

Influence of Noise on the Function of a “Physiological” Neural Network

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Abstract. A model neural network with stochastic elements in its millisecond dynamics is investigated. The network consists of neuronal units which are modelled in close analogy to physiological neurons. Dynamical variables of the network are the cellular potentials, axonic currents and synaptic efficacies. The dynamics of the synapses obeys a modified Hebbian rule and, as proposed by v. d. Malsburg (1981, 1985), develop on a time scale of a tenth of a second. In a previous publication (Buhmann and Schulten 1986) we have confirmed that the resulting noiseless auto-associative network is capable of the well-known computational tasks of formal associative networks (Cooper 1973; Kohonen et al. 1984, 1981; Hopfield 1982). In the present paper we demonstrate that random fluctuations of the membrane potential improve the performance of the network. In comparison to a deterministic network a noisy neural network can learn at lower input frequencies and with lower average neural firing rates. The electrical activity of a noisy network is very reminiscent of that observed by physiological recordings. We demonstrate furthermore that associative storage reduces the effective dimension of the phase space in which the electrical activity of the network develops.

assemblies. The issue of selforganization, which, of course, is central to many phenomena of the material and biological world, has been given systematic attention in recent years through the work of Prigogine, Eigen, Haken and others (for Refs. see Ebeling and Feistl 1982). The problem to bridge the vast hierarchy of structures and processes between the electrically active neural membrane and higher brain function will probably occupy scientists for many years to come. This problem is approached today from both of its extremes. Physiological scientists are studying those properties of nerve cells, e.g., the plasticity of synapses (Kandel and Schwartz 1982; Changeux et al. 1984; Kelso et al. 1986), which are assumed to be most essential for the selforganization and function of neural assemblies. Neurochemists are making much progress identifying the molecular messengers which the central nervous system employs to organize its function on the lowest inter-cellular level (Goelet et al. 1986). At the same time theoretical scientists have approached the problem of brain function on the macroscopic level of very many interconnected neurons. Exploiting the concepts developed for non-linear selforganizing dynamical systems and analogies between neural and statistical mechanical systems the theoretical studies have shown that functions of a surprising complexity, which one may well call intelligent, can be realized by model neural systems with rather simple properties of its individual components (Hopfield 1982; Rumelhart et al. 1986). This connection between macroscopic complexity and microscopic simplicity may provide an answer to the question raised above about the relationship of single cell properties and human intelligence.

Most previous theoretical studies on neural assemblies have aimed to simplify the neuronal units as much as possible going to the extreme of identifying neurons with two state spins. While it is certainly advantageous to strive for an extreme in abstraction to

1 Introduction

One of the traditional themes of scientific investigations which reemerges today with new vigour concerns the molecular, structural and functional self-organization of neural networks in the brain. The question posed is how the molecular and structural properties of single nerve cells give rise to the self-organization and intelligent function of large neural

gain understanding, such abstraction may impede an identification of properties of the model system with the properties of the physiological system. We have, therefore, adopted in our work on the theory of neural networks a description of neuronal units in close analogy to their physiological counterparts. The cost of such an approach is, of course, that the resulting theory does not yield to mathematical methods derived from the physics of spin glasses (Hopfield 1982, 1984; Amit et al. 1985a, b, 1987; Kinzel 1985) but rather requires numerical simulations by means of coupled non-linear differential equations. Our first results were, nevertheless, encouraging although may be not surprising: we could demonstrate that the recent suggestions about information processing in formal neural networks (Hopfield 1982) can also be realized through models with closer analogies to the biological system.

Most models of neuronal networks involve two variables: axonic currents represented by a two state neuron, and synaptic efficacies, represented by a continuous interval of values with upper and lower bounds. We have modified in our previous model the corresponding dynamics of neural networks in two respects: (1) axonic currents are represented by continuous time-dependent functions; (2) cellular potentials are added as a third class of (continuous) variables. These modifications move the model closer towards the realm of physiological neurons, but the resulting model neurons are still much more primitive than their physiological counterparts. The relationship between model neural networks and brain tissue touches on the cardinal question of theoretical neurobiology, namely what properties of real neurons are essential for the computational capabilities of the brains. In respect to this question we have assumed that model neurons should be endowed with a memory buffer which stores information about (1) at which time a neuron fired last, and (2) what signals converged onto a neuron before firing. However, our model neglects many other properties of physiological neurons which are likely to be also essential. The most important properties of this kind concern the detailed time course of the generation of postsynaptic potentials from presynaptic signals and the time course of the propagation of axonic pulses.

In our previous model with cellular potentials, axonic currents and synaptic efficacies as dynamic variables we demonstrated that such networks endowed with the Hebbian rule (Hebb 1949) for the plasticity of their synapses are capable of associative storage, adaptive filtering and other basic intelligent tasks. The cellular potentials and axonic currents of our model had been monitored during execution of tasks. The results showed two weaknesses: (1) the

receptor input to our model system had to be very intense, driving the spike activity of the neurons at a very high frequency; (2) the neurons engaged in associative recognition tasks fired in an extremely synchronous fashion and did not act as disorderly as recordings typically show. We decided, therefore, that a realistic model of neural systems should include a noise source. However, we were concerned about the possibility that a noisy network loses its information processing abilities. In this article we will present our investigation on model neural networks with a noise source. The surprising and important result is: a noise source which affects the cellular potentials improves the performance of neural networks as measured by association and filtering tasks. We believe, therefore, that we have identified another microscopic property basic for the function of the brain, namely noise acting on the neural cellular potentials. This conclusion, if true, has great ramifications for the interpretation of recordings from cells in the brain. Our conclusion confirms results by Little and Shaw (1975) who detected an enhanced storage capacity when noise had been added to their model network.

At this stage we cannot identify the kind of noise which contributes to brain function in the suggested way. Three possibilities exist: (1) membrane noise originating from the stochastic closing and opening of ion channels; (2) the noise which originates from the release of neurotransmitter (Shaw and Vasudevan 1974), e.g. acetylcholine; (3) the noise connected with afferent synaptic signals. Since the first two sources of noise can be altered by neurochemical agents, the conclusion reached in this paper may shed new light on the relationship between brain function and brain chemistry.

Our previous theory of neural networks (Buhmann and Schulten 1986) described a set of interconnected neurons, the membrane potentials of which evolve according to deterministic rules. The dynamics which were assumed for the potential of a certain nerve cell involves the relaxation to the resting state as well as the interaction of that neuron with all those neurons which have formed efferent synapses. The assumed time scale of the dynamics of membrane potentials measures about a millisecond. The connections to sensory organs or other neural networks were taken into account by a primary set of receptors which provide input to the neurons. The receptor-neuron connections constituted a local projection of the activity pattern presented by the receptors and had been realized either by a one-to-one projection or by a center-surround connectivity. A second most important aspect of the theory involved a dynamic plasticity of the synapses which endows the network with its abilities for adaptive filtering and associative memory. The synaptic

strengths change according to the Hebbian-like synchronicity rule, i.e. the synaptic strengths increase if the pre- and postsynaptic cells fire a spike synchronously and decrease if there exists no synchronicity between pre- and postsynaptic spikes. The postulated synchronicity rule deviates from the traditional interpretation of Hebb's rule which requires conjunction of integrated activities and not coincidence of single spikes for synaptic plasticity. For the sake of numerical convenience we allowed, in violation of Dale's law, efferent and afferent synapses of a single neuron to be excitatory as well as inhibitory; however, we did not allow a change of synaptic type. In such network the synaptic connectivity is random initially and contains no information. The network undergoing the described dynamics possesses the ability to learn and associatively stores patterns frequently presented by the receptors. The network can suppress background-noise added to the patterns and can select and store the prototype pattern if the patterns are subject to variations between presentations.

As argued above we generalize here the deterministic model and add a noise source to the dynamics of the membrane potential. The fluctuations of the membrane potentials induce spontaneous spike activity. This spontaneous activity requires some alteration of the Hebbian synchronicity rule assumed in our previous description of noise-less neural networks (Buhmann and Schulten 1986). The accidental synchronicity of two spontaneous spikes in two neurons should not strengthen the corresponding synapse. In the same vein, a spontaneous spike should not weaken a synapse if the postsynaptic cell does not fire accidentally at the same moment. To avoid the destruction of the synaptic connectivity by spontaneous activity we supplement the Hebbian rule by the condition that synapses are only modified in case the mean spike rate of a neuron \bar{v}_i considerably exceeds the spontaneous rate v_s .

The noise acts in our model on the cellular potential and only indirectly on the spike activity. We will demonstrate in this publication that such noise facilitates neural firing in response to weak receptor input. In the deterministic model weak inputs cause only changes of the membrane potential in the sub-threshold range, fail to stimulate firing of neurons and, therefore, do not alter synaptic connectivity. The observed performance of our model system presented below will demonstrate that the noise regulates the level of attention, perhaps comparable to the level of arousal in animals, and is an essential feature of the information processing abilities of the neural network and not a mere source of disturbance better suppressed. Our results below also show that the stochasticity in the dynamics of the membrane potential prevents the

unnatural behaviour in the spike activity of the deterministic model mentioned above, namely a phase-locking of spikes of different neurons and an excessive spike activity. Furthermore, we will demonstrate that the inclusion of noise reproduces closely the noisy appearance of the observed electrical activity of neural units (Abeles 1982).

The inclusion of fluctuations of the membrane potential in the theory of neural networks allows also to simulate the nonspecific influence of large neural nets on a small neural assembly, the latter being described in detail by membrane potentials and time-dependent synaptic strengths, the former being accounted for by background noise.

In Sect. 2 of this paper we introduce the equations which describe the time evolution of the membrane potentials $U_i(t)$ under the influence of noise. The resulting behaviour of a single neuron is considered for a case without receptor input in Sect. 3 and for a case with receptor input in Sect. 4. In Sect. 5 we introduce the dynamics which governs synaptic plasticity. In the last part of this paper we analyse by means of the correlation integral method of Grassberger and Procaccia (1983) the effective dimension of the phase-space in which the dynamics of the membrane potentials U_i evolves.

2 Dynamics of the Membrane Potential

In our model the membrane potentials represent the fastest dynamical variables and evolve on a time scale of a few milliseconds. Their dynamics involves relaxation to the resting value as well as increase and decrease due to synaptic interactions between neurons. We define as the resting potential $U_0 = 0$ mV and assume a relaxation time $T_R = 2.5$ ms. The postsynaptic dendritic membrane sums up postsynaptic potentials induced by presynaptic spikes and correspondingly alters the cellular potential. Each presynaptic action potential of neuron k during an interval $T_U = 1$ ms contributes a postsynaptic potential change of neuron i which is determined by the synaptic strength $S_{ik}(t)$. The time decay of the influence of a presynaptic spike is approximated by

$$G_k(\Delta t_k/\tau) = \exp\left(-\frac{\Delta t_k}{\tau}\right) \quad (1)$$

$\Delta t_k = t - t_k^0$ measures the time that has passed since the last spike of neuron k at t_k^0 , τ is a time constant defined further below. This description reproduces satisfactorily the synaptic interaction between neurons (Buhmann and Schulten 1986).

The kinetic equations for the membrane potentials, which also include stochastic fluctuations, constitute a

system of non-linear coupled Langevin equations

$$\frac{dU_i}{dt} = -\frac{U_i}{T_R} + \varrho[\Delta t_i] \left(\omega \sigma[A_i(t)] + \frac{\eta}{\sqrt{T_R/2}} \xi(t) \right). \quad (2)$$

The first term approximates the relaxation of the membrane potential $U_i(t)$. The second term describes the communication of the postsynaptic cell i with the connected neurons and receptors and adds Gaussian white noise with the strength $\eta/\sqrt{T_R/2}$. The strength of the noise yields a distribution of the membrane potential $U_i(t)$ with variance η ($\eta = 10$ mV). The mean value U_0 of the distribution vanishes if the neuron receives no other afferent inputs. $\xi(t)$ is a Gaussian normal-distributed variable with the properties

$$\langle \xi(t) \rangle = 0, \quad \langle \xi(t) \xi(t+t') \rangle = \delta(t'). \quad (3)$$

Afferent contributions to the potential are accounted for by $A_i(t)$ which describes the neural and receptor excitation of neuron i . The activity of the presynaptic neurons k and receptors j are weighted by the synaptic strengths $S_{ik}(t)$ and R_{ij} , respectively:

$$A_i(t) = \sum_k S_{ik} G_k(\Delta t_k/T_U) + \sum_j R_{ij} G_j^R(\Delta t_j^R/T_U). \quad (4)$$

The function $\sigma[A_i(t)]$ restricts potential changes to the saturation value $(\omega T_U)^{-1}$

$$\sigma[A_i(t)] = \begin{cases} A_i(t), & \text{if } |A_i(t)| \leq (\omega T_U)^{-1}; \\ (\omega T_U)^{-1}, & \text{if } A_i(t) > (\omega T_U)^{-1}; \\ -(\omega T_U)^{-1}, & \text{if } A_i(t) < -(\omega T_U)^{-1}. \end{cases} \quad (5)$$

As in the deterministic model (Buhmann and Schulten 1986) total and relative refractory periods are taken into account by a function $\varrho[\Delta t_i]$

$$\varrho[\Delta t_i] = \Theta(\Delta t_i - T_{F_a}) \left(1 - G_i \left(\frac{\Delta t_i - T_{F_a}}{T_{F_r}} \right) \right). \quad (6)$$

The step function $\Theta(\Delta t_i - T_{F_a})$ suppresses the sensitivity of neuron i to afferent excitation during the total refractory period $T_{F_a} = 5$ ms. During the relative refractory period of $T_{F_r} = 2.5$ ms the neuron gradually regains its sensitivity to incoming excitation or inhibition.

The continuous time evolution is interrupted whenever the potential reaches the threshold $U_T = 30$ mV. Instantaneously, the membrane potential is set to a value normal-distributed around the refractory potential $U_F = -15$ mV. In addition the neuron fires a spike. To account for the latter the time t_i^0 is updated and the memory function $G_i(\Delta t_i/t)$ is set to the value 1 [see (1)]. The overall behaviour is described formally

$$\text{if } U_i(t) \geq U_T \text{ then } \begin{cases} t_i^0 = t, \\ U_i(t) = U_F + \xi(t), \\ G_i(\Delta t_i/\tau) = 1, \end{cases} \quad (7)$$

where $\xi(t)$ [see (2) and (3)] induces a distribution of the refractory potential.

A most important parameter of the network dynamics is the coupling strength ω in (2) which describes the effect of axonic currents on the membrane potentials U_i . In Buhmann and Schulten (1986) the coupling constant had been expressed in terms of all other neural parameters such that the resulting neural dynamics neither becomes completely quiescent nor like epileptic hyperactivity. In the present investigation suitable values of ω depend also on the noise level. This issue will be discussed further below.

3 Behaviour of a Single Neuron

Before we investigate the behaviour of many interacting neurons in a network we consider the membrane potential of a single neuron excited solely by Gaussian white noise (Goel and Richter-Dyn 1974; Holden 1976; Sampath and Srinivasan 1977). This situation is described by the Langevin equation

$$\frac{dU_i}{dt} = -\frac{U_i}{T_R} + \varrho[\Delta t_i] \frac{\eta}{\sqrt{T_R/2}} \xi(t), \quad (8)$$

i.e. (2) with the afferent excitation $A_i(t)$ omitted.

If the neuron rests in the sensitive state and the potential dynamics is not affected by the factor $\varrho[\Delta t_i]$, i.e. $\varrho[\Delta t_i] = 1$, (8) corresponds to an Ornstein-Uhlenbeck process with absorbing boundary conditions. The corresponding Fokker-Planck equation for the time-dependent distribution $p(U_i, t)$ of U_i values is given by

$$\frac{\partial}{\partial t} p(U_i, t) = \frac{\eta^2}{T_R} \frac{\partial}{\partial U_i} \left(\frac{\partial}{\partial U_i} + \frac{U_i}{\eta^2} \right) p(U_i, t). \quad (9)$$

Equation (9) with absorbing boundary condition $p(U_T, t) = 0$ is solved by parabolic cylindrical or Weber functions (Goel and Richter-Dyn 1974). Without the boundary condition the equilibrium distribution is given by a Gaussian distribution

$$p_0(U_i) = \frac{1}{\sqrt{2\pi\eta^2}} \exp\left(-\frac{U_i^2}{2\eta^2}\right). \quad (10)$$

The equilibrium distribution of the membrane potential which evolves according to (6)–(8) must be determined numerically. In Fig. 1 we compare the resulting distribution with the Gaussian distribution (10). Figure 1 shows that in case of a low spontaneous spike rate ν_s ($\nu_s = 4 \text{ s}^{-1}$) the exact distribution deviates only little from the Gaussian distribution (10).

The spontaneous spike rate of a neuron can then be determined from the inverse mean first passage time τ_{FP} for the Ornstein-Uhlenbeck process (9) to reach

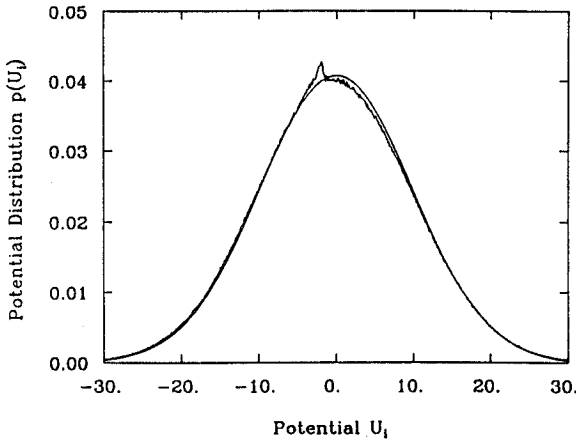


Fig. 1. The equilibrium distribution of a membrane potential evaluated according to (8) is compared to a Gaussian distribution with variance $\eta = 10$ mV. The differences between the two distributions, i.e. the peak at $U_i = -3$ mV and the small shift to negative potentials, are due to the deterministic dynamics during the refractory period and due to the discontinuous evolution when the potential reaches the threshold

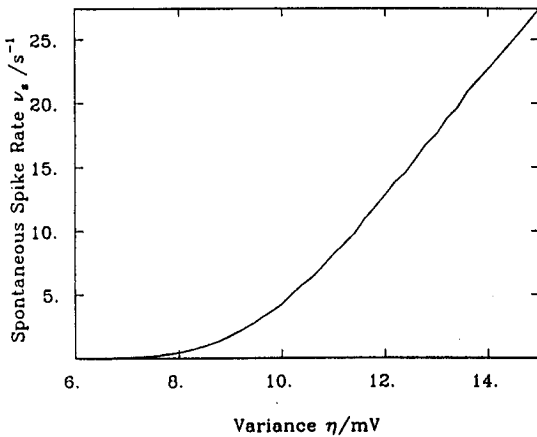


Fig. 2. Dependence of the spontaneous spike rate v_s of a neuron on the variance of the membrane fluctuations. The spike rate v_s has been evaluated according to (11) and (12)

the threshold. The result is

$$v_s = \frac{1}{\tau_{FPT} + T_{F_a} + 2T_{F_r}} \quad (11)$$

with (see Schulten et al. 1981)

$$\tau_{FPT} = \int_{U_0}^{U_T} \left[\frac{\eta^2}{T_R} p_0(U) dU \right] \int_{-\infty}^U p_0(X) dX. \quad (12)$$

Figure 2 shows the dependence of the spontaneous spike rate v_s on the noise amplitude. An increase of the noise amplitude increases the spontaneous spike rate. If the noise is too weak ($\eta < 7$ mV) a neuron does not reach the threshold and the spontaneous spike rate

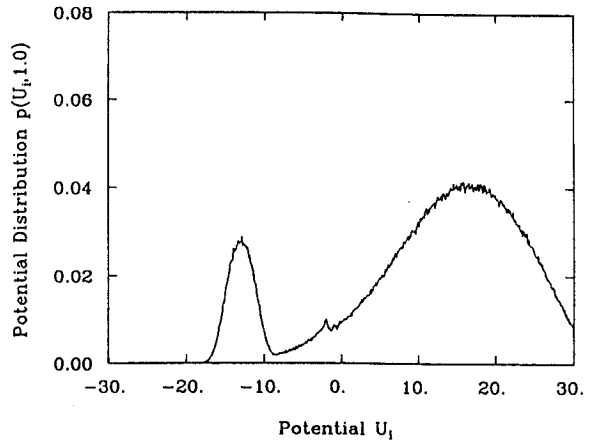


Fig. 3. Time-dependent distribution of the cellular potentials after a single receptor input at $t=0$ are shown 1 ms after input spike. During the following 3 ms a second maximum of the distribution grows at the refractory potential U_F and relaxes to the resting potential U_T . After 10 ms the distribution of the cellular potential approaches again the equilibrium. For the receptor-neuron coupling ωR_{ij} a value of 30 mV/ms has been assumed

vanishes. In the subsequent simulations η assumes a value 10 mV which corresponds to a spontaneous firing rate $v_s \approx 4$ s⁻¹.

4 Reaction of a Single Neuron to a Non-Stochastic Receptor Input

We will now investigate the influence of a receptor input on a single noisy neuron. The receptor fires a spike at $t=0$ and is then quiescent. This process is described by the Langevin equation

$$\frac{dU_i}{dt} = -\frac{U_i}{T_R} + q[\Delta t_i] \left(\omega R_{ij} \exp\left(-\frac{t}{T_U}\right) + \frac{\eta}{\sqrt{T_R/2}} \xi(t) \right). \quad (13)$$

At $t < 0$, before the receptor fires, the potential assumes the equilibrium distribution in Fig. 1. After the receptor spike, the potential distribution shifts towards the threshold potential. The firing of neuron i becomes then more probable and as a result a second maximum of $p(U_i, t)$ develops around the refractory value U_F . The new peak shifts to the resting potential within the refractory period $T_{F_a} + 2T_{F_r}$. This behaviour is illustrated in Fig. 3.

The probability $v(t)$ that the neuron fires at time t after a receptor spike is presented in Fig. 4. $v(t)$ rises from its equilibrium value v_s and within 5 ms relaxes again towards this value. The time dependence of $v(t)$ depends on the strength of the receptor-neuron connection ωR_{ij} . Weak coupling between receptors and

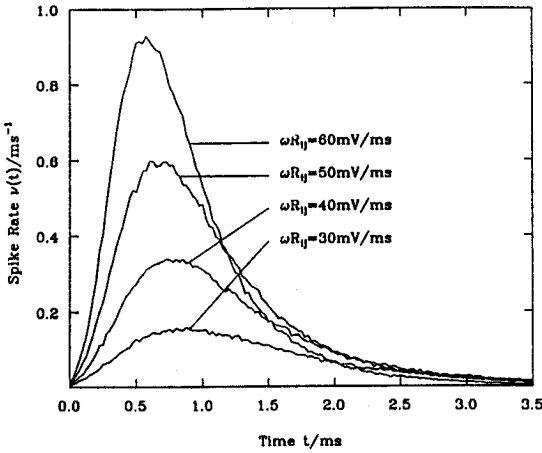


Fig. 4. Time dependence of the firing probability after a receptor spike at $t=0$

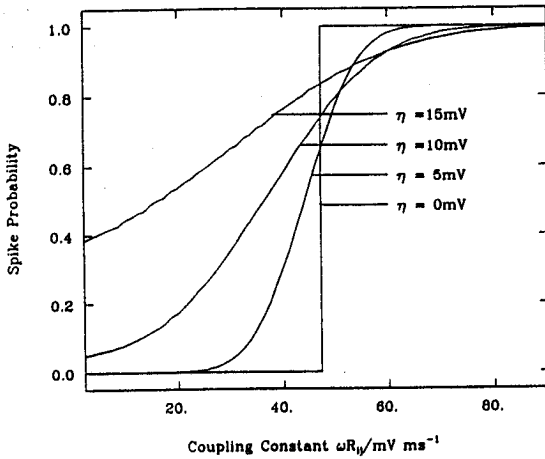


Fig. 5. Dependence on the coupling strength of the total probability of a neuron to fire within 5 ms after it received a receptor spike

neurons yields only a small instantaneous increase of $v(t)$. In case of strong coupling the receptors definitely fire within the time period T_U and, hence, $v(t)$ exhibits a strong instantaneous increase.

If one neglects the spontaneous spike rate the total probability of a neuron to fire after a receptor spike is $\int_0^{5 \text{ ms}} v(t) dt$. The dependence of this probability on the coupling constant ωR_{ij} is presented in Fig. 5 for four different noise levels.

In case of weak coupling ($\omega R_{ij} < 20 \text{ mV/ms}$) the probability for firing after a receptor spike is small. In case of strong coupling ($\omega R_{ij} > 60 \text{ mV/ms}$) firing is certain. The noise level regulates the gain of the firing probability. Strong noise ($\eta = 15 \text{ mV}$) yields a gradual increase of the total firing probability with increasing coupling constant. In the deterministic limit of vanish-

ing noise ($\eta = 0 \text{ mV}$) the response of a neuron to an afferent spike is that of a threshold automaton, i.e. the firing probability rises discontinuously at $\omega R_{ij} = 47 \text{ mV/ms}$. This behaviour demonstrates that the noise amplitude η plays the same role as temperature in stochastic spin-like models, e.g. in the Boltzmann machine (Hinton et al. 1984).

5 Synaptic Plasticity in Stochastic Neural Networks

It has been suggested by many authors (see for example Kohonen 1984 and references therein; Grossberg 1972) that neural networks which store information non-locally in synaptic connections are capable of associative memory. For the purpose of such information storage we introduce plasticity of the synapses. The synaptic changes in our model evolve on a time scale of 0.1–0.3 s. The changes depend on the synchronicity or asynchronicity of the pre- and postsynaptic spikes according to the Hebbian rule. Experimentally, there exist only vague and indirect indications of fast varying synapses derived from the analysis of EEG oscillations (Freeman 1977). Recently, Kelso et al. (1986) have observed, albeit on a longer time scale, synaptic efficacies altered by synchronous pre- and postsynaptic activity.

In addition to the Hebbian rule we postulate that synaptic changes occur only when the mean spike frequency considerably exceeds the spontaneous spike rate. If both neurons satisfy this condition their synapse can be strengthened in case of synchronous firing. If only the presynaptic neuron fires with a high enough spike rate the synapse $S_{ik}(t)$ is weakened after each presynaptic spike. The mean spike rate is given by the expression

$$\bar{G}_i(t) = \frac{1}{T_G} \sum_{v=0}^{\infty} \Theta(t-t_v) \exp\left(-\frac{t-t_v}{T_G}\right). \quad (14)$$

The time constant T_G assumes values between 50 ms and 200 ms. For a periodic spike train which is limited by the refractory period and, therefore, has a minimum spike interval $T_F = T_{F_a} + 2T_{F_r}$, the maximum value of $\bar{G}_i(nT_F)$ in the limit $T_F \ll T_G$ is $(T_F)^{-1}$.

In our model the strength $S_{ik}(t)$ of the synapse connecting neuron k to neuron i is governed by the equation

$$\frac{dS_{ik}}{dt} = \begin{cases} -\frac{S_{ik}(t) - S_{ik}(0)}{T_S} + \Omega_k \eta_k \left(\frac{\Delta t_k}{T_M}\right) \kappa(G_i, G_k), & \text{if } S_u \geq |S_{ik}| \geq S_l; \\ -\frac{S_{ik}(t) - S_{ik}(0)}{T_S}, & \text{else.} \end{cases} \quad (15a)$$

$\kappa(G_i, G_k)$ and Ω_κ are defined as follows

$$\kappa(G_i, G_k) = \begin{cases} 1, & \text{if } G_i > G_k > e^{-1} \wedge \bar{G}_i > v_e \wedge \bar{G}_k > v_e; \\ -1, & \text{if } G_k > e^{-1} > G_i \wedge \bar{G}_i < v_i \wedge \bar{G}_k > v_e; \\ 0, & \text{else} \end{cases} \quad (15b)$$

$$\Omega_\kappa = \begin{cases} \Omega \frac{1+\kappa}{2} + \Omega\alpha \frac{1-\kappa}{2}, & \text{if } S_{ik} \in [0.9S_u, S_u] \vee S_{ik} \in [-0.1S_u, -S_l]; \\ \Omega, & \text{else.} \end{cases} \quad (15c)$$

Equation (15a) holds both for excitatory and inhibitory synapses and conserves the sign of the synaptic strength $S_{ik}(t)$. The first term describes the relaxation of the synaptic efficacies towards their initial values and accounts for a gradual loss of stored information ($T_S \approx 2$ s). The second term describes how pre- and postsynaptic activities change the synaptic strength. The effect of this term decays exponentially in time after each presynaptic pulse as described by $G_k \left(\frac{\Delta t_k}{T_M} \right)$. The short decay time $T_M = 2.5$ ms implies that the synaptic efficacy is affected only by a synchronous presynaptic pulse. The function $\kappa(G_i, G_k)$ switches between excitatory growth, inhibitory decrease and passive relaxation of the synapses. If the mean rate of postsynaptic spikes $\bar{G}_i(t)$ exceeds the threshold $v_e \approx 40 \text{ s}^{-1}$, i.e. a rate much larger than the rate of spontaneous activity v_s , and if the presynaptic neuron fires before the postsynaptic neuron, then the synapse $S_{ik}(t)$ is strengthened. Conversely, the synaptic strength decrease if the presynaptic neuron k has fired but the mean postsynaptic activity $\bar{G}_i(t)$ is below $v_i \approx 1 \text{ s}^{-1}$, i.e. below a rate much smaller than the mean firing rate v_s . In all other cases the synaptic strength is not affected by the second term in (15a).

Ω_κ defined as in (Buhmann and Schulten 1986) models a hysteresis behaviour which guarantees that a newly learned pattern does not destroy the patterns already stored in a network. This factor in (15a) slows down changes of those synaptic efficacies $S_{ik}(t)$ which have reached the upper saturation values S_u and $-S_l$ for excitatory and inhibitory synapses, respectively. The parameter α appearing in the definition (15c) of Ω_κ assumes a value below 0.1 ($0 < \alpha < 0.1$).

6 Noise-Induced Synaptic Plasticity

A simple microcircuit involving a single receptor and two neurons can demonstrate the mechanism of synaptic changes which are induced by noise of the membrane potential. This microcircuit is presented in

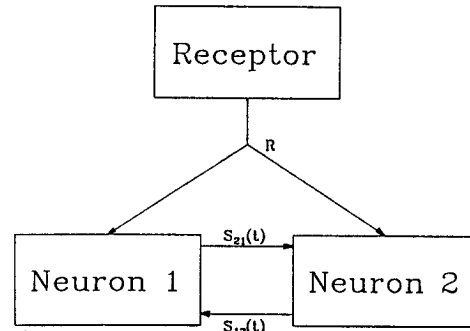


Fig. 6. Microcircuit of one receptor and two neurons to test the dependence of the synaptic dynamics on the noise level. The strength of the receptor-neuron coupling ωR has been kept constant during the simulations. The neuron-neuron connections S_{21} and S_{12} change according to the dynamics described in Sect. 5

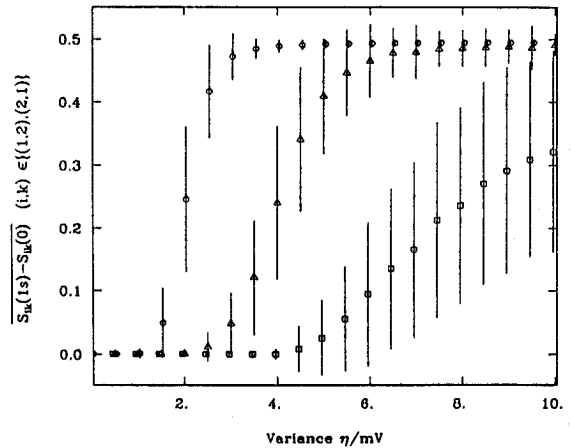


Fig. 7. The deviation of the average synaptic strength from the initial values, i.e. the quantity $(S_{21}(1s) + S_{12}(1s))/2 - S(0)$, is shown as a function of the noise level η . Strong coupling ($\omega R = 40 \text{ mV/ms}$) leads to a saturation of the synaptic strength if the noise level exceeds 4 mV (\circ). For a coupling constant $\omega R = 35 \text{ mV/ms}$ (Δ) the necessary noise level for saturating synaptic growth increases to $\eta = 6 \text{ mV}$. A coupling of $\omega R = 30 \text{ mV/ms}$ (\square) yields no saturation of the synaptic growth within one second. The synapses which are not saturated at the upper boundary S_u experience strong fluctuations as the error bars show

Fig. 6. The frequency of the receptor spikes lies in the interval $[50 \text{ s}^{-1}, 80 \text{ s}^{-1}]$. Initially, the neurons are coupled only weakly by two synapses with strengths S_{21} and S_{12} . These synapses evolve according to the dynamics discussed in Sect. 5. Since the two neurons receive input from the same receptor unit their afferent activities are completely synchronous and the Hebbian rule should induce a strong excitatory synaptic interaction. In fact, both S_{21} and S_{12} starting from positive values $S(0)$ increase in time. However, the evolution of these synaptic efficacies depend on the strengths of the neuron-receptor coupling ωR as well as on the noise amplitude η . This dependence is illustrated in Fig. 7. This figure shows that an increase of η is accompanied

by an increase of the mean synaptic strength $(S_{12} + S_{21})/2$ reached after 1 s of activity. A coupling $\omega R = 40$ mV/ms between receptor and neuron yields saturated synapses between the two neurons if the noise amplitude η exceeds 4 mV. In the limit of deterministic dynamics ($\eta = 0$ mV) the two neurons never reach the threshold potential and, therefore, cannot strengthen their synaptic connections. Obviously, the two neurons detect the synchronous excitation presented by the receptor only if noise provides a sufficiently high level of attention. In the case of weaker coupling, i.e. $\omega R = 35$ mV/ms and $\omega R = 30$ mV/ms synaptic growth can still be induced by noise, however, it becomes much more unreliable as demonstrated by the large error bars in Fig. 7.

Table 1. Network Parameters

| <i>Constants of the fast dynamics</i> | |
|---------------------------------------|--|
| $T_U = 1.0$ ms | time constant of a neural spike |
| $T_R = 2.5$ ms | relaxation time of membrane potential |
| $T_{F_a} = 5.0$ ms | absolute refractory period |
| $T_{F_r} = 2.5$ ms | relative refractory period |
| $T_I = 20.0$ ms | time between two presentations of pattern |
| $R/S \approx \sqrt{N}$ | coupling ratio between receptors and neural network |
| $U_T = 30$ mV | threshold potential |
| $U_0 = 0$ mV | resting potential |
| $U_F = -15$ mV | refractory potential |
| $\eta = 10$ mV | noise level |
| $v_s = 4$ s ⁻¹ | spontaneous spike rate |
| $\omega R_{ij} = 45$ mV/ms | coupling constant |
| <i>Constants of the slow dynamics</i> | |
| $T_M = 2.5$ ms | coincidence interval of two spikes |
| $T_S \approx 2.0$ s | synaptic relaxation |
| $T_G \approx 0.2$ s | time scale for averaging spike frequency |
| $\Omega = 1/(300$ ms) | time scale of synaptic changes |
| $\alpha \approx 0.1$ | hysteresis factor |
| $S_u = 1.7S$ | maximal synaptic value |
| $S_i = 0.01S$ | minimal synaptic value |
| $N \approx 150$ | number of synapses per neuron |
| $S \approx 60$ | synaptic strength (arbitrary units) |
| $v_e \approx 40$ s ⁻¹ | minimal spike rate for excitatory synaptic growth |
| $v_i \approx 1$ s ⁻¹ | maximal spike rate for inhibitory decrease of synapses |

7 Behaviour of a Network with Receptor Input

In this section we will demonstrate that a neural network with stochastic fluctuations of its cellular potentials shows associative properties which are superior to those of a network without noise. The ability for associative storage originates from the

ability of a network to change its connectivity structure. For a demonstration we start a network with a homogeneous, i.e. information free, connectivity structure with an equal number of excitatory and inhibitory synapses. Such network stores a pattern presented by the receptors and associatively reconstructs the original pattern even if only an incomplete or disturbed pattern is presented. This ability is demonstrated below through a series of computer simulations. Further functions of the deterministic model network (Buhmann and Schulten 1986) like the abstraction of a prototype pattern from a series of patterns or the learning of more than a single pattern have been confirmed for the stochastic network as well (Buhmann 1987), but will not be demonstrated.

The computer simulations followed exactly our previous procedure for networks without noise (Buhmann and Schulten 1986). The simulations involve three different stages. During a first stage the neural network has to learn the pattern *brain* presented through the receptors with a frequency of 50 s⁻¹. This frequency is actually 20 times smaller than that employed in (Buhmann and Schulten 1986) for a noiseless network. The first (learning) stage lasts between 0.3 and 1.0 s. Depending on the difficulties assumed during the learning task, i.e. if strong noise disturbs the synchronicity of the presented pattern, the network needs to learn for a longer time than, say, in the case of a receptor pattern free of background noise.

During the second stage the receptors are inactive and the electrical activity of the network relaxes to the resting level. The neurons fire only spontaneous spikes. The second stage is introduced to separate the learning stage from the following test period. This interval of quiescence suppresses possible memory effects in the activity of the network. Therefore, any associative abilities acquired by the network can only be based on changed synaptic connectivity.

During the third stage, the test stage, the network has to process incomplete or disturbed patterns and compare them with the information in storage. For example, the receptors present the test pattern *bra n* which differs from the originally learned pattern by the missing letter *i*. The patterns presented during the learning stage and the test stage are shown in Fig. 8.

7.1 Learning and Association of a Pattern

The first simulation should confirm the associative properties which have been demonstrated already for the deterministic network, i.e. the association of a test pattern to a pattern previously learned. During the training session of the network all receptors representing *brain*, the pattern to be learned, fire synchronously

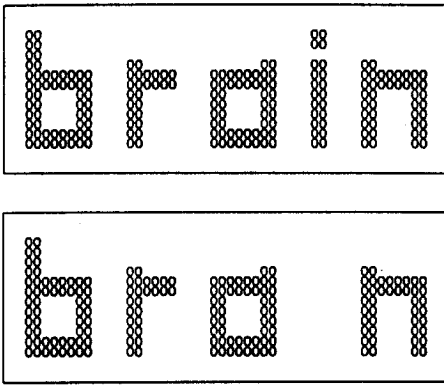


Fig. 8. Learning pattern and test pattern employed in our simulations. The mark \circ indicates that the receptor at that position fires with a rate T_T^{-1} , i.e. the function $G_T^R(\Delta t_j^R/T_T)$ is set to 1 at this rate, and evolves according to (1) between these times

with a frequency of $\nu_i = 50 \text{ s}^{-1}$. The coupling constant ω is set to 45 mV/ms which effects the firing of about 70% of excited neurons. A homogeneous background noise with a spike rate of 10 s^{-1} is superimposed.

First we will analyse the behaviour of the network during the learning stage. Initially the synaptic strengths are selected randomly with the constraint that the sum of the afferent synaptic strengths $\sum_k S_{ik}$ vanishes. This implies that the local distribution of excitatory synapses is equal to the local distribution of inhibitory synapses.

During the learning stage, the synaptic strengths change drastically. The dynamics of the synapses reacts to coincidences of pre- and postsynaptic spikes if both neurons fire with a spike rate above ν_e . The excitatory synapses grow up to the excitatory saturation value S_m , and the inhibitory synapses decay to the saturation strength $-S_i$. At the end of the learning stage after 1 s, most of the neurons representing the dark pixels in the word brain have synchronized their firing behaviour by lateral excitatory interaction.

The network reaction to a receptor input at the end of the learning stage at 1 s is shown in Fig. 9. Only seven figure neurons have not reached the threshold within 2 ms after the presentation of the figure brain. Due to the learned excitatory connectivity between neurons representing the word brain the assembly reacts more synchronously and the fault rate – the number of pattern neurons which fail to fire – decays below 3%.

Figure 10 shows the membrane potential of neuron (15, 8) at the end of the learning stage. During the last 250 ms of the network training the time period between two spikes fluctuates slightly around 20 ms, the period of the receptor input. Figure 10 demonstrates also that the electrical activity of the model neurons resembles in its noisy appearance neurophysiological recordings from nerve cells in brains.

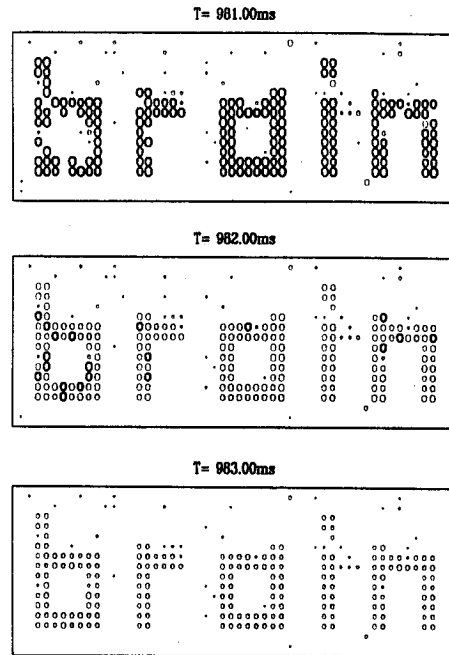


Fig. 9. The activity function $G_A(\Delta t_j/T_M)$ is shown for a trained network. At the end of the learning stage nearly all excited neurons have synchronized their firing behaviour and reach the threshold (Pattern brain presented at 980 ms)

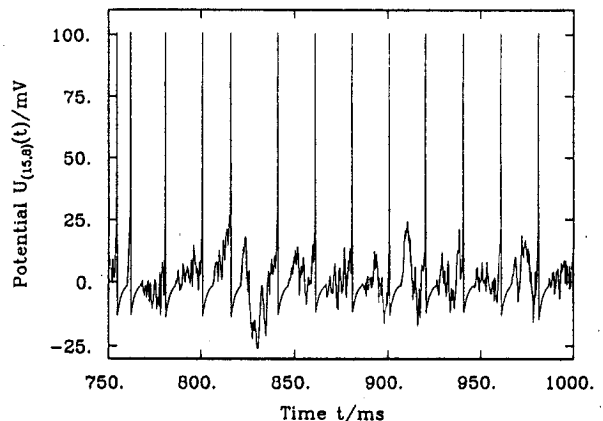


Fig. 10. The membrane potential $U_{(15,8)}(t)$ of neuron (15, 8), a neuron of the letter r, is shown during the last quarter of the learning stage. The synchronization effected by pattern learning is confirmed by the regular and periodic spike train of a single neuron. The firing is shown by the spikes of 100 mV, which are included in this diagram whenever the membrane potential reaches the threshold

The success of the learning session is documented in Fig. 11. The incomplete test pattern *bra n*, shown in Fig. 8, is associatively restored when presented to the network. The neurons representing the missing letter *i* react with a delay-time of 1–3 ms. All members of the group of figure neurons for the letter *i* fire nearly synchronously with the other members.

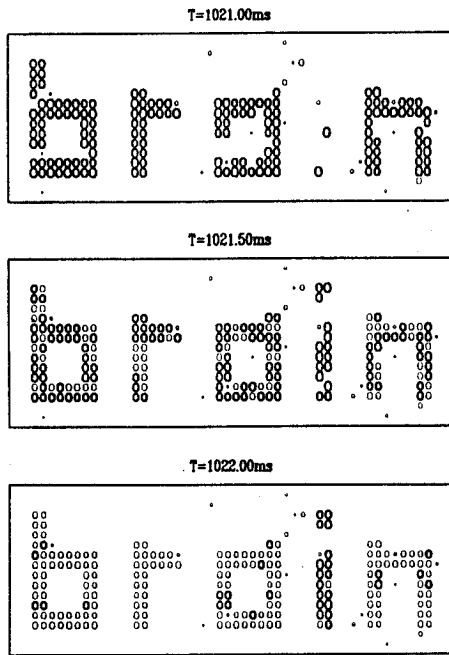


Fig. 11. Network activity during the association task described in Sect. 7.1 (Pattern *bra n* presented at 1020 ms)

7.2 Changes in the Synaptic Connectivity

The synchronization of the neural activity shown in the Figs. 9 and 10 and the associative abilities demonstrated by Fig. 11 can be understood if we investigate the synaptic structure established after the learning session. Figure 12a and b shows the afferent and efferent synapses of the reference neuron (37, 4) representing the dot of the letter *i*. The size of the squares and the diamonds encodes the growth of the excitatory and the inhibitory synapses, respectively. The size of the symbols scales proportionally to the difference $S_{ik}(t) - S_{ik}(0)$ between the actual synaptic strength and the strength of the initial synapse. All the neurons representing the figure brain have developed excitatory or inhibitory synapses to the reference neuron, both saturating at the values S_u or $-S_b$, respectively. During an association task the excitatory synapses support the firing of the reference cell, whereas the inhibitory synapses do not prevent the reference cell from firing. Afferent synapses of the reference cell coming from background neurons, apart from small fluctuations, rest at the initial synaptic strength. The reason for this behaviour is that the synaptic growth term in (15a) is proportional to the presynaptic activity $G_k(\Delta t_k/T_U)$ which vanishes for a background-neuron.

The efferent synapses of the neuron (37, 4) have nearly the same structure as the afferent synapses. Connections leading to a figure neuron are grown to the excitatory saturation value. The neurons belonging to the figure *brain* are connected by symmetric syn-

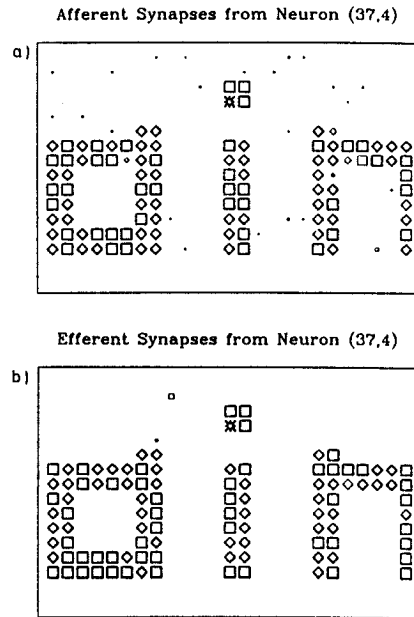


Fig. 12a and b. The afferent and efferent synapses of the reference neuron (37, 4). The quantity $S_{ik}(t) - S_{ik}(0)$ is presented by the size of the square and diamond symbols. The diamond \diamond denotes inhibitory synapses, the square \square denotes excitatory synapses. The connections to background neurons are found to experience small fluctuations around the initial value. The boldface symbols indicate synapses which have reached the upper saturation boundaries $-S_b$ and S_u

apses because there exists no rigid time order for firing. A difference between afferent and efferent synapses exists for the figure-background connections. The synapses which start from the reference cell and lead to a background cell have decayed to the lower saturation boundaries S_b and $-S_u$ for excitatory and inhibitory synapses. This change of the synaptic structure effects a suppression of background activity, even in the case that the network has learned more than a single pattern.

The influence of strong disturbing noise on the learning process has been confirmed by various computer simulations. The efferent and afferent synapses have only partially reached their excitatory saturation values. In contrast to the synaptic structure of the figure neuron (37, 4) shown in Fig. 12 some efferent and afferent synapses of a disturbed cell are still developing at the end of the learning stage due to lack in coincidences between pre- and postsynaptic activity. Neurons with such rudimentary synaptic structure cannot be excited during an association task and, hence, give rise to faults in the associated pattern.

7.3 Learning of a Sparsely Presented Pattern

In the simulations discussed in the previous sections the receptors always present the whole pattern synchron-

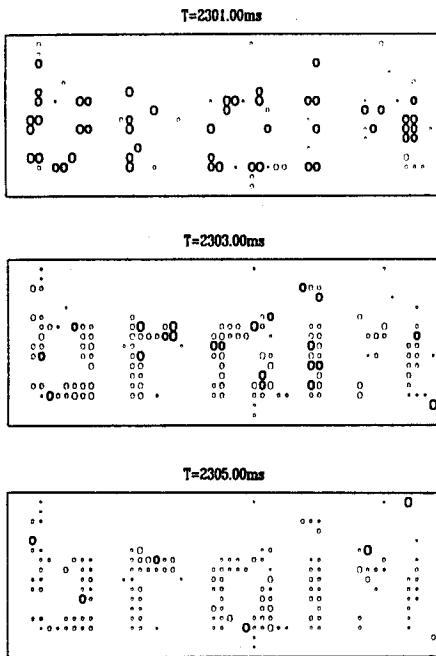


Fig. 13. Activity function of a network during the learning stage when at every presentation only 50% of the pattern receptors are excited

ously. This is a sufficient but not a necessary condition for learning. Building of neural assemblies only requires that the synaptic changes due to synchronous spike events prevail over the decrease of the synaptic strength caused by relaxation or asynchronicity. In the following simulation only 50% of the receptors belonging to the pattern presented by the receptors fire at the times the pattern is presented to the network. At these instances the network sees only half of the pattern pixels and has to reconstruct the complete pattern from the detected spike coincidences. This is possible because at different presentations the identity of the receptors presenting the pattern differs such that after several presentations the network has had the chance to detect coincidences between any pairs of pattern receptors. Due to fluctuations of the membrane potential which increases the sensitivity of the neurons the network can indeed store the sparsely presented pattern. The training session has to be prolonged, though. The factor by which the learning session has to be extended is proportional to the inverse square of the synchronization degree defined as the ratio of synchronously firing receptors to all pattern receptors. For example, if only half of the brain receptors fire synchronously the network has to learn four times longer than in the case of full synchronization of the receptors.

Figure 13 shows the network activity after presentation of the pattern by the receptors. An electrophysi-

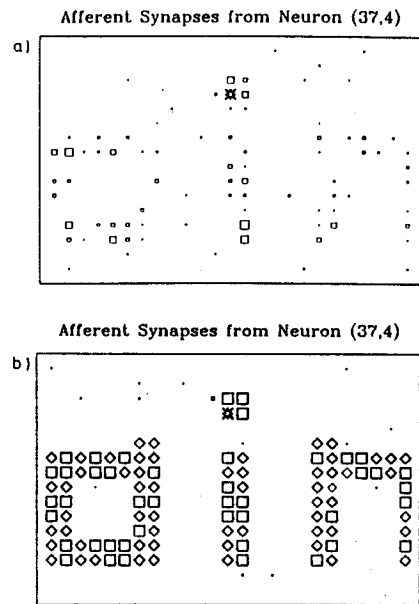


Fig. 14a and b. Evolution of the synapses during the learning stage. The pattern brain is sparsely presented (only 50% at each presentation). The learning stage had been extended four times because of the reduced probability for spike coincidences. a $t = 1000$ ms; b $t = 3668$ ms

ological experiment which detects the neural spikes will see only the activity of those neurons denoted by the bold-faced circles (O). The figure demonstrates that, if only the millisecond dynamics is analyzed, it is very hard to reveal from the latter the whole pattern.

In Fig. 14 we present the evolution of the synapses afferent to neuron (37,4) [the dot of the letter *i*]. During the learning stage which lasts 3.7 s the network has detected the hidden correlations which exists among the pattern pixels and has built up a synaptic structure which contains the information of the whole pattern *brain*. Figures 13 and 14 prove that the synchronization of *all* the receptors in each presentation is not a necessary condition for learning. For associative pattern storage the synaptic growth due to spike coincidences must prevail, however, over the relaxation of synapses.

8 Structure of the Phase-Space of the Network

Equation (2) approximates the electrophysiological behaviour of real neurons evolving on the millisecond time scale. The total membrane potential of a network

$$EEG(t) = \sum_i \left(U_i(t_i) + 80 \text{ mV} \cdot G_i \left(\frac{\Delta t_i}{T_U} \right) \right) \quad (16)$$

can also be given a physiological interpretation, namely that of the EEG-signal (electro-encephalogram-

signal) of the neural system. The membrane potentials $U_i(t)$ below the threshold and the action potentials contribute to this signal. The action potentials are described by the functions $G_i(\Delta t_i/T_U)$ which, for this purpose, are scaled by the factor 80 mV corresponding to the peak potential difference developed during firing. The stochastic behaviour of the EEG-signal shows a significant dependence on the internal connectivity of the neural system. In the following analysis the neural network is stimulated by noisy receptors with a mean spike rate of 40 s^{-1} . No systematic pattern is presented to the network.

We will investigate the EEG-signal of a neural assembly and the membrane potential of a single neuron in an assembly for three different synaptic structures. First we consider a network which has no synaptic connections. The second network considered contains neurons connected by synapses which are weak in comparison to the receptor-neuron connections ($|R_{ik}|/|S_{ik}(t)| \gg 1$). The third structure contains neurons which have connections with nearly the same strengths as the receptor-neuron connections ($|R_{ik}|/|S_{ik}(t)| \approx 1$). The synapses in the latter two structures are completely saturated after a previous learning stage and separate the network into two sets of neurons, a figure assembly and a background assembly. The neurons belonging to the background assembly after the learning process are not interacting and are inhibited by the assembly of figure neurons.

In this investigation we want to test in how far learning and the concurrent development of an ordered synaptic structure reduces the effective dimension of the phase space in which the electrical activity of a network takes place. One can imagine that the dynamics of an unstructured network explores a higher dimensional space of states than that of a structured network. In order to be unbiased in an exploration of this possibility one should drive the system with stochastic input since any ordered input may impose a reduction of the dimension of the network dynamics as well. The main problem for the intended analysis is a proper procedure to obtain the desired information. Such procedure has been suggested recently by Grassberger and Procaccia (1983) for the analysis of the effective dimensions of strange attractors in non-linear dynamical systems.

Following Grassberger and Procaccia (1983) we build up an n -dimensional embedding space with the n -dimensional vectors

$$\mathbf{X}^{(n)} = [f(i\Delta t), \dots, f(i\Delta t + (n-1)\tau)].$$

Δt is the sampling interval of the signal $f(t)$ which, in our case, is either the signal $\text{EEG}(t)$ or the potential $U_i(t)$. Δt ranges between 4 and 10 ms ($\Delta t = 4$ in our simulations). For the time constant τ we choose the

value 10 ms. The scaling behaviour of the correlation integral

$$C_n(r) = \lim_{N \rightarrow \infty} \sum_{\substack{i, j=1 \\ i \neq j}}^N \Theta(r - |\mathbf{X}_i^{(n)} - \mathbf{X}_j^{(n)}|) \quad (17)$$

for small r contains the information about the phase-space density of the system, e.g. tests whether the dynamics of the system is dominated by a strange attractor. In order to obtain this information one tests whether the correlation integral exhibits a power dependence for small r

$$C_n(r) \sim r^{d(n)}. \quad (18)$$

Such power dependence is indicative of a strange attractor. The exponent $d(n)$ called correlation exponent measures the effective dimension of the phase-space filled by the trajectory $f(t)$.

In our analysis of $\text{EEG}(t)$ we cannot expect to reveal a strange attractor because the activation of the network is completely stochastic and not deterministic chaotic. Therefore, the correlation integral (17) will not exhibit a simple power dependence (18) and the correlation exponent $d(n)$ will increase with increasing embedding dimension n . Nevertheless, also a non-constant $d(n)$ should be indicative of the effective dimension of the network dynamics. In fact, we found that $d(n)$ depends significantly on the synaptic connectivity of the neurons. Strongly coupled neurons with excitatory interactions yield a lower correlation exponent $d(n)$ than weakly coupled or uncoupled neurons.

The correlation integral of a neural potential produced by a pattern neuron in a strongly coupled network is shown in Fig. 15. The embedding space has dimension $n = 10$. The slope of the straight line yields the correlation exponent. Obviously, the correlation integral does not obey a power law. The exponent for small distances r differs from the exponent for inter-

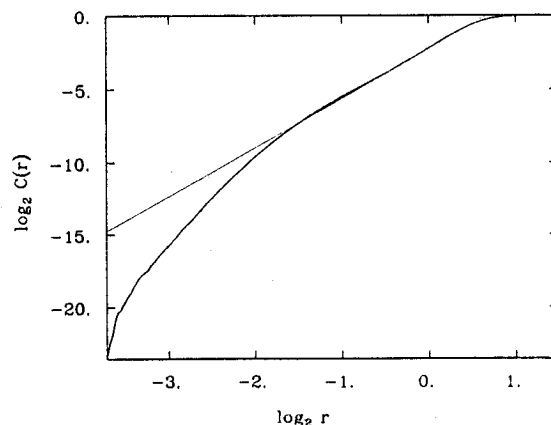


Fig. 15. The correlation integral $C(r)$ as a function of r . In the double logarithmic plot the slope of the straight line determines the correlation exponent d

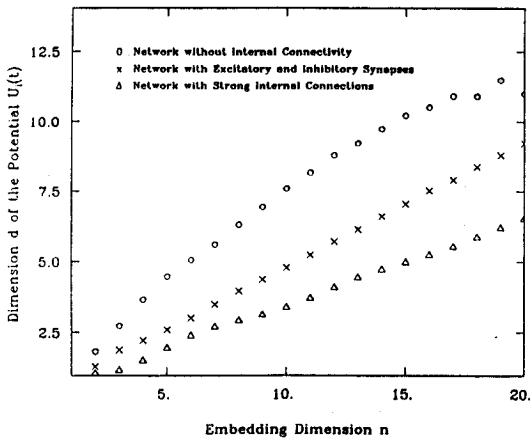


Fig. 16. The correlation exponent $d(n)$ of the membrane potential produced by a single assembly-neuron is shown for various dimensions n of the embedding space. Depending on the internal connectivity of the network the firing behaviour shows a lower correlation exponent and, therefore, less stochasticity

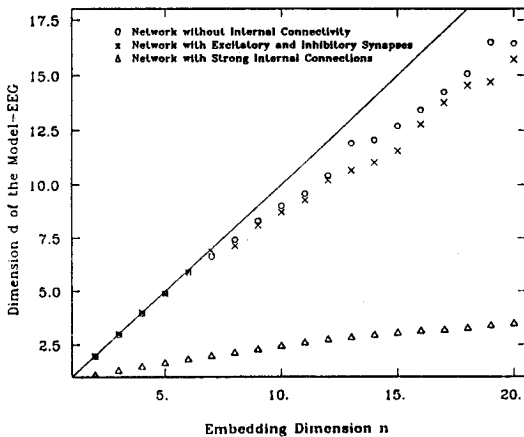


Fig. 17. The correlation exponent $d(n)$ of the EEG-signal (16) for three different networks

mediate r . The two separated ranges can be interpreted as an ordering phenomenon for greater distances r . The firing of the assembly-neuron obeys the dynamics of the assembly and is not dominated by the stochastic receptor input. The reaction of the whole assembly reduces the degrees of freedom which are found in a network without synaptic interaction between the neurons. For small distances r the fluctuations around the attracting point $X=(0, \dots, 0)$, an n -dimensional representation of the resting potential U_0 , show a completely stochastic behaviour.

In Fig. 16 the correlation exponent $d(n)$ of the membrane potential of a figure assembly-neuron, i.e. a neuron which codes for the figure learned and not for the background, and the corresponding dimension n of the embedding phase-space are shown. It is significant that the network with strong internal connections

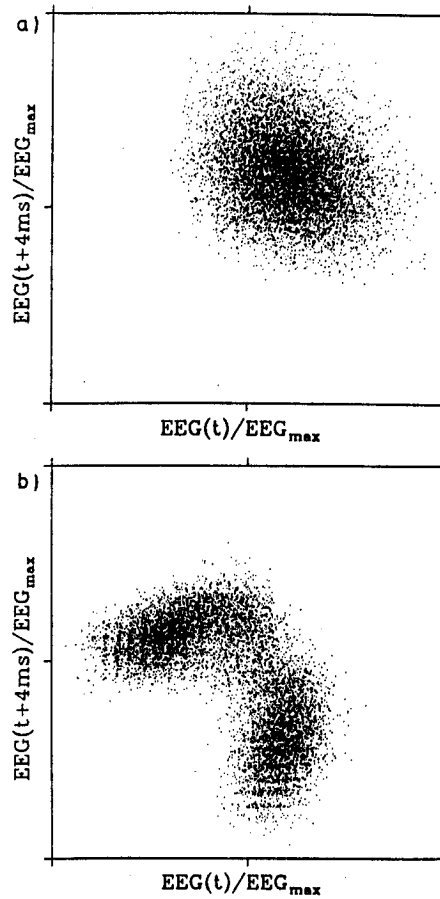


Fig. 18a and b. Poincaré maps of the EEG-signal produced a) for an unconnected and b) for a strongly connected network

produces a less chaotic signal with a smaller correlation exponent than the networks with weak synapses or without synapses.

The scaling behaviour of the EEG-signal produced by the strongly coupled network can also be classified into a chaotic range of the potential fluctuations and into a partially chaotic range of the network activity. This is demonstrated in Fig. 17. The reduction in dimension of the EEG-signal produced by the weakly coupled network cannot be significantly detected.

A more concrete idea of the embedding space is given by the Poincaré maps of the EEG-signals (Fig. 18a and b). A Poincaré map shows an embedding space of dimension $n=2$. With increasing strengths of the internal neural connections the Poincaré map exhibits more structure and indicates that the analysed signal is less chaotic than signals from networks without synapses.

9 Conclusion

We have presented a model neural network which mimics physiological neural systems also with respect

to stochastic fluctuations of its cell potentials. The noise does not destroy the network's ability to learn and to associatively reconstruct patterns. On the contrary, noise controls the level of activity and enables the network to associatively store and adaptively filter weak receptor inputs which would otherwise be neglected. We argue that noise is of functional importance for the nervous system. Neurons which experience electrical membrane noise or noise from synapses respond to afferent action potentials with a firing probability determined by synaptic strengths and noise level. The noise level corresponds to temperature in the neural model of Little (1974, 1975), in the Boltzmann machine (Hinton et al. 1984) and in spin glass neural network models (Amit et al. 1985, 1987). Stochasticity in neural dynamics assures that a neural network explores the necessary variety of network states. This enables the network to solve complex optimization problems as they have been described in vision research (Koch et al. 1986) or should enable invariant pattern recognition (Bienenstock 1984; Malsburg and Bienenstock 1985). The latter problem is the subject of our own current research.

In order for neural networks to explore the advantages of inherent noise the traditional rules for synaptic plasticity need to be extended. In addition to the customary Hebbian rule based on synchronicity between pre- and postsynaptic spikes a second condition for synaptic changes is introduced to protect the synaptic structure against destruction by spontaneous activity. The mean spike rates \bar{v} of the pre- and postsynaptic neurons have to be considerably above the spontaneous spike rate v_s for excitatory growth. For the decrease of the synapses the postsynaptic spike rate must be below v_s .

The analysis of cellular potentials as monitored through EEG-signals demonstrated that stochastically excited networks with strong connectivity within neural assemblies produce a less chaotic EEG-signal than networks without or with weak internal connections. Perhaps these results provide a key to the understanding of strange attractors in EEG dynamics of sleeping man discovered recently (Babloyantz 1985, 1986; Dvorak and Siska 1986).

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References

- Abeles M (1982) Local cortical circuits. Springer, Berlin Heidelberg New York
- Amit, DJ, Gutfreund H, Sompolinsky H (1985a) Spin glass models of neural networks. *Phys Rev A* 32:1007
- Amit DJ, Gutfreund H, Sompolinsky H (1985b) Storing infinite numbers of patterns in a spin glass model of neural networks. *Phys Rev Lett* 55:1530
- Amit DJ, Gutfreund H, Sompolinsky H (1987) Statistical mechanics of neural networks near saturation. *Ann Phys* 173:30-67
- Babloyantz A (1985) Strange attractors in the dynamics of brain activity. In: Haken H (ed) *Complex Systems - operational approaches*. Springer, Berlin Heidelberg New York, pp 116-122
- Babloyantz A, Destexhe A (1986) Low-dimensional chaos in an instance of epilepsy. *Proc Natl Acad Sci USA* 83:3513-3517
- Bienenstock E (1984) Dynamics of central nervous system. In: Beckmann M, Krelle W (eds) *Dynamics of macrosystems*. Springer, Berlin Heidelberg New York, pp 3-20
- Buhmann J (1987) Thesis, Technical University of Munich (in preparation)
- Buhmann J, Schulten K (1986) Associative recognition and storage in a model network of physiological neurons. *Biol Cybern* 54:319-335
- Changeux JP, Heidmann T, Patte P (1984) Learning by selection. In: Marler T, Terrace HS (eds) *The biology of learning*. Springer, Heidelberg Berlin New York, pp 115-133
- Cooper LN (1973) Proceedings of the Nobel symposium on collective properties of physical systems. Academic Press, New York, pp 252-264
- Dvorak I, Siska J (1986) On some problems encountered in the estimation of the correlation dimension of the EEG. *Phys Lett A* 118:63-66
- Ebeling W, Feistl R (1982) *Physik der Selbstorganisation und Evolution*. Akademie Verlag, Berlin
- Freeman WJ (1977) Spatial properties of an EEG Event in the olfactory bulb and cortex. *Electroencephalogr Clin Neurophys* 44:586-605
- Goel NS, Richter-Dyn N (1974) *Stochastic models in biology*. Academic Press, New York San Francisco London
- Goelet P, Castellucci VF, Schacher S, Kandel ER (1986) The long and the short of long-term memory - a molecular framework. *Nature* 322:419-422
- Grassberger P, Procaccia I (1983) Measuring the strangeness of strange attractors. *Physica* 9D:189-208
- Grossberg S (1972) Pattern learning by functional-differential neural networks with arbitrary path weights. In: Schmitt K (ed) *Delay and functional differential equations and their applications*. Academic Press, New York, pp 121-159
- Hebb DO (1949) *Organization of behavior*. Wiley, New York
- Hinton GE, Sejnowski TJ, Ackley DH (1984) Boltzmann machines: constraint satisfaction networks that learn. Tech Rep No CMU-CS-84-119 Pittsburgh, PA: Carnegie-Mellon Univ
- Holden AV (1976) Models of the stochastic activity of neurones. Springer, Berlin Heidelberg New York
- Hopfield JJ (1982) Neural networks and physical systems with emergent collective computational abilities. *Proc Natl Acad Sci USA* 79:2554-2558
- Hopfield JJ (1984) Neurons with graded response have collective computational properties like those of two-state neurons. *Proc Natl Acad Sci USA* 81:3088-3092
- Kandel ER, Schwartz JR (1982) Molecular biology of learning: modulation of transmitter release. *Science* 218:433-443
- Kelso SR, Ganong AH, Brown TH (1986) Hebbian synapses in *Hippocampus*. *Proc Natl Acad Sci USA* 83:5326-5330
- Kinzel W (1985) Learning and pattern recognition in spin glass models. *Z Phys B - Condensed Matter* 60:205-213

- Koch C, Marroquin J, Yuille A (1986) Analog "neuronal" networks in early vision. *Proc Natl Acad Sci USA* 83:4263-4267
- Kohonen T (1984) *Selforganization and associative memory*. Springer, Berlin Heidelberg New York
- Kohonen T, Oja E, Lehtiö P (1981) Storage and processing of information in distributed associative memory systems. In: Hinton G, Anderson JA (eds) *Parallel models of associative memory*. Lawrence Erlbaum Associates, Hillsdale, New Jersey
- Little WA (1974) The existence of persistent States in the brain. *Math Biosci* 19:101-120
- Little WA, Shaw GL (1975) A statistical theory of short and long term memory. *Behav Biol* 14:115-133
- Malsburg Cvd (1981) The correlation theory of brain function. Internal report 81/2, Dept. Neurobiologie, MPI f. Biophysikalische Chemie, Göttingen
- Malsburg Cvd (1985) Nervous structures with dynamical links. *Ber Bunsenges Phys Chemie* 6:703-710
- Malsburg Cvd, Bienenstock E (1985) Statistical coding and short-term synaptic plasticity: a scheme for knowledge representation in the brain. In: Bienenstock E, Fogelman Soulié F, Weisbuch G (eds) *Disordered systems and biological organization*. Springer, Berlin Heidelberg New York, pp 247-272
- Rumelhart DE, Hinton GE, Williams RJ (1986) Learning representations by back-propagating errors. *Nature* 323:533-536
- Sampath G, Srinivasan SK (1977) Stochastic models for spike trains of single neurons. Springer, Berlin Heidelberg New York
- Schulten K, Schulten Z, Szabo A (1981) Dynamics of reactions involving diffusive barrier crossing. *J Chem Phys* 74:4426-4432
- Shaw G, Vasudevan R (1974) Persistent states of neural networks and the random nature of synaptic transmission. *Math Biosci* 21:207

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