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 presentation of 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 interpreted this special 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 milliseconds 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 uninstruction state of the network. This model is based as closely as possible on physiological facts. We like to show that such physiological neural networks are capable of associative 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 excited 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 the neuronal net. The activity of the neurons is interpreted as the output of the network. The backward bended arrows indicate the feedback due to the effect of the neuron activity on the synaptic strengths $S_{ij}(t)$ between neuron $i$ and $j$ in the neuronal network.

The connections between the receptors and the "physiological" neurons have a local center-surround organization with peak value $R$. Receptors $j$ which are living in the neighborhood 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 neuronal net, the projection being locally convoluted with the center-surround 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 $2t_{c}$ and suppress the longer ranged noise which is part of the background.

The synapses $S_{ij}(t)$ which carry action potentials from cell $k$ to cell $i$ are initially chosen at random, i.e. $S_{ij}(0) = \pm\delta$ with equal probability for an excitatory or an inhibitory synapse. Hence, the network is initially completely uninstruction, 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 1933) 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 $R/S > 1$ the network is very strongly coupled to the receptors, whereas in the case $R/S < 1$ 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 ions across the cell membrane and takes place on the time scale $T_{f} = 2.5$ ms. In living nerve cells the resting membrane 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 $S_{ij}(t)$. Cell $i$ continuously sums up the various excitatory and inhibitory postsynaptic potentials. If a threshold of $U_{th} = 30$ 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 $G(t)$ describes the differential change of the postsynaptic membrane potential in neuron $i$. The effect of the spike of neuron $k$ on the postsynaptic cell $i$ depends on the characteristic time $T_{f}$. The evolution of the membrane potential in cell $i$ 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. After this refractory period the neural cell becomes refractory to the passage of current for a time period $T_{f}/2$. The kinetic equation which describes the evolution of the membrane potential and which includes all aspects discussed is

$$\frac{dU}{dt} = \begin{cases} -\frac{U(t)}{T_f} + \text{eq}[I(t) - \text{eq}[I(t) - U(t)]; \text{eq}[I(t)], \text{if } U(t) \leq U_{th} \leq U(t); \text{eq}[I(t)], \text{else.}} \end{cases}$$
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 \( \omega \). This constant \( \omega \) will be determined in Sect. 5.

\( g(dT_i) \) in (1) is a function which accounts for the existence of the total and the relative refractory period. \( d_i = t - t_{ref} \) measures the time that has passed since the last spike of neuron \( i \) at \( t_{ref} \). \( A_i(t) \) in (1) is the activity function which sums up all spikes converging on the cell \( i \) and weights them with the corresponding synaptic strength \( S_i \). Affected contributions of the receptors, firing with an input frequency \( T_i \), are also included in \( A_i(t) \):

\[
A_i(t) = \sum S_i G_i(d_i/T_i) + \sum R_k G_k(d_i/T_k).
\]

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

\[
\begin{align*}
\sigma(A(t)) &= \begin{cases} 
A(t) & \text{if } |A(t)| \lesssim (\omega T_i)^{-1} \\
(\omega T_i)^{-1} & \text{if } A(t) > (\omega T_i)^{-1} \quad \text{or} \quad (\omega T_i)^{-1} > A(t) \quad \text{or} \quad A(t) < - (\omega T_i)^{-1}
\end{cases}
\end{align*}
\]

The factor \( g(dT_i) \) in (1) has been chosen such that the sensitivity of the neuron is suppressed by means of the stepfunction \( \Theta(dT_i - T_i) \) or reduced within the total and the relative refractory period, respectively. We choose the following functional form represented in Fig. 3

\[
\begin{align*}
\sigma(dT_i) &= U_i \Theta(dT_i - T_i) \quad \text{for } \quad 1 - G_i \left( \frac{dT_i - T_i}{T_i/2} \right) \quad \text{for } \quad t_i = t_i - T_i.
\end{align*}
\]

\( G_i \) occurring 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

\[
G_i(dT_i/t) = e^{-dT_i/\tau}, \quad \text{with } \quad dT_i = t_i - t_i.
\]

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

\[
U_i(t) = U, \quad G_i(dT_i/T_i) = 1, \quad \text{if } U_i(t) \geq U \quad \text{then} \quad U_i(t) = U_i, \quad 0
\]

5. Rescaling of the Network: The Effective Excitation Time

Two different dynamic field variables enter the network dynamics, the cell potentials \( U_i/dT_i/T_0 \) and the synaptic strengths \( S_i \). The dynamics of these variables proceeds on two very different time scales, the potentials \( U_i/dT_0/T_0 \) being the fast variables, the synaptic strengths \( S_i \) the slow variables. The coupling constant \( \omega \) of the two fields \( U_i/dT_i/T_i \) and \( S_i \) dominates the time evolution of the fast variables, the cell potentials \( U_i/dT_i/T_i \). In order to preserve 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 adjusted to the network parameters, i.e. to the number of synapses per neuron, to the time constants \( T_0, T_0, T_0, T_0 \), 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 \( \omega \). For this purpose we seek the mean-field equation for the cell potential of neuron \( i \), excited by the mean affective 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 \( U_i/dT_i/T_0 = 0 \) will reach threshold after the interval \( T_E \) and, hence, will fire with a frequency \( (T_E + 2T_0)^{-1} = (15 \text{ ms})^{-1} \) if it is excited by a mean affective activity of the same spike frequency.

The mean of the neuron and receptor activity \( \tilde{A}_i \) of (2) is needed for this description. The sum \( \sum S_i(dT_i/T_i) \) 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 \( S_i \) are not correlated with the memory functions \( G_i(dT_i/T_i) \) of the neurons \( k \). The distribution of the sum \( \sum S_i(t)/n \) 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 \( \Delta S = \sqrt{N} \).

\[
\text{(9)}
\]

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 \( G_i(dT_i/T_i) \) we regard the firing of the nerve cell as a renewal process with a spike frequency \( 1/\tau \) and a constant probability \( \tau \) to fire in the interval \( T_i \). The distribution function of the interval between two spikes decays exponentially with the characteristic time \( \tau \) and has the form

\[
p_i(t) = \frac{1}{\tau} e^{-t/\tau}.
\]

We average \( G_i(dT_i/T_i) \) with the distribution function \( p_i(t) \) of (10) at \( dT_i \) inserting the inverse neuronal spike frequency \( \tau = T_0 + 2T_0 + T_0 \) and obtain

\[
\langle \sum S_i G_i(dT_i/T_i) \rangle = \Delta S \int dt \tau \left( 1 - e^{-t/\tau} \right) \left( 1 - e^{-t/\tau} \right) = \Delta S T_0 T_0 + 2T_0 + T_0
\]

The contribution of the receptors \( \sum S_i G_i(dT_i/T_i) \) to the activity function \( A_i(t) \) in (1) is also averaged over time and over the distribution of the connection strengths \( R \). The intervals between two receptor spikes again are assumed to be exponentially distributed according to (10) with the input time \( dT_i = t_i \). The average connection strength \( \langle R \rangle \) is set to \( R \), the peak value of the center-surround function. We obtain the average receptor contribution to the activity function \( A_i(t) \)

\[
\langle \sum R_k G_i \rangle = \langle R \rangle \frac{T_0}{T_0 + 2T_0 + T_0} + R \frac{T_0}{T_0 + 2T_0 + T_0}
\]

and the averaged activity function \( A_i(t) \)

\[
\langle \psi \rangle = \Delta S \frac{T_0}{T_0 + 2T_0 + T_0} + R \frac{T_0}{T_0 + 2T_0 + T_0}
\]

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

\[
\frac{dU_i}{dt} = -U_i \frac{T_0}{T_0} + \langle \psi \rangle.
\]

The solution (14) with the initial value \( U_i(0) = 0 \) determines the coupling constant \( \omega \) if we introduce the self-consistence condition that the threshold potential \( U_i \) is reached at time \( T_0 \). The coupling constant \( \omega \) as a
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 \( S_0(s) \), connecting neuron \( k \) to neuron \( i \), evolves on the time scale \( T^2 = 300 \) ms and is governed by the equation

\[
\frac{dS_0}{dt} = \begin{cases} 
- \frac{S_0(t) - S_0(0)}{T_2} + G_3 \frac{dt/T_2}{(G_4 \cdot G_5)} \cdot S_0(t) \cdot G_2 \cdot G_4 \cdot G_5, \\
\frac{S_0(t)}{T_2} + G_3 \frac{dt/T_2}{(G_4 \cdot G_5)} \cdot S_0(t) \cdot G_2 \cdot G_4 \cdot G_5 \quad \text{if} \quad S_0(t) \geq S_1, \\
- \frac{S_0(t) - S_0(0)}{T_2} + G_3 \frac{dt/T_2}{(G_4 \cdot G_5)} \cdot S_0(t) \cdot G_2 \cdot G_4 \cdot G_5 \quad \text{else},
\end{cases}
\]

(16)

which holds for excitatory and inhibitory synapses.

The first term \(- \frac{S_0(t) - S_0(0)}{T_2}\) accounts for the relaxation of the synapses to their initial values with the relaxation constant \( T_2 \approx 2 \) s. 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 \( k(G_2 \cdot G_4 \cdot G_5) \) which distinguishes four different activity states of a pair of neurons \( k \) and \( i \) as presented below

\[
\begin{array}{c|c|c|c|c}
S_0(t)/S_2 & G_3(dt/T_2) & G_2 \cdot G_4 \cdot G_5 \cdot S_0(t) & dS_0/dt \\
\hline
(a) & > -1 & > -1 & +1 & > 0 \\
(b) & < -1 & > -1 & -1 & < 0 \\
(c) & < -1 & < -1 & 0 & = 0 \\
(d) & > -1 & < -1 & 0 & = 0 \\
\end{array}
\]

(17)

These values of \( k(G_2 \cdot G_4 \cdot G_5) \) 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 \( S_0(t) \) are given by \( S_0 = 1.7 \) S and \( S_0 = 0.01 \) S.

In the first state \((a)\) in (17) denotes an activity of the neuronal pair when both the pre- and postsynaptic cells \( i \) and \( k \) fire simultaneously within an interval \( T_2 = 15 \) ms. The rule \( k(G_2 \cdot G_4 \cdot G_5) = 1 \) 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 postsynaptic neuron has built up an action potential and the postsynaptic cell remains quiescent. In this case the factor \( k(G_2 \cdot G_4 \cdot G_5) = -1 \) 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 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 synaptic strengths \( S_0 \) of an excitatory synapse experiences if the postsynaptic neuron \( k \) fires at \( t = 0 \) and the postsynaptic cell \( i \) answers with a spike at \( t = T_2 \). If no interval separates the two spikes \( t_2 = 0 \) the synaptic strength \( S_0 \) grows most strongly (solid line). A time delay shorter than \( T_2 \) results in an asymptotic synaptic strength above the initial value. If the interval between the two spikes exceeds \( T_2 \) 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 \( G_2 = [0.9 \cdot S_0, S_2] \) if excitatory, or are very small, i.e., in the range \( G_2 = [-0.1 \cdot S_0, -S_1] \) if inhibitory. Synapses in these two ranges, therefore, must be guarded, i.e., their dynamics is to be slowed down. For this purpose the time scale of the synaptic changes \( T_k \) is adapted to the current value of synaptic strengths. A decrease of the synaptic strength \( k = -1 \) in the ranges \( G_2 \) and \( G_3 \) should require much more time than an increase \( k = +1 \). The functional form of the factor \( G_3 \) chosen in our model for this purpose and represented in Fig. 7 is

\[
G_3 = \frac{\Omega_1(t + \kappa)}{\Omega_2(t + \kappa)},
\]

(18)

where \( \kappa \) is defined as \( \kappa = \frac{1}{2} \) and \( \Omega_1 = (1 + \kappa) \cdot \Omega_2(1 - \kappa) \).

The parameter \( \kappa \) a employed here lies between 0 and 0.2 (\( \kappa \geq 0 \)). The switching between the two values of

![Fig. 7. Hysteresis in the synaptic growth factor \( G_3 \). An increase of the synaptic strength \( S_0(t) \) evolves an order of magnitude faster than a decrease if the synapse has reached the upper saturation boundaries \( S_0 \) and \( -S_1 \).

Table 1. Network parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( T_0 )</td>
<td>1.0 ms</td>
</tr>
<tr>
<td>( T_2 )</td>
<td>2.5 ms</td>
</tr>
<tr>
<td>( T_0 )</td>
<td>5.0 ms</td>
</tr>
<tr>
<td>( T_1 )</td>
<td>1.0 ms</td>
</tr>
<tr>
<td>( T_2 )</td>
<td>1.5 ms</td>
</tr>
<tr>
<td>( R/S )</td>
<td>( \sqrt{N} )</td>
</tr>
<tr>
<td>( U_0 )</td>
<td>30 mV</td>
</tr>
<tr>
<td>( U_0 )</td>
<td>0 mV</td>
</tr>
<tr>
<td>( U_0 )</td>
<td>-15 mV</td>
</tr>
<tr>
<td>( \kappa )</td>
<td>0.5</td>
</tr>
<tr>
<td>( \Omega_1(t + \kappa) )</td>
<td>( \Omega_2(1 - \kappa) )</td>
</tr>
</tbody>
</table>

![Fig. 6. Time-dependence of the synaptic strength \( S_0(t) \) in response to one spike in the pre- and postsynaptic cell for different spike intervals \( \Delta t \). For \( \Delta t = 0 \) the synapse grows by a maximum amount, an interval \( \Delta t = 20 \) ms causes a strong decrease of \( S_0(t) \) (solid line: \( \Delta t = 0 \) ms; dashed line: \( \Delta t = 1 \) ms, dotted line: \( \Delta t = 3 \) ms, dash-dotted line: \( \Delta t = 20 \) ms). ]
In the 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 $U_i(t)$ and the information whether the neuron has fired or not. The membrane potentials $U_i(t)$ 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 untrained 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.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-300 ms</td>
<td>learning of the figure brain</td>
</tr>
<tr>
<td>300-320 ms</td>
<td>relaxation of the membrane potentials</td>
</tr>
<tr>
<td>320-360 ms</td>
<td>association of the missing i in the figure brain</td>
</tr>
</tbody>
</table>

The second stage has been introduced to assure that the potential $U_i(t)$ and the membrane function $G_i(t)$ are high enough to induce the associative and the action potential 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 the receptor belonging to the figure (figure neurons) have fired and are resting in the refractory phase. The spikes have reached already the sensitive phase and are again summing up postsynaptic potentials. The neurons in the immediate surroundings of the pattern which do not receive input from those receptors (background neurons), are inhibited by the center-surround connectivity $R_{jk}$ between the receptors $j$ 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.

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 $U_i > U_T$ and by the next value minus 1 if $U_i <= U_T$. The cell potential has reached the threshold. If the membrane function $G_i(t)$ exceeds 1, the integer is italicized. Negative potentials are represented by a blank, or by an italic zero 0, if $G_i(t)$ exceeds 1.

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 $S_{ij}(t)$.

In Fig. 10 we represent, both before and after the learning stage, the strengths of the synapses which connect the neuron (17, 4) [the neuron receiving input from the receptor which represents the point of the i 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
often fired synchronously, are strengthened. The synaptic strengths $S_i(t)$ of these synapses are saturated either at the value $S_i = 99$, if the corresponding synapse is excitatory, or at the value $S_i = -1$, if the corresponding synapse is inhibitory. Excitatory and inhibitory synapses connecting the figure neuron (31,4) to background neurons are saturated at the lower boundary values $S_i = 0$ or $S_i = -1$, respectively. Figure 10 also demonstrates the nonlocal properties of the storage. Each neuron contains in its synaptic connections a blueprint of the pattern that is the end result 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 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 $b$, 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 Fig. 12a, 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 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: association of the incomplete letter E in FEET.

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 inhibitory connected with the brain-neurons. If the receptors are coupled strongly enough to the network ($R_S = 1/V$) 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)

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 $S_L$ and $-S_L$ where they have saturated after the first learning stage, to the upper saturation boundaries $S_U$ and $-S_U$. 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 displaced cyclically by one pixel row. Figure 16 shows two consecutive patterns of the series presented to the network.

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 occurred so rarely that the synaptic changes according to the rule 17a are negligible in comparison to synaptic relaxation.

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)
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. The simulation was started with a resting activity G₉(Td/T₀) of the receptors, the afferent activity ΣRₐG₉(Td/T₀) entering in (2), and the network reaction is 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 receptor-neuron connections differentiates the input pattern (Marr 1982) and filters out a homogeneous background activity. As a result strong noise also has the effect that the afferent activity ΣRₐG₉(Td/T₀) decreases together with the local differences in the activity function G₉(Td/T₀). Therefore, the neurons must be more sensitive than in case of an association test with undisturbed figures to let association succeed.

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 the inhibitive activity is much higher.

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

<table>
<thead>
<tr>
<th>Time (ms)</th>
<th>Activity Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>740-780</td>
<td>First Test Pattern with Strong Noise (780-840 ms: Noise)</td>
</tr>
<tr>
<td>840-880</td>
<td>Second Test Pattern with Strong Noise (880-940 ms: Noise)</td>
</tr>
</tbody>
</table>

In Fig. 20a and b we represent the total activity ΣRₐG₉(Td/T₀) of the assembly of those neurons which represent either the pattern FT 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 noise input is received Fig. 8e, 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
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.

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 ms)^{-1}. Except for this noise the simulation repeats the three stages of Sect. 7.1. Figure 21 presents the neuronal activity G(4i) for the beginning and the end of the learning stage. At the beginning the network fires with an intermediate spike frequency of (50 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 frequency G(4i) of the undisturbed learning.

The association test is presented in Fig. 21c. The missing letter l of the figure is associated without fault after 4 ms. The faults which developed during the learning stage in the figure bra, 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. Synchrony is being measured on a time scale of a few milliseconds. This ability may enable neural models to provide a primitive state of development to separate figure from background. In this way 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 neurotransmitters.

Previous models as well as ours point out clearly that what matters in neural networks 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 details into its calculations and cannot remain abstract. Further modelling of physiological details, e.g. noise induced postsynaptic potentials, are currently investigated by us.

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References


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