neurons.

3 Dynamics of the Membrane Potential

The fast dynamics of a neuron involves its membrane potential which changes on the time scale of a few milliseconds. The membrane potential characterizes the state of the neuron, specifies the sensitivity of the cell and its ability to fire an action potential in the next millisecond. A membrane potential considerably below the resting value indicates that the neuron is strongly inhibited by other neurons in the network. Conversely, a neuron with its membrane potential above the resting potential is sensitive to the incoming flux of action potentials and, if further excited, will reach the threshold.

In our model two important contributions to the dynamics of the potential are included. A first term describes the relaxation of the membrane potential to the resting potential. This relaxation is due to the Ohmic resistance and the active transport of iones across the cell membrane and takes place on the time scale $T_R = 2.5$ ms. In living nerve cells the resting potential lies at -70 mV. In our simulations we shift the scale of the electric potential and choose the resting value as zero.

The second term describes the change of the membrane potential due to interactions with other neurons. If the cell k which forms a synapse with neuron i has fired, a postsynaptic potential appears in cell i. The value of the postsynaptic potential corresponds to the synaptic strength $S_{ik}(i)$. Cell i continuously sums up the various excitatory and inhibitory postsynaptic potentials. If a threshold of $U_T = 30$ mV is exceeded the neuron fires an action potential and excites or inhibits nerve cells to which it connects. Subthreshold potentials relax to the resting potential as described by the first term.

Our model simplifies the dynamics of an action potential. If the neuron k fires, a monotonously decreasing function $G_i(\Delta t_i/T_u)$ describes the differential change of the postsynaptic membrane potential neuron i. The effect of the spike of neuron k on the postsynaptic cell i decays with the characteristic time $T_u=1$ ms. The evolution of the membrane potential can be considered as a renewal process (Abeles 1982) where only the latest spike enters into the interaction with another neuron. The dynamics forget the past of the nerve cell longer ago than the latest spike because a spike completely resets the internal state of the firing neuron.

The various processes to restore the cell membrane after firing are described by two refractory periods, a total refractory period and a relative refractory period. During the total refractory period the neuron possesses no sensitivity for any synaptic stimulus. The neuron gradually gains the capability to build up a potential during the relative refractory period. These two refractory periods limit the maximal spike frequency of the neuron and prevent the nerve cell from permanent firing. The total refractory period lasts for a period $T_F = 5$ ms. The effect of the relative refractory period decays on a time scale $T_F/2$.

The kinetic equation which describes the evolution of the membrane potential and which includes all aspects discussed is

$$\frac{dU_i}{dt} = \begin{cases} -U_i/T_R + \omega \varrho [\Delta t_i] \sigma [A_i(t)], \\ \text{if } U_F \leq U_i(t) \leq U_T; \\ -U_i/T_R, \text{ clsc.} \end{cases}$$
(1)

The first term in (1) describes the relaxation to the resting potential, the second term the effect of the communications of the ith neuron with the receptors and with other neurons. The key parameter which scales the neuronal communication is the coupling constant ω . This constant ω will be determined in Sect. 5.

 $\varrho[\Delta t_i]$ in (1) is a function which accounts for the existence of the total and the relative refractory period. $\Delta t_i = t - t_{0i}$ measures the time that has passed since the last spike of neuron i at t_{0i} . $A_i(t)$ in (1) is the activity function which sums up all spikes converging on the cell i and weighs them with the corresponding synaptic strength $S_{ik}(t)$. Afferent contributions of the receptors, firing with an input frequency T_i^{-1} , are also included in $A_i(t)$:

$$A_i(t) = \sum_k S_{ik} G_k (\Delta t_k / T_U) + \sum_i R_{ij} G_k^R (\Delta t_k^R / T_U). \tag{2}$$

 $\sigma[A_l(t)]$ in (1) is a sigmoidal function which represents saturation in the afferent signal. Potential changes beyond $(\omega T_U)^{-1}$ are restricted to a saturation value. A sensitive neuron with vanishing membrane potential, which is maximally excited by the saturating afferent activity $(\omega T_U)^{-1}$, reaches the threshold potential changes of the potential greater than the saturation value do not occur. Postsynaptic potentials below $(\omega T_U)^{-1}$ are counted linearly, i.e.

$$\sigma[A_i(t)] = \begin{cases} A_i(t), & \text{if } |A_i(t)| \le (\omega T_U)^{-1}; \\ (\omega T_U)^{-1}, & \text{if } A_i(t) > (\omega T_U)^{-1}; \\ -(\omega T_U)^{-1}, & \text{if } A_i(t) < -(\omega T_U)^{-1}. \end{cases}$$
(3)

The factor $\varrho[\Delta t_i]$ in (1) has been chosen such that the sensitivity of the neuron i is suppressed by means of the stepfunction $\Theta(\Delta t_i - T_F)$ or reduced within the

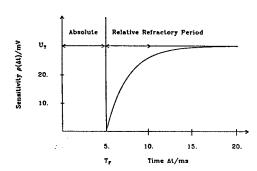


Fig. 3. The refractory state of the neuron after the firing at t = 0 is accounted for by $\varrho(\Delta t)$. During the absolute refractory period $[0,T_T]$ the neuron is insensitive to afferent activity. During the relative refractory period the sensitivity is gradually restored and reaches the asymptotic value U_T

total and the relative refractory period, respectively. We choose the following functional form represented in Fig. 3

$$\varrho[\Delta t_i] = U_T \Theta(\Delta t_i - T_F) \left(1 - G_i \left(\frac{\Delta t_i - T_F}{T_F/2} \right) \right). \tag{4}$$

 G_i occuring in (1), (2), and (4) is a memory function which describes the influence of the last firing of the neuron i on the network dynamics. For simplicity we choose an exponential dependence

$$G_i(\Delta t_i/\tau) = \exp\left(-\frac{\Delta t_i}{\tau}\right)$$
, with $\Delta t_i = t - t_{0i}$. (5)

When the threshold potential is reached and the cell fires, the continuous time evolution of the membrane potential $U_i(\Delta t_i/T_v)$ is interrupted. At that moment the past of the neuron is forgotten, the membrane potential is set to the refractory value U_F and the memory function $G_i(\Delta t_i/T_v)$ starts again with the value 1:

if
$$U_i(t) \ge U_T$$
 then
$$\begin{cases} t_{0i} = t, \\ U_i(t) = U_F, \\ G_i(\Delta t_i/\tau) = 1. \end{cases}$$
 (6)

4 Behavior of a Single Neuron

We investigate first the time evolution of a neuron i connected with a single receptor j. In this case the activity function $A_i(t)$ in (2) is restricted to one term which measures the input from the receptor j. The system of coupled first order differential equations (1) is reduced to

$$\frac{dU_i}{dt} = -\frac{U_i}{T_P} + \omega U_T R_{ij} \exp\left(-\frac{\Delta t_j^R}{T_U}\right). \tag{7}$$

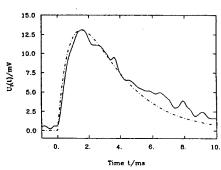


Fig. 4. The postsynaptic potential induced by a single receptor spike as calculated according to (6) is compared with the experimental data of Kuffler and Yoshikami. The data are rescaled to the maximum of the solution (8)

In the case of a single receptor spike at t=0 this equation can be solved analytically. For the solution we assume a vanishing initial value $U_i(0)$ and a neuron initially in the sensitive state. The solution yields a two-exponential description of the postsynaptic potential

$$U_i(t) = \omega U_T R_{ij} \frac{T_R T_U}{T_R - T_U} \left(\exp\left(-\frac{t}{T_R}\right) - \exp\left(-\frac{t}{T_U}\right) \right). \tag{8}$$

Figure 4 compares the time-dependence of (8) with the observations of Kuffler and Yoshikami (1975). The good agreement demonstrates that the present model describes satisfactorily the potential dynamics of single recursors.

5 Rescaling of the Network: The Effective Excitation Time

Two different dynamic field variables enter the network dynamics, the cell potentials $U_i(\Delta t_i/T_{ii})$ and the synaptic strengths $S_{ik}(t)$. The dynamics of these variables proceeds on two very different time scales, the potentials $U_i(\Delta t_i/T_{ii})$ being the fast variables, the synaptic strengths $S_{ik}(t)$ the slow variables. The coupling constant ω of the two fields $U_i(\Delta t_i/T_U)$ and $S_{ik}(t)$ dominates the time evolution of the fast variables, the cell potentials $U_i(\Delta t_i/T_0)$. In order to prevent the state of permanent (epileptic) firing or of quiescence in the network the coupling constant of the synaptic strengths and the cell potentials has to be adapted to the network parameters, i.e. to the number of synapses per neuron, to the time constants T_R , T_{II} , T_F , T_I defined in (1) and to the ratio R/S of the mean receptor-neuron connection value R and the initial synaptic strength S.

In this section we will develop an expression for the coupling constant ω . For this purpose we seek the mean-field equation for the cell potential of neuron i, excited by the mean afferent activity of the other neurons. We introduce a new time constant, the effective excitation time T_E of a neuron. In the mean field approximation, a sensitive, quiescent neuron $(U_i(\Delta t_i/T_0) = 0)$ will reach threshold after the interval T_E and, hence, will fire with a frequency $(T_E + 2T_F)^{-1} \approx (15 \text{ ms})^{-1}$ if it is excited by a mean afferent activity of the same spike frequency.

The mean of the neuron and receptor activity $A_i(t)$ of (2) is needed for this description. The sum $\Sigma_k S_{ik}(t) G_k(\Delta t_k/T_u)$ which appears in this equation must be averaged over the possible synaptic strengths and over time. We assume that both averages are independent, i.e. the randomly chosen synaptic strengths $S_{ik}(0)$ are not correlated with the memory functions $G_k(\Delta t_k/T_u)$ of the neurons k. The distribution of the sum $\Sigma_k S_{ik}(t)$ in an equilibrated network with just as

much excitatory as inhibitory synapses in the limit of many synapses approaches a Gaussian distribution with mean value 0 and variance

$$\Delta S = S \sqrt{N} . (9)$$

The variance estimates the fluctuation in the afferent neuronal interactions which are responsible for the firing of a neuron. N determines the number of afferent synapses per neuron.

To compute the time average of the memory function $G_k(\Delta t_k/T_U)$ we regard the firing of the nerve cell as a renewal process with a spike frequency $1/\tau$ and a constant probability dt/τ to fire in the interval dt. Then the distribution function of the interval between two spikes decays exponentially with the characteristic time τ and has the form

$$p_{\tau}(\Delta t) = \frac{1}{\tau} \exp\left(-\frac{\Delta t}{\tau}\right). \tag{10}$$

We average $G_k(\Delta t_b/T_v)$ with the distribution function (10) of Δt_k inserting the inverse neuronal spike frequency $\tau = T_F + 2T_F$ and obtain

$$\langle \Sigma_k S_{ik} G_k (\Delta t_k / T_U) \rangle = \Delta S \int_{-\infty}^{1} dt' p_s(t-t') \exp\left(-\frac{t-t'}{T_U}\right)$$
$$= \Delta S \frac{T_U}{T_E + 2T_E + T_U}. \tag{11}$$

The contribution of the receptors $\Sigma_j R_{ij} G_j^R (\Delta t_j^R / T_U)$ to the activity function $A_i(t)$ in (1) is also averaged over time and over the distribution of the connection strengths R_{ij} . The intervals between two receptor spikes again are assumed to be exponentially distributed according to (10) with the input time $\Delta t_j^R = T_l$. The average connection strength $\langle R_{ij} \rangle$ is set to R, the peak value of the center-surround-function. We obtain the average receptor contribution to the activity function $A_i(t)$

$$\langle \Sigma_k R_{ik} G_k^R \rangle = R \frac{T_U}{T_1 + T_U} \tag{12}$$

and the averaged activity function $A_i(t)$

$$\langle PSP \rangle = \Delta S \frac{T_U}{T_F + 2T_F + T_U} + R \frac{T_U}{T_I + T_U}. \tag{13}$$

If in (1) the function $A_i(t)$ is replaced by $\langle PSP \rangle$ we obtain the mean-field equation

$$\frac{dU_i}{dt} = -\frac{U_i}{T_R} + \omega U_T \langle PSP \rangle. \tag{14}$$

The solution of (14) with the initial value $U_i(0) = 0$ determines the coupling constant ω if we introduce the self-consistence condition that the threshold potential U_T is reached at time T_E . The coupling constant ω as a

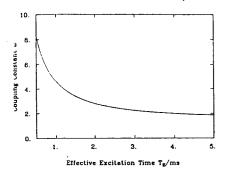


Fig. 5. The coupling constant $\omega(T_E)$, determined by (15), is shown as a function of the effective excitation time T_E

function of the effective excitation time T_E is given by the expression represented in Fig. 5.

$$\omega = \left(\langle PSP \rangle T_R \left(1 - \exp\left(-\frac{T_E}{T_R} \right) \right) \right)^{-1}. \tag{15}$$

Equation (15) furnishes a choice of the coupling coefficient ω [see (1)] which assures that the neural network avoids the states of epileptic hyperactivity or of abnormal quiescence. For this purpose one sets the parameter T_c to a desired value in the range of 1 ms to 3 ms. A need for the application of (15) arises, for example, if one wishes to fit the average firing rate $(T_E + 2T_E)^{-1}$ in the network to an experimental value. If one would alter for this purpose solely one of the parameters T_R , T_U , T_F , R or S in (1), the network dynamics could become completely unstable. A rescaling of the synaptic strengths $S_{ik}(t)$ through the choice (15) of the coupling constant ω avoids this difficulty. Another situation in which the need to apply a rescaling according to (15) arises is a change of the number of synapses per neuron.

6 Learning Through Synaptic Plasticity

In our model of learning, information is stored nonlocally in the synaptic connections of the network. The time evolution of the synapses, two orders of magnitude slower than the evolution of the cell potentials, couples adiabatically to the firing of the neurons and averages over many pre- and postsynaptic signals according to Hebbian rules. During the learning process which lasts between a tenth of a second and a few seconds the synapses between pairs of neurons are changed according to the activity states of the pairs. The synaptic evolution on a time scale below a second, first suggested by v. d. Malsburg (1981) and Little and Shaw (1975), until now is only a fiction. However, the

idea appears to be worthy of consideration since it endows neural networks with remarkable abilities as the results of this paper (Sect. 7) demonstrate. Experimentally, there exist only vague indications of a stable synaptic plasticity on the time scale below a second (Freeman 1977) since longer lasting effects of the synaptic changes are difficult to detect in electrophysiological experiments.

In our model, the plasticity of the synapse with the strength $S_{ik}(t)$, connecting neuron k to neuron i, evolves on the time scale $\Omega^{-1} = 300$ ms and is governed by the equation

$$\frac{dS_{ik}}{dt} = \begin{cases} -\frac{S_{ik}(t) - S_{ik}(0)}{T_{S}} + \Omega G_{k}(\Delta t_{k}/T_{M}) \kappa(G_{i}, G_{k}), \\ \text{if } S_{w} \ge |S_{ik}| \ge S_{i}; \\ -\frac{S_{ik}(t) - S_{ik}(0)}{T_{S}}, \text{ else} \end{cases}$$
(16)

which holds for excitatory and inhibitory synapses. The first term $-\frac{S_{ik}(t) - S_{ik}(0)}{T_S}$ accounts for the relaxation of the synapses to their initial values with the relaxation constant $T_S \approx 1-2s$. This term describes that stored information is forgotten. The second term in (16) causes the growth of the synapses. This term is governed by the function $\kappa(G_i, G_k)$ which distinguishes four different activity states of a pair of neurons i and k as presented below

	$G_i(\Delta t_i/T_M)$	$G_k(\Delta t_k/T_M)$	$\kappa(G_i, G_k)$	dS _{ik} /dt
(a)	>e ⁻¹	>e ⁻¹	+1	>0
(b)	<e^-1< td=""><td>> e⁻¹</td><td>-1</td><td><0</td></e^-1<>	> e ⁻¹	-1	<0
(c)	$>e^{-1}$	<e^-1< td=""><td>-1</td><td><0</td></e^-1<>	-1	<0
(d)	$>e^{-1}$ $$	<e<sup>-1</e<sup>	0	=0
				(17)

These values of $\kappa(G_i, G_k)$ enter (16). The reader should note that according to (16) the synaptic strengths do not change signs, i.e. excitatory synapses remain excitatory, inhibitory synapses remain inhibitory. The upper and lower bounds of the synaptic strength values $S_{ik}(t)$ are given by $S_u = 1.7 \, \text{S}$ and $S_1 = 0.01 \, \text{S}$.

The first state (a) in (17) denotes an activity of the neuronal pair when both the pre- and postsynaptic cells i and k fire synchronously within an interval $T_M = 15$ ms. The rule $\kappa(G_i, G_k) = 1$ in this case has been introduced by Hebb (1949) and implies growth of excitatory synapses and weakening of the inhibitory synapses in this state.

In the second state (b) only the presynaptic neuron has built up an action potential and the post-synaptic cell remains quiescent. In this case the factor

 $\kappa(G_i, G_k) = -1$ according to (16) diminishes the excitatory synapses and increases the strengths of the inhibitory synapses.

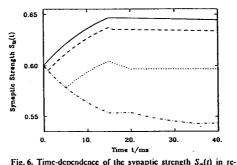
In the third state (c) only the postsynaptic cell has fired. The result on the synapses is the same as that for state (b).

In the fourth state (d) neither the postsynaptic cell k nor the presynaptic cell i is active. In this state the second term in (16) has no effect.

Figure 6 shows the changes which the strength S_{ik} of an excitatory synapse experiences if the presynaptic neuron k fires at t=0 and the postsynaptic cell i answers with a spike at $t=t_0$. If no interval separates the two spikes $(t_0=0)$ the synaptic strength S_{ik} grows most strongly (solid line). A time delay shorter than $\Delta t_0^* \rightarrow T_M \ln \left(\frac{2e+1}{e+2}\right)$ results in an asymptotic synaptic strength above the initial value. If the interval between the two spikes exceeds Δt_0^* the synaptic strength asymptotically decreases below the initial value.

The rules (17) promote the cooperation of cells activated at the same time and build up a cell assembly of excitatorily connected and cooperating neurons. This assembly separates itself from the set of cells with an asynchronous activity by inhibitory synapses to that set.

If more than one pattern is to be learned by the network an additional mechanism has to be introduced which protects the patterns learned earlier from destruction by patterns learned later. The synapses which contribute to a learned pattern are either very large, i.e. in the range $G_1 = [0.9 S_w, S_w]$ if excitatory, or are very small, i.e. in the range $G_2 = [-0.1 S_w, -S_1]$, if inhibitory. Synapses in these two ranges, therefore,



rig. 6. Introduce the synapse strength $S_{A}(t)$ in the sposs to one spike in the pre- and in the postsynaptic cell for 4 different spike intervals $\Delta \tau$. For $\Delta \tau = 0$ the synapse grows by a maximum amount, an interval $\Delta \tau = 20$ ms causes a strong decrease of $S_{A}(t)$. (solid line: $\Delta \tau = 0$ ms, dashed line: $\Delta \tau = 1$ ms, dotted line: $\Delta \tau = 5$ ms, dash-dotted line: $\Delta \tau = 20$ ms)

must be guarded, i.e. their dynamics is to be slowed down. For this purpose the time scale of the synaptic changes Ω_{κ}^{-1} is adapted to the current value of synaptic strengths. A decrease of the synaptic strength $(\kappa=-1)$ in the ranges G_1 and G_2 should require much more time than an increase $(\kappa=+1)$. The functional form of the factor Ω_{κ} chosen in our model for this purpose and represented in Fig. 7 is

$$\Omega_{\kappa} = \begin{cases} \Omega_{2}^{1}(1+\kappa) + \Omega \alpha_{2}^{1}(1-\kappa), & \text{if } S_{ik} \in G_{1} \vee S_{ik} \in G_{2}; \\ \Omega, & \text{elsc.} \end{cases}$$
(18)

The parameter α employed here lies between 0 and 0.2 ($0 \le \alpha \le 0.2$). The switching between the two values of

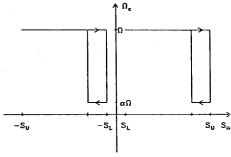


Fig. 7. Hysteresis in the synaptic growth factor Ω_{κ} : An increase of the synaptic strength $S_{\omega}(t)$ evolves an order of magnitude faster than a decrease if the synapse has reached the upper saturation boundaries S_{κ} and $-S_{1}$

Table 1. Network parameters Constants of the Fast Dynamics

 $T_{ij} = 1.0 \text{ ms}$ Time constant of a neural spike Relaxation time of the membrane potential $T_{\rm b} = 2.5 \, {\rm ms}$ $T_{\rm c} = 5.0 \, {\rm ms}$ Refractory period $T_1 = 1.0 \text{ ms}$ Time between two receptor spikes Effective excitation time, tuning *T_E*≈1.5 s parameter of the network activity $R/S \approx \sqrt{N}$ Coupling ratio between receptors and neural network $U_r = 30 \text{ mV}$ Threshold potential $U_0 = 0 \text{ mV}$ Resting potential U_F=-15 mV Refractory potential Constants of the Slow Dynamics $T_{\rm w} = 15.0 \, \text{ms}$ Coincidence interval of two spikes Synaptic relaxation $T_s \approx 1.0 \text{ s}$ Q = 1/(300 ms)Time scale of synaptic changes $\alpha \approx 0.1$ Hysteresis factor $S_{*} = 1.7 S$ Maximal synaptic value $S_1 = 0.01 \text{ S}$ Minimal synaptic value $L_{a} = 1.5$ Dimension of the excitatory center $L_i = 3.5$ Dimension of the inhibitory surrounding N≈150 Number of synapses per neuron Synaptic strength (arbitrary units) S≈60

 Ω_{κ} leads to a hysteresis effect which protects the stored information against overwriting by a later pattern.

The kinetic parameters which determine the neuronal dynamics of our model network are collected in Table 1.

7 Behavior of the Network with Receptor Input

The neural network proposed shows astonishing abilities to process and store information. This is demonstrated by simulation calculations. We will show that a homogeneous, i.e. uninstructed, network changes its synaptic structure and, as a result, its electrical activity patterns under the influence of stationary patterns. For a better visualization of the abilities of the network we chose the words brain and FEET (Fig. 8a and c) as input figures to the network.

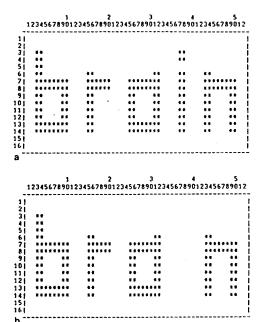
The figures brain and FEET are offered to the network with a frequency $T_1 \approx 1$ ms. The memory functions $G_i^R(\Delta t_i^R/T_H)$ of the receptors representing the figure brain are synchronously set to the value 1 after the interval T_i . The input frequency of the receptor activity $G_{i}^{R}(\Delta t_{i}^{R}/T_{i})$, chosen for convenience, can be critizised as unrealistically high. With a more sensitive network, i.e. $T_E \approx 1$ ms, the neurons could be excited by a much lower spike frequency of the receptors. The synchronicity of the receptor activity is postulated as a hypothesis and is not discussed in our model. We suppose by this hypothesis, that the synchronous receptors detect the same features, i.e. the pixel intensity or the colour, and, therefore, belong to the pattern. The stochastic activity of the receptors can be synchronized by lateral connections, as briefly discussed by v. d. Malsburg (1981).

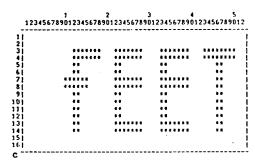
In the first simulation, described in Sect. 7.1, we present the figure brain to the network and analyse the changed connectivity. The success of the instruction is examined by an associative task. The network has to restore the letter i missing in the test figure (Fig. 8b).

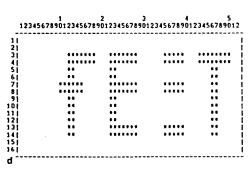
In the second simulation, discussed in Sect. 7.2, the network is required to learn and to associate two figures, brain and FEET. The problems arising from the interference of the two figures are analysed and the necessity of the hysteresis in the synaptic growth discussed in Sect. 6 is demonstrated.

A third simulation course, discussed in Sect. 7.3, deals with the abstraction of a prototype figure from a series of figures varying to a small degree. The network disregards the variations in the figures to be learned, i.e. abstracts the prototype figure.

Fig. 8a-4. Learning patterns (a,c) and test patterns (b,d) employed in our simulations. A "" indicates that the receptor at that position fires with a rate T_i^{-1} , i.e. the function $G_i^p(At_i^p/T_u)$ is set to 1 at this rate, and evolves according to (5) between these







In a last simulation, described in Sect. 7.4, the reaction of a trained network to a noisy figure is studied.

In all four simulations we analyse the evolution of the network monitoring the membrane potentials $U_i(\Delta t_i/T_v)$ and the information whether the neuron has fired or not. The membrane potentials $U_i(\Delta t_i/T_v)$ describe the millisecond dynamics of the neuron Potentials of the neurons representing the associated part of the test figure provide a measure of the success of learning and association.

7.1 The Learning of the Figure "Brain"

The first simulation has three different stages. In the first stage the uninstructed network learns the presented figure brain and changes its synaptic connections. In the second stage the receptors are quiescent and the membrane potentials relax to the resting state. In the third stage the success in learning is tested by the associative task to restore the missing letter i in the test figure. The following table summarizes the chronological order of the different stages.

0-300 ms: learning of the figure brain 300-320 ms: relaxation of the membrane potentials 320-360 ms: association of the missing i in the figure

The second stage has been introduced to assure that the potential $U_i(\Delta t_i/T_U)$ and the memory function $G_i(\Delta t_i/T_U)$ relax and that no after-effects of the millisecond dynamics can deceive the associative restoration of the test figure. The interval of 20 ms in which the network receives no input spikes from the receptors guarantees that only the changed synaptic strengths and not the membrane potentials contain information about the learned figure.

The reaction of the network after the presentation of the figure brain in stage 1 is represented in Fig. 9a which shows the membrane potentials after 30 ms. Most of the neurons which receive input from receptors belonging to the figure (figure neurons) have fired and are resting in the refractory phase or have reached already the sensitive phase and are again summing up postsynaptic potentials. The neurons in the immediate surrounding of the pattern which do not receive input from those receptors (background neurons), are inhibited by the center-surroundconnectivity R_{ii} between the receptors i and neurons i. A few of the background neurons in the upper half of the network are excited at the beginning of the learning course because they are connected to the figure, neurons by enough excitatory synapses. These connections raise their membrane potential, although not above threshold.

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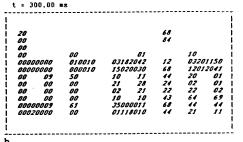
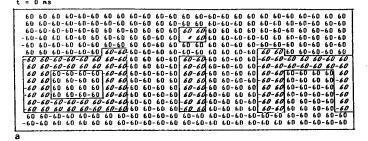


Fig. 9a, b. Membrane potentials after 30 ms (a) and 300 ms (b). The values of the membrane potentials are divided by the threshold value U_T , and are represented by the next value if positive. The symbol "a" indicates that the cell potential has reached the threshold. If the memory function $G_L(\Delta t_d/T_U)$ exceeds 1/e the integer is italicized. Negative potentials are represented by a blank, or by an italic zero 0, if $G_L(\Delta t_d/T_U)$ exceeds 1/e

After 300 ms (Fig. 9b), i.e. at the end of the learning stage, the background cells are strongly inhibited and only the neurons belonging to the figure show a positive membrane potential or are in the refractory state.

The strong inhibition of the background neurons by the figure neurons originates from the altered synaptic connectivity of the network. The figure neurons during the learning stage have fired between 20 and 30 spikes, whereas the background cells have never reached the threshold. This activity leads, according to (17), to alterations of the synaptic strengths $S_{ii}(t)$.

In Fig. 10 we represent, both before and after the learning stage, the strengths of the synapses which connect the neuron (37,4) [the neuron receiving input from the receptor which represents the point of the in brain] with the other neurons. According to the kinetics laid down in (16), (17), the synapses between two figure neurons k and i, i.e. neurons which have



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Fig. 10. Synaptic changes during learning of the pattern brain. The initial strengths and the strengths at t = 300 ms are shown for the synapses which start from the cell (37.4), the position of which is represented "*". Those synapses which end at a figure neuron are indicated by bold italic numbers

often fired synchronously, are strengthened. The synaptic strengths $S_n(t)$ of these synapses are saturated either at the value $S_{\nu} = 99$, if the corresponding synapse is excitatory, or at the value $-S_1 = -1$, if the corresponding synapse is inhibitory. Excitatory and inhibitory synapses connecting the figure neuron (37.4) to background neurons are saturated at the lower boundary values S_t or $-S_{tot}$ respectively. Figure 10 also demonstrates the nonlocal properties of the storage. Each neuron contains in its synaptic connections a blueprint of the pattern stored in the network.

After the relaxation of the membrane potentials in stage 2, the network is excited in stage 3 by the test figure, the latter being identical to the pattern brain learned in stage 1, except that the letter i is missing. The time evolution of the membrane potentials during the first few milliseconds of this association task is represented in Fig. 11.

The neurons which obtain input spikes from the receptors react immediately with a raised membrane potential. At t = 324 ms, 4 ms after the beginning of the association test, several of the neurons belonging to the new figure bra n have fired and the potentials of the remaining neurons exceed the value 20 mV. At t = 325 ms all except three neurons of this set have fired a spike whereas the potentials of neurons representing the missing i have reached threshold or are just below threshold. Figure 11 also illustrates the mechanism

underlying the associative properties of the network. The figure neurons are connected excitatorily with each other, as shown by Fig. 10. If a subgroup of the figure neurons, e.g. the neurons of the letters a and n, fires spikes strong postsynaptic membrane potentials are evoked in the neurons of the missing letter i. These postsynaptic potentials compensate the missing receptor inputs and stimulate the postsynaptic cells to fire, too. The firing of the pattern neurons which are stimulated by other figure neurons and not directly by the receptors is delayed for only about 1 to 2 ms. At time t = 326 ms all cells of the letter i have fired. The network has restored the test pattern, associating the missing figure components.

7.2 Learning and Association of Several Patterns

The pattern brain in the preceding simulation has been stored with high redundancy. Each cell contains information about the whole pattern and the storage capacity of the synaptic structure is not exhausted at all. Now we will investigate the possibility to store and associate several independent patterns with partial overlap. For this purpose we simulate the learning and storage of the patterns brain and FEET, represented in Fig. 8a and c, the overlap of which involves nearly a fourth of their neurons. This simulation should demonstrate that more than one pattern can be stored in the network. We do, however, not investigate its full

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Fig. 11a-c. Membrane potentials during the association task described in Sect. 7.1 (for definition of the potential values see Fig. 9)

storage capacity which by far exceeds the information content of the two patterns considered here (Palm 1980; Hopfield 1982). The chronological order of the different stages of this simulation is the following:

0-300 ms: learning of the figure brain 300-320 ms: relaxation of the membrane potentials 320-720 ms: learning of the figure FEET 720-740 ms: relaxation of the membrane potentials 740-800 ms: association of the missing letter i in brain 800-820 ms: relaxation of the membrane potentials 820-880 ms: association of the incomplete letter E in FEET.

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Fig. 12a, b. Membrane potentials during the simulation described in Scct. 7.2. The state of the network is shown at the end of the two learning stages (for definition of the potential values see

The membrane potentials evolve during the first 300 ms, the learning stage of the pattern brain, exactly as in the simulation described in Sect. 7.1. When the receptors present the second pattern some interference effects between the stored pattern brain and the presented pattern FEET arise in the network. At the beginning of the second learning stage, the neurons belonging to both figures try to inhibit the neurons which belong only to the second figure FEET. During the first learning stage of the pattern brain, the neurons which represent FEET but not brain have constituted part of the background and, therefore, have become inhibitorily connected with the brain-neurons. If the receptors are coupled strongly enough to the network $(R/S \approx 1/N)$ these inhibitory connections are suppressed and the learning of the second pattern is not disturbed by the information already stored in the network. In Fig. 12b, at t=700 ms, the end of the second learning stage, the membrane potentials reflect the presented pattern FEET.

The association properties are tested by the test patterns, Fig. 8b and d. The network associates the missing letter i and completes the letter E without any

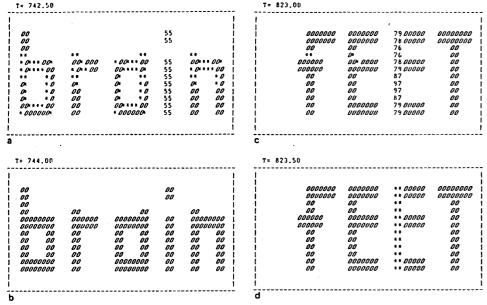


Fig. 13a-d. Membrane potentials of the simulation described in Sect. 7.2 during the association of the first (a, b) and of the second test pattern (c, d) (for definition of the potential values see Fig. 9)

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Fig. 14a, b. Strengths of the synapses which originate from the neurons (37,10) and (33,4) after the learning of the two patterns brain and FEET (for definition of the synaptic strengths values see Fig. 10)

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Fig. 15. Strengths of the synapses which originate from the neuron (37,4) after the two learning stages. The neuron (37,4) belongs to both patterns (for definition of the synaptic strengths values see Fig. 10)

faults. The millisecond dynamics of the membrane potentials during the association tests shows no significant differences to the simulation in Sect. 7.1. In Fig. 13a-d the successful association of both patterns is demonstrated.

More insight into the information storage is gained by considering the synaptic structure. Figure 14 represents the strengths of the synapses which connect neuron (37,10) (a) and neuron (33,4) (b) with their respective neuronal surroundings. These two neurons belong to the pattern brain (37,10) and to the pattern FEET (33,4), respectively. The synaptic changes correspond to the results of the preceding simulation where only one pattern is presented.

In Fig. 15 we represent the synaptic structure of neuron (37,4) which belongs to both brain and FEET. The synapses connecting this neuron to neurons of the second figure grow from the lower boundaries S, and -S., where they have saturated after the first learning stage, to the upper saturation boundaries S_n and $-S_n$. The inverse process, the decrease of the synapses connecting neuron (37,4) to neurons of the first pattern brain, is prevented by hysteresis in the synaptic growth factor (see Sect. 6). According to the rule (17b) the synapses between cell (37,4) and neurons of the pattern brain are diminished during the learning of the pattern FEET, but this process is retarded by an order of magnitude and changes the respective synapses by about 5 percent only. As a result, information about the pattern learned earlier is conserved.

7.3 Abstraction of a Prototype Figure

Another property of the network, the abstraction of a prototype figure, will be demonstrated now. In this simulation the receptors present a series of figures which contains the word brain and two moving horizontal bars. Every 20 ms the network receives a new pattern of the series, the bars being diplaced cyclically by one pixel row. Figure 16 shows two consecutive patterns of the series presented to the network.



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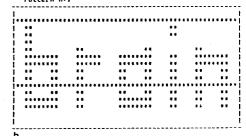


Fig. 16a, b. Two successive patterns of the series of patterns presented to the network in the simulation described in Sect. 7.3. The pattern consists of the word brain and two moving bars (compare a and b)

The result of the association test after the learning stage of 300 ms is shown in Fig. 17. The success of the reconstruction proves that the network disregards the moving bars and learns only the invariant figure brain. If the modified synaptic strengths of the network are analysed at t=300 ms, only very small traces originating from the added bars can be detected. Synchronicity between the neurons representing the pattern brain and the neurons representing the bars has occured so rarely that the synaptic changes according to the rule 17a are negligible in comparison to synaptic relaxation.

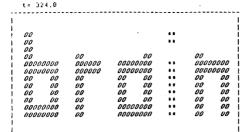


Fig. 17. The network instructed by a series of patterns as shown in Fig. 16 associates the missing i without faults

7.4 Noise Suppression by a Trained Network

In the preceding three simulations the network had to restore, after the learning stage, an incomplete but stationary test figure. A trained autoassociative network with the dynamics as in (1) is also capable to suppress, from the test figure, strong non-stationary background noise which contains no correlations over an interval greater than 1 ms. In the following simulations all receptors belonging to the second test figure fire simultaneously with a frequency of 1 ms⁻¹. In addition to this synchronous firing, the receptors representing the background fire uncorrelated noise spikes with an average spike frequency 1 ms⁻¹.

In Fig. 18a-c, respectively, the activity $G_I^R(\Delta t_I^R/T_U)$ of the receptors, the afferent activity $\Sigma_I R_{ij} G_I^R(\Delta t_I^R/T_U)$ entering in (2), and the network reaction are shown. The receptors present an extremely noisy picture to the network. The local averaged intensity, which is defined as the spike frequency of the receptors, does not discriminate the figure from the noisy ground. Only the simultaneity of all spikes belonging to the test figure separates the information from the meaningless noise. A neuron has to sum up the activity of several milliseconds to reach the threshold. A figure which involves strong correlation in time raises the probability of activating the neuron, whereas an uncorrelated noisy pattern facilitates relaxation of the membrane potential and inhibition of the neuron.

The center-surround-organisation in the receptorneuron connections differentiates the input pattern twice (Marr 1982) and filters out a homogeneous background activity. As a result strong noise also has the effect that the afferent activity $\sum_i R_{ij} G_j^R (\Delta t_i^R/T_u)$ decreases together with the local differences in the activity function $G_j^R (\Delta t_j^R/T_u)$. Therefore, the neurons must be made more sensitive than in case of an association test with undisturbed figures to let association succeed.

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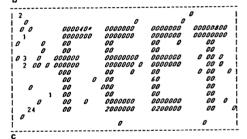


Fig. 18a-c. The simulation of the neural network as described in Sect. 7.4: Shown are the memory function $G_1^R(At_1^R/T_U)$ (a), the afferent contribution $\Sigma_1R_{11}G_1^R(At_1^R/T_U)$ of the receptors (b) and the membrane potentials of the neurons which react to the receptor input. (c) The input pattern contains the test pattern of Fig. 9d and a strong background noise. The receptor-neuron connection strength R is given the value 1. The network suppresses the noise and filters out the hidden information

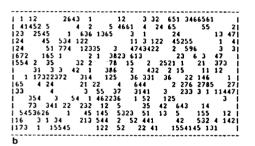
Neural membrane potentials are shown in Fig. 18c. The cooperating neurons representing the pattern FEET have resonantly coupled to the noisy input pattern and inhibit the background neurons, although these also receive receptor spikes.

If the receptors present only noise, i.e. a pattern without a synchronous component, the situation changes drastically, as shown in Fig. 19. Neurons react with an intermediate spike frequency of (150 ms)⁻¹. No correlated firing of the neurons representing one of

the two stored patterns is detected. The membrane potentials vary stochastically between the refractory and the threshold potential as demonstrated in Fig. 19c.

The simulation summarized in Fig. 20 demonstrates that both learned patterns can be restored even in the presence of strong noise. The learning stages are chosen as in the simulation with two learning patterns, described in Sect. 7.2. The association and restoration

12828*2861668*8*666858442666816*2*655**64******5666561 2**8*84*822165*61*4816*68**614*458*4**611212**63821* 8* 68*8*1161*46***66***1*14*6183856285*83384313*66** 18*6646**18886*6*66666458228482*65*686**6**2465618568 16+15833+83+**185+6+444 +25 ###6+#4#25+55###4612685## 1 * * 85 288 * * 2 * 1 4 * 466 * 1 854 * * 6 * 5 * 688 5 2 * 26 25 # 446 * 566 24 # 448 | 1 *** 5 * 368 * 544548 * 188482 * * 668 * 382 * 63* * 8 * 383384 * 265 * * 65 18 228828*2*46882 86568**31 8835*8*3583**42 42*2*85661 126*36**85**6263*6*524*8*8388*6*8581**8166864*85325861 | *8536*48*434544864*861 68613*8*486356856**18***4666* 6*321246*51 1544*54**5*B*B158688618*282*881*4668688* 18**823*31 **3686**888*33 *6*82154**65145545113*25 216*661**668815*68418866*568218*83*625***4365*6862381 16*8*8***6145683 **3*8*84**6868*5565*86*36626**14388 14*5 8* *4**4265*585*8*12*18*82**651 11*8385**66*63*8 18**82*64*888*6 5361868*4**256*56884386***3** 8*638 61



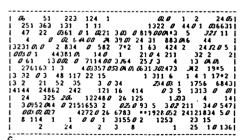


Fig. 19a—c. The simulation of the neural network as described in Sect. 7.4: Shown are the memory function $G_I^p(Al_I^p/T_U)$ (a), the afferent contribution $E_IR_{i,j}G_I^p(Al_I^p/T_U)$ of the receptors (b) and the membrane potentials of the neurons which react to this input. The input pattern is a strong background noise only. No cooperating neuronal assembly is excited and the network reacts only with an intermediate spike frequency of (150 ms)⁻¹

of a pattern with strong background noise is tested according to the following time table:

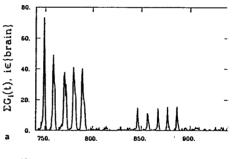
740-780 ms: First Test Pattern with Strong Noise

780-840 ms: Noisc

840-880 ms: Second Test Pattern with Strong Noise

880-940 ms: Noisc.

In Fig. 20a and b we represent the total activity $\Sigma_i G_i(\Delta t_i/T_u)$ of the assembly of those neurons which represent either the pattern brain or the pattern FEET We first consider the assembly of neurons representing the figure brain. The total activity of this assembly is represented in Fig. 20a. When the first test input (Fig. 8d) is presented from t=740 ms to t=780 ms the assembly of the pattern brain fires nearly synchronously. However, when only noisy input is received in the interval t=780 ms to t=840 ms this assembly reacts with a low spike rate. The noise input does not stimulate firing of the whole assembly but only elicits



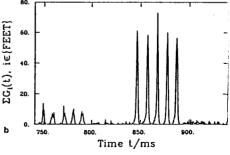
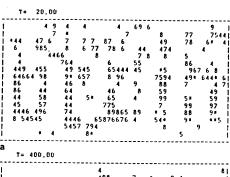
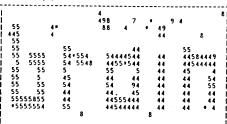


Fig. 20a, b. The simulation of the neural network as described in Sect. 74: shown are the activity response of the assemblies representing the figure brain (a) and FEET (b). During the interval [740 ms, 780 ms] the test figure 8b with added noise was presented. During the interval [840 ms, 880 ms] the test figure 8c (c) The input pattern is a strong background noise only. No cooperating neuronal assembly is excited and the network reacts only with an intermediate spike frequency of (150 ms)⁻¹

very few neuronal spikes. When the pattern FEET is presented in the interval t=840 ms to t=880 ms the assembly corresponding to brain exhibits a level of activity which is significantly below the response during the 740 ms to 780 ms interval. The response is due to those cells which are common to both the brain and the FEET neuronal assemblies.





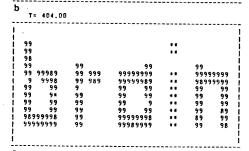


Fig. 21a-c. The memory functions $G_k(At_i/T_u)$ of the simulation 7.5 are shown at the beginning, i.e. t = 20 ms, (a) and at the end, i.e. at t = 400 ms, (b) of the learning stage. The pattern to be learned has to be abstracted from the figure brain disturbed by strong noise. In the association stage the figure brain is presented without noise. The response is shown in (c). The values of the memory functions which ly between 1 (firing) and 0 (resting state) are coded by integer numbers, i.e. 9 stands for $G_k(At_i/T_w) = 0.9$ etc.

"" denotes a neuron which is firing momentarily and a blank indicates neurons momentarily at rest

This simulation shows that the network can distinguish between a test pattern embedded in strong noise and a pattern which contains only noise and no underlying information. The noisy patterns can be restored associatively.

7.5 Learning of a Noisy Pattern

In a final simulation we test the ability of an untrained network to learn a pattern which during the whole learning stage is disturbed by a strong background noise. The noise is presented by an additional background activity of the receptors with an average frequency of $(2 \text{ ms})^{-1}$. Except for this noise the simulation repeats the three stages of Sect. 7.1. Figure 21 presents the neuronal activity $G_i(\Delta t_i/T_M)$ at the beginning (t=20 ms) and at the end (t=400 ms) of the learning stage. At the beginning the network fires with an intermediate spike frequency of $(50 \text{ ms})^{-1}$. Several background neurons are observed to fire excited by the noise.

During the learning stage the activity of the background neurons is stochastic and uncorrelated, whereas the neurons corresponding to the figure brain fire synchronously and, thereby, build up excitatory connections. At the end of the learning stage, the assembly of figure neurons inhibits the background. Apart from a few mistakes the network has recognized the figure brain and has separated it from the background. However, the time the network needs to detect the correlations in the pattern and to store the presented information in the current simulation is longer than the time needed in the case of undisturbed learning. Also the time between two neuronal spikes exceeds the average inverse frequence $T_E + 2T_F$ of the undisturbed learning.

The association test is presented in Fig. 21c. The missing letter i of the figure is associated without fault after 4 ms. The faults which developed during the learning stage in the figure bra n, i.e. in the letters b and r, cannot be corrected.

8 Conclusion

We have presented a network model with neural units which are closely related to their physiological counterparts with respect to the dynamics of their membrane potential. The neural units are coupled by plastic synapses the dynamics of which is governed by few local rules. These rules induce global cooperation and competition and, thereby, endow the network with the ability for noise filtering and for associative storage and recall of large patterns. The basis of the rules of synaptic plasticity is the discrimination between states of pairwise synchronous and asynchronous neural

activity, synchronicity being measured on a time scale of a few milliseconds. This ability may enable neural networks in a primitive state of development to separate figure and ground on the basis of synchronous and asynchronous inputs and, in a more advanced state of development, it may contribute to higher brain functions.

Our work was motivated by our belief that brain models should not be too distant from physiological detail. The results obtained in the present article are not beyond those of previous attempts to describe brain function by simple physical modelling. One may, in fact, consider our work as a proof that previous abstract models of neural networks (Cooper 1973; Hopfield 1982; Kohonen 1984) have a physiological basis. However, the present approach may also show its value above that of abstract network models when one starts to investigate how more complex local interactions of neurons affect the global network function. Examples for such investigation are the inclusion of measures of synchronicity between more than two neurons or the effect of local neurochemical agents which act beyond the range of neutrotransmitters.

Previous models as well as ours point out clearly that what matters in neural assemblies are not the spikes of single neurons but rather the cooperative or competitive activity of many neurons. In this respect theoretical approaches are likely to make an important contribution to the Neurosciences in that they alone can relate the knowledge of the local behaviour of neuronal tissue to the global behaviour which codes for the "atoms" of information processed in the brain. To play this role, theoretical brain science has to incorporate physiological detail into its calculations and cannot remain abstract. Further modelling of physiological details, e.g. noise induced postsynaptic potentials, are currently investigated by us.

Acknowledgement. It is a pleasure to acknowledge many interesting discussions with Christoph v. d. Malsburg on the subject of this paper and his useful comments on the manuscript.

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Received: February 26, 1986

Professor Dr. Klaus Schulten Joachim Buhmann Physikdepartment Technische Universität München James-Franck-Strasse D-8046 Garching Federal Republic of Germany