A Physiological Neural Network as an Autoassociative Memory

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1. Introduction

We consider a neural network model in which the single neurons are chosen to resemble closely known physiological properties. The neurons are assumed to be linked by synapses which change their strength according to Hebbian rules [1] on a short time scale (100ms) [2]. Each nerve cell receives input from a primary set of receptors, which offer learning and test patterns without changing their own properties. The activity of the neurons is interpreted as the output of the network (see Fig.1). The backward bended arrows in Fig.1 indicate the feed-back due to the effect of the neuron activity on the synaptic strengths S_{ik} between neuron k and i in the neural network.

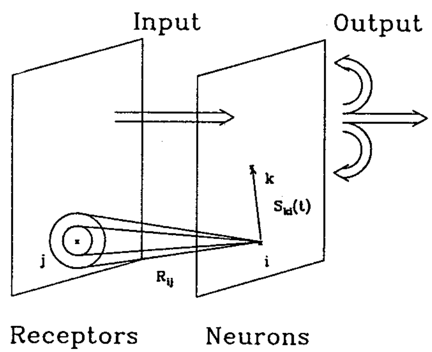

Figure 1
Schematic presentation of the model investigated: Receptors send spikes to a network of neurons. The connectivity between the receptors j and neurons i is given by the matrix R_{ij}, the connectivity between the neurons is given by $S_{ik}(t)$. The resulting activity of the neural network is affected by an activity-dependent alteration of $S_{ik}(t)$, i.e. the network experiences a feed-back as indicated.

Initially the synapses S_{ik} which carry action potentials from cell k to cell i are chosen at random, i.e. the network is initially completely uninstructed. The connections between the receptors and the 'physiological' neurons possess no plasticity and have a local center-surround-organization. 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

NATO ASI Series, Vol. F20
Disordered Systems and Biological Organization
Edited by E. Bienenstock et al.

excitatory center have an inhibitory effect on neuron i. 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 center-surround function.

2. Dynamics of the Cell Potential

The fast dynamics of a neuron involves its cell potential which changes on the time scale of a few milliseconds. In our model two important contributions to the dynamics of the potential are included. A first term describes the relaxation of the cell potential which takes place on the time scale T_R (T_R=2.5ms). The second term accounts for the change of the cell potential due to interactions with other neurons. If the cell k which forms a synapse with neuron i has fired, a postsynaptic potential difference corresponding to the synaptic strengths S_{ik} appears in cell i. The cell i continuously sums up the various excitatory and inhibitory postsynaptic potentials. If a threshold U_T=30mV is exceeded the neuron fires an action potential and excites or inhibits nerve cells connected to it. Sub-threshold potentials relax against the resting potential.

The dynamics of an action potential is simplified by the following rules: If the neuron k fires, a monotonously decreasing function

$$G_i(\Delta t_i/\tau) = \exp(-\Delta t_i/\tau) \quad \text{with } \Delta t_i = t - t_{0i} \; , \qquad (1)$$

describes the differential change of the postsynaptic potential in the neuron i. In Eq.(1) t_{0i} indicates the time of the latest firing. The effect of the spike of neuron k on the postsynaptic cell i decays with the characteristic time T_U (T_U=1ms).

The kinetic equation which describes the time evolution of the cell potential is

$$dU_i/dt = \begin{cases} -U_i/T_R + \omega \; U_T \; \rho(G_i(\Delta t_i/T_F)) \; A_i(t) & U_F < U_i(t) < U_T \\ U_F & \text{else} . \end{cases} \qquad (2)$$

The first term in the upper equation describes the relaxation to the resting potential, the second term the communications of the i-th neuron with the receptors and with other neurons. The key parameter which scales the neuronal communication is the coupling constant ω. This constant ω can be used to rescale the network dynamics [3] by the equation

$$\omega = \{\langle PSP \rangle \; T_R \; [1-\exp(-T_E/T_R)]\}^{-1} \qquad (3)$$

where <PSP> estimates the average postsynaptic potential. The parameter T_E, the effective excitation time of the neuron, determines the time which a neuron requires to reach the

threshold U_T if it has rested in the sensitive state and if the connected neurons fire with a average spike rate $(T_E+2T_F)^{-1}$. Equation (3) furnishes a choice of the coupling coefficient ω which assures that the neural network avoids the states of epileptic hyperactivity or of abnormal quiescence.

$A_i(t)$ in Eq.(2) is the activity function which sums up all spikes converging on the cell i and weights them with the corresponding synaptic strength S_{ik}. External contributions of the receptors presented with an input frequency T_I^{-1} are included in

$$A_i(t) = \Sigma_k S_{ik} G_k(\Delta t_k/T_U) + \Sigma_j R_{ij} G_j^R(\Delta t_j^R/T_U) \; . \qquad (4)$$

$\rho(G_i)$ in Eq.(2) is a function which accounts for the existence of the total and relative refractory period T_F=5ms. The factor ρ is chosen such that the sensitivity of the neuron i is suppressed or reduced in the total and relative refractory period, respectively. We choose the following functional form

$$\rho[G_i(\Delta t_i/T_F] = \theta(\Delta t_i - T_F) \; \{1-G_i[2(\Delta t_i - T_F)/T_F)]\} \; . \qquad (5)$$

When the threshold potential is reached and the cell fires, the continuous time evolution of the cell potential i is interrupted and the memory function $G_i(\Delta t_i/T_U)$ starts again with the value 1. In addition the cell potential is set to the refractory value of U_F=-15 mV:

$$\text{if} \quad U_i(t) > U_T \quad \text{then} \quad U_i(t) \rightarrow U_F \quad \text{and} \quad t_{0i} = t \qquad (6)$$

3. Learning through Synaptic Plasticity

In our model of learning information is stored nonlocally in the synaptic connections of the network. The plasticity of the synapse with the strength S_{ik}, leading from the neuron k to i, evolves on the time scale Ω^{-1}=300ms and is governed by the Eq.

$$dS_{ik}/dt = \begin{cases} -\mathcal{R}(S_{ik}) + \Omega G_k(\Delta t/T_M) \kappa(G_i, G_k) & \text{if} \quad S_u > |S_{ik}| > S_l \\ -\mathcal{R}(S_{ik}) & \text{else} \end{cases} \qquad (7)$$

$$\mathcal{R}(S_{ik}) = [S_{ik}(t) - S_{ik}(0)]/T_S$$

which holds for excitatory and inhibitory synapses. The first term $\mathcal{R}(S_{ik})$ accounts for the relaxation of the synapses to their initial values during the time $T_S \approx$1s. The second term in (7) causes a 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
$> e^{-1}$	$> e^{-1}$	+1	> 0
$< e^{-1}$	$> e^{-1}$	-1	< 0
$> e^{-1}$	$< e^{-1}$	-1	< 0
$< e^{-1}$	$< e^{-1}$	0	$= 0$

(8)

Figure 2 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 $t=t_0$. A time delay shorter than $\Delta t_0^* = T_M \ln(2e+1)/(e+2)$ results in an asymptotic synaptic strength above the initial value, otherwise below.

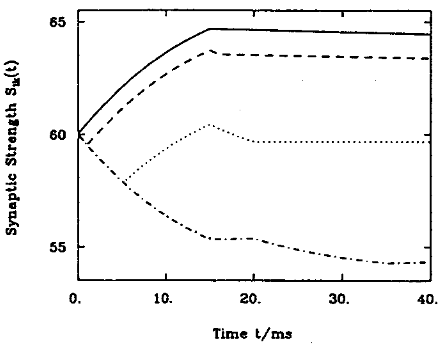

Figure 2
Time-dependence of the synaptic strengths $S_{ik}(t)$ in case of two spikes in the pre- and postsynaptic cells for 4 different spike intervals $\Delta\tau$. For $\Delta\tau=0$ the synapse grows at a maximum rate, an interval $\Delta\tau=20$ms causes a strong decrease of $S_{ik}(t)$ ($T_M=15$ms); (———) $\Delta\tau=0$ms, (- - - -) $\Delta\tau=1$ms, (·······) $\Delta\tau=5$ms, (-·-·-) $\Delta\tau=20$ms.

4. Behavior of the Network with Receptor Input

The first simulation has two different stages. In the first stage the uninstructed network learns the presented figure **brain** (Fig.3a) and changes its synaptic connections. In the second stage the success in learning is tested by the associative task to restore the missing letter i in the test figure **bra n**.

1. stage	0 - 300 ms	:	learning of the figure **brain**
	300 - 320 ms	:	relaxation of the cell potentials
2. stage	320 - 360 ms	:	association of the missing i in **bra n**

The interval of 20ms between stage 1 and 2 in which the network receives no input spikes from the receptors guarantees that only the changed synaptic strengths and not the cell potentials contain information about the learned figure.

The reaction of the network after the presentation of the figure **brain** in stage 1 is presented in Fig.3b which shows the cell potentials after 10 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 sum up postsynaptic potentials in the sensitive phase. 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 cell potential but not above the threshold.

At t=290ms (Fig.3c) the background cells are strongly inhibited and only the neurons belonging to the figure show a positive cell potential or are in the refractory state.

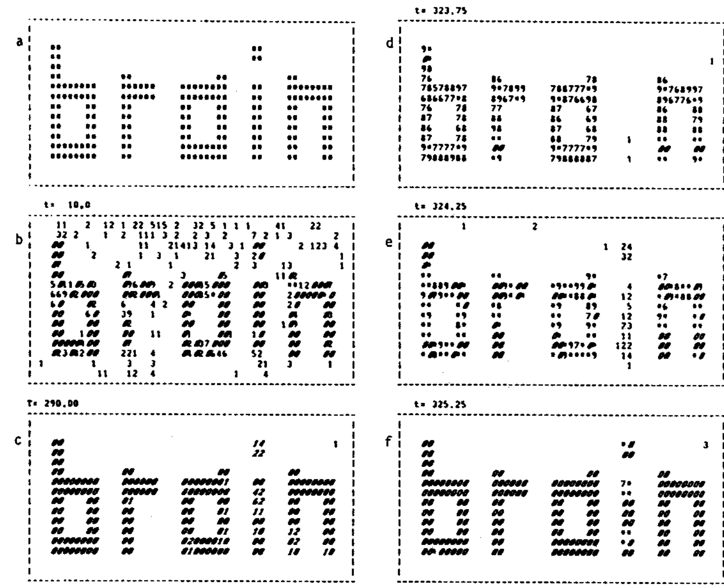

Figure 3
Learning Pattern (a) and Cell Potentials after 10ms (b) and 290ms (c). The values of the cell potentials are divided by the threshold value U_T and represented by the next integer value if positive. The symbol * indicates that the cell potential has reached the threshold. If the memory function $G_i(\Delta t_i/T_U)$ exceeds 1/e the integer is italicized. Negative potentials are presented by a blank or by a italic zero *0* if $G_i(\Delta t_i/T_U)$ exceeds 1/e. On the right side the cell potentials are presented during the restoration of the missing i.

After the relaxation of the cell potentials the network is excited in stage 2 by the test figure which is identical to the pattern **brain** learned in stage 1 except that the letter i is missing. The time evolution of the cell potentials during the first few milliseconds of this association task is presented in the Figs.3d,e,f. The neurons which obtain input spikes from the receptors react immediately with a raised cell potential. At t=323.75ms, 3.75 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 15mV. At t=325.25ms all except one neurons of this set have fired a spike whereas the potentials of neurons representing the missing i have reached the threshold or are just below the threshold. The Figures 3d,e,f reveal also the mechanism which underlies the associative properties of the network. If a subgroup of the figure neurons, e.g. the neurons of the letters a and n, fires spikes, strong postsynaptic cell 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 with a delay of 1-2ms.

In Figure 4 we present both before and after the learning stage the strengths of the synapses which connect the neuron (37,4) representing the point of the i in **brain** with the other neurons. According to the kinetics laid down in Eqs. (7),(8) the synapses between two figure neurons k and i are strengthened. The synaptic strengths S_{ik} of these synapses are saturated either at the value S_u=99 if the corresponding synapse is excitatory, or at the value $-S_l$=-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_l or $-S_u$, respectively. Figure 4 also demonstrates the nonlocal properties of the storage. Each single neuron contains in its synaptic connectivity a blueprint of the pattern stored in the network.

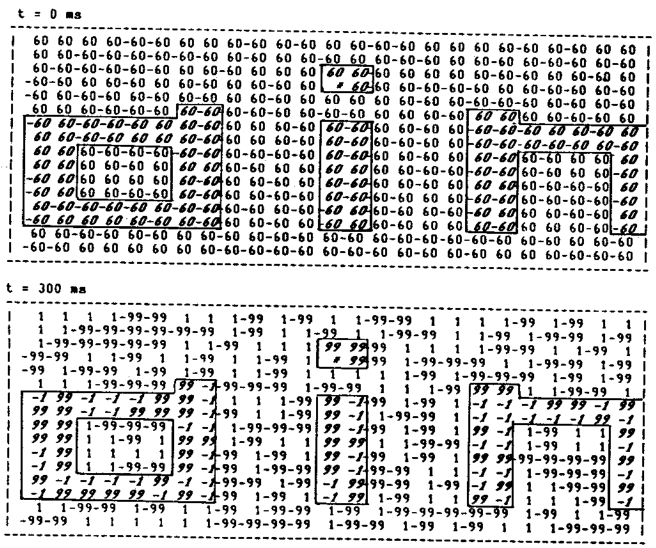

Figure 4
Synaptic changes during the learning of **brain**: The initial synaptic strengths and the strengths at t=300ms of the synapses starting from cell (37,4) are presented. Synapses which end at a figure neuron are indicated by italic, bold numbers.

5. Conclusion

In the network model presented above neural units closely related through the dynamics of their cell potential to their physiological counterparts interact by few local rules of synaptic plasticity. These rules induce global cooperation and competition and, thereby, endow the network with the ability for associative storage and recall of patterns. The basis of the rules of synaptic plasticity is a discrimination between states of pairwise synchroneous and asynchroneous neural activity, synchronicity being measured on a time scale of a few ms. Beyond the investigated behavior of the network, also more complex computational properties emerge in the neural net discussed, i.e. the ability to filter strong noise out of a presented pattern, to build up a prototype pattern from a series of varying patterns and to store several patterns [3].

6. <u>References</u>

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J. Buhmann, K.Schulten: to be submitted to Biol.Cybern.