INFLUENCE OF NOISE ON THE BEHAVIOUR OF AN AUTOASSOCIATIVE NEURAL NETWORK

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ABSTRACT

Recently, we simulated the activity and function of neural networks with neuronal units modelled after their physiological counterparts[1,2]. Neuronal potentials, single neural spikes and their effect on postsynaptic neurons were taken into account. The neural network studied was endowed with plastic synapses. The synaptic modifications were assumed to follow Hebbian rules, 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 time scale of the synaptic plasticity was that of mental processes, i.e. a tenth of a second as proposed by v.d. Malsburg[3]. In this contribution we extend our previous study and include random fluctuations of the neural potentials as observed in electrophysiological recordings[4]. We will demonstrate that random fluctuations of the membrane potentials raise the sensitivity and performance of the neural network. The fluctuations enable the network to react to weak external stimuli which do not affect networks following deterministic dynamics. We argue that fluctuations and noise in the membrane potential are of functional importance in that they trigger the neural firing if a weak receptor input is presented. The noise regulates the level of arousal. It might be an essential feature of the information processing abilities of neuronal networks and not a mere source of disturbance better to be suppressed. We will demonstrate that the neural network investigated here reproduce the computational abilities of formal associative networks[5,6,7].

INTRODUCTION

The neural system investigated is composed of a set of interconnected neurons the membrane potentials of which evolve according to deterministic rules and according to stochastic fluctuations. The connections to sensory organs or to other neural networks are taken into account by a primary set of receptors which send input to the neurons. The receptor–neuron connections form a local projection of the activity pattern presented by the receptors as modelled by a one–to–one or a center–surround connectivity. The system is schematically presented in Fig 1.
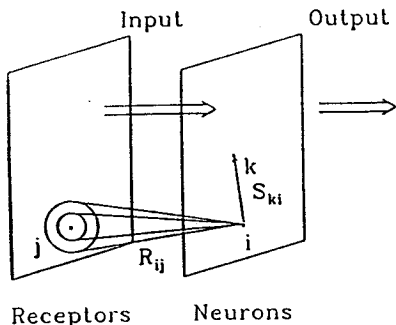

Figure 1: Schematic presentation of the neural model investigated: Receptors send spikes to a network of neurons. The resulting activity of the neural network is affected by an activity-dependent alteration of the synapses $S_{ik}(t)$, i.e. the network experiences a feed-back as indicated.

DYNAMICS OF THE MEMBRANE POTENTIAL

The dynamics of the membrane potentials involves two processes, the relaxation of the membrane potential and the neural interaction as determined by the somatic integration rule. Axonal spikes are generated whenever the membrane potential reaches a threshold value. The postsynaptic excitation by presynaptic spikes is described by an exponential activity function with decay time $T_U = 1ms$

$$G_k(\Delta t_k/\tau) = exp\left(-\frac{\Delta t_k}{\tau}\right), \text{ with } \Delta t_k = t - t_{0k}. \qquad (1)$$

$\Delta t_k = t - t_{0k}$ measures the time that has elapsed since the last spike of neuron k at t_{0k}.

The kinetic equations of the membrane potentials $U_i(t)$ which also include the stochastic fluctuations are given by a system of non–linear coupled Langevin equations

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

The first term in (2) approximates the relaxation of the membrane potential $U_i(t)$ to its resting value $U_0 = 0mV$ within a time interval $T_R = 2.5ms$. The second term in (2) describes the communication of the postsynaptic cell i with the connected neurons and receptors and adds Gaussian white noise $\xi(t)$ with the strength $\eta/\sqrt{T_R/2}$. The noise produces a Gaussian distribution of the membrane potential $U_i(t)$ with mean value $U_0 = 0mV$ and variance $\eta = 10mV$. Afferent impinging activities in addition to the noise are integrated to the total postsynaptic excitation $A_i(t)$. The activity of the presynaptic neurons k or receptors j are weighted by the time-dependent synaptic strengths $S_{ik}(t)$ or the static receptor connection strengths R_{ij} respectively

$$A_i(t) = \sum_k S_{ik}(t)G_k(\Delta t_k/T_U) + \sum_j R_{ij}G_k^R(\Delta t_k^R/T_U). \qquad (3)$$

The sigmoidal function $\sigma[A_i(t)]$ with a linear behaviour for small $A_i(t)$ and a saturation value for strong activity prevents potential changes which are unphysiologically large. The total and relative refractory periods are taken into account by the function $\rho[\Delta t_i]$ which suppresses the sensitivity of neuron i to afferent excitation during a total refractory period $T_F = 5ms$. This function also lets the neuron gradually regain its sensitivity to incoming excitation or inhibition during a relative refractory period of $5ms$.

The continuous time evolution of the potential in our model is interrupted if the neuron reaches the threshold $U_T = 30mV$ and fires a spike. Instantaneously the membrane potential is set to a value normally distributed around the refractory potential $U_F = -15mV$. In this event the time of the last spike t_{0i} is updated and the memory function $G_i(\Delta t_i/T_U)$ is set to the value 1. This behaviour is represented as follows:

$$\text{if } U_i(t) \geq U_T \text{ then } \begin{cases} t_{0i} = t, \\ U_i(t) \approx U_F, \\ G_i(\Delta t_i/\tau) = 1. \end{cases} \qquad (4)$$

The reaction of a neuron to a receptor input depends on the coupling constant ω and the connection strength R_{ik}. In the case of strong coupling the excited neuron will always reach the threshold whereas weak coupling causes only small postsynaptic potentials which never reach the threshold. Figure 2 shows the probability that a neuron which received a receptor spike at $t = 0ms$ will fire within $5ms$. This probability is presented as a function of the coupling strength ωR_{ik} for three different noise levels ($\eta = 0, 6, 10mV$). Due to the synapse dynamics the mean spike probability of the neuron $\omega \overline{A_i(t)}$ is time-dependent and can be shifted by learning.
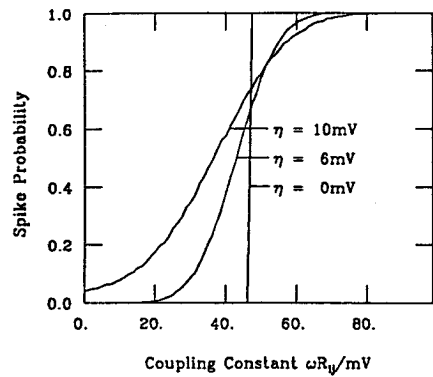
Figure 2: The probability to reach the threshold within $5ms$ after a receptor spike depends on the coupling between receptors and neurons. The gain of the curve strongly depends on the noise level η. In our computer simulations we have employed in most cases the value $\eta = 10mV$ and $\omega R_{ij} = 45mV$.

SYNAPTIC PLASTICITY IN THE STOCHASTIC NEURAL NETWORK

In our neural network with stochastic firing we introduced a plasticity of the synapses on a time scale of $0.2 - 0.5s$ [2]. According to the Hebbian rules the synaptic dynamics was assumed to depend on the synchronicity or asynchronicity of the pre- and postsynaptic spikes. In addition to the Hebbian rules we require for synaptic modifications in the present study that the mean spike frequencies $\overline{\nu_i}, \overline{\nu_k}$ of both neurons exceed considerably the spontaneous spike rate $\nu_s \approx 5s^{-1}$. If both neurons satisfy this condition in the case of synchronous firing the synapse can be strengthened. If only the presynaptic neuron fires with a high spike rate the synapse $S_{ik}(t)$ is weakened after each presynaptic spike. Details are described in Ref. 2.

The plasticity of the synapse with the strength $S_{ik}(t)$ connecting neuron k to neuron i is governed by the equation

$$\frac{dS_{ik}}{dt} = \begin{cases} -\dfrac{S_{ik}(t) - S_{ik}(0)}{T_S} + \Omega\, G_k\left(\dfrac{\Delta t_k}{T_M}\right) \kappa(G_i, G_k), & \text{if } S_u \geq |S_{ik}| \geq S_l; \\ -\dfrac{S_{ik}(t) - S_{ik}(0)}{T_S}, & \text{else} \end{cases} \quad (5a)$$

with

$$\kappa(G_i, G_k) = \begin{cases} 1, & \text{if } G_i > G_k > e^{-1} \ \wedge \ \overline{\nu_i} \gg \nu_s \ \wedge \ \overline{\nu_k} \gg \nu_s; \\ -1, & \text{if } G_k > e^{-1} > G_i \ \wedge \ \overline{\nu_i} \ll \nu_s \ \wedge \ \overline{\nu_k} \gg \nu_s; \\ 0, & \text{else.} \end{cases} \quad (5b)$$

Equation (5a) holds both for excitatory and inhibitory synapses. The first term describes a relaxation process which leads to the gradual loss of stored information. The second term effects a change of the synaptic strength. The influence of this term decays exponentially with the presynaptic activity $G_k\left(\frac{\Delta t_k}{T_M}\right)$. The short decay time $T_M = 2.5ms$ guarantees the Hebbian synchronicity condition for synaptic changes. The function $\kappa(G_i, G_k)$ switches between increase of the synaptic strength $(\kappa = 1)$, decrease $(\kappa = -1)$ and passive relaxation $(\kappa = 0)$ of the synapses to the initial value $S_{ik}(0)$. The characteristic time Ω^{-1} determines the time scale for synaptic modifications. The values assumed for Ω^{-1} were in the range $0.2 - 0.5s$.

LEARNING AND ASSOCIATION OF A PATTERN

The neural network presented showed remarkable associative properties in spite of the stochastic fluctuations of the membrane potentials. Starting from a homogeneous structure of synaptic connections with equal numbers of excitatory and inhibitory neurons the network learned a pattern presented by the receptors and associatively reconstructed the original pattern when only incomplete or disturbed patterns were presented.
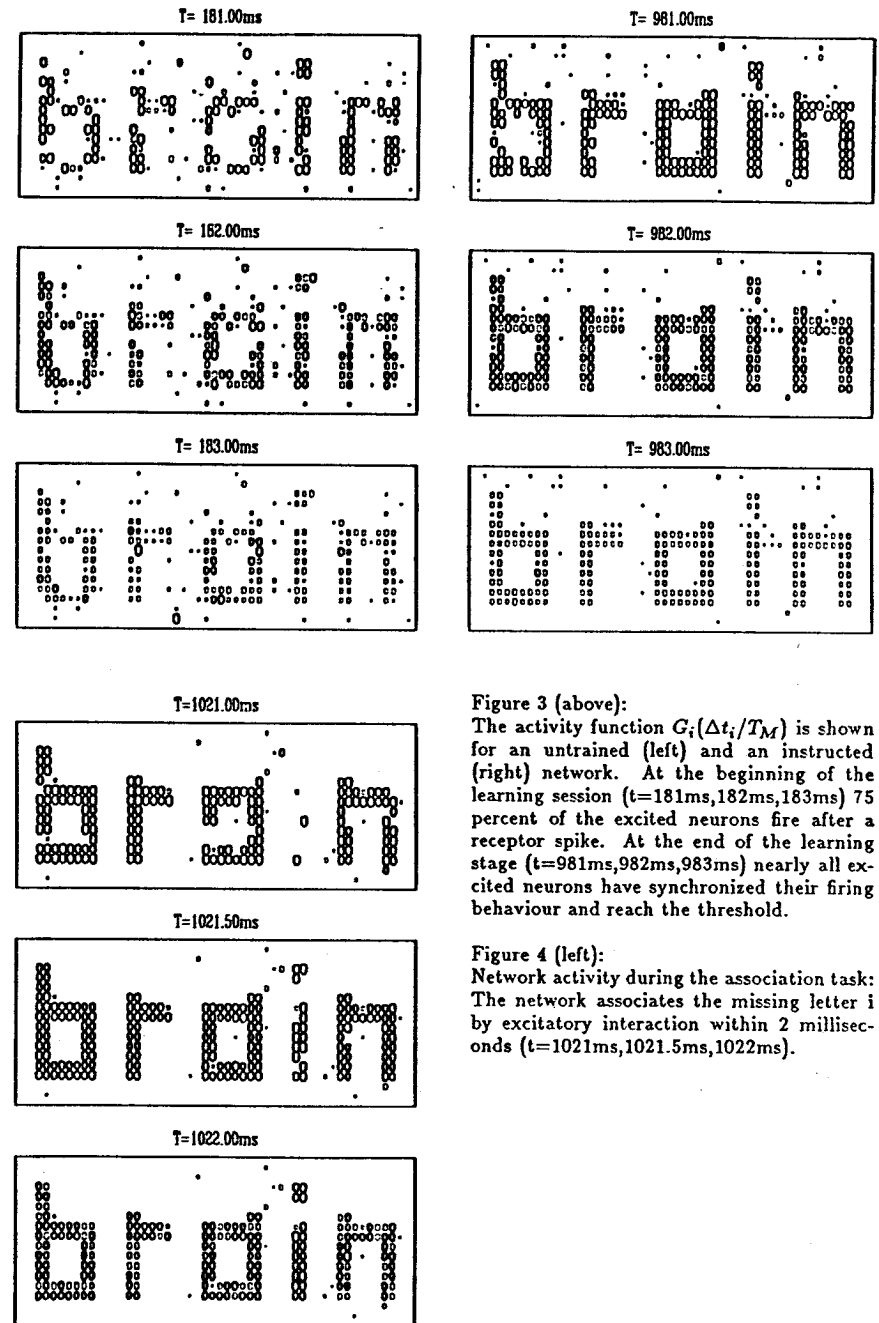

Figure 3 (above):
The activity function $G_i(\Delta t_i/T_M)$ is shown for an untrained (left) and an instructed (right) network. At the beginning of the learning session (t=181ms,182ms,183ms) 75 percent of the excited neurons fire after a receptor spike. At the end of the learning stage (t=981ms,982ms,983ms) nearly all excited neurons have synchronized their firing behaviour and reach the threshold.

Figure 4 (left):
Network activity during the association task: The network associates the missing letter i by excitatory interaction within 2 milliseconds (t=1021ms,1021.5ms,1022ms).

The simulations of the network were carried out in three different stages. During a first stage which lasted $0.3 - 1.5s$ the neural network had to learn the pattern brain synchronously presented by the receptors with a frequency of $50s^{-1}$. A homogeneous background noise with a spike rate of $10s^{-1}$ was superimposed on the pattern. The coupling constant ωR_{ii} was set to $45mV$ which effected the firing of about 75 percent of excited neurons. In a second stage lasting $50ms$ the receptors rested quiescent and the electrical activity of the network relaxed to the spontaneous spike rate. During a third stage the receptors presented the test pattern bra n which differed from the originally learned pattern by the letter i being left out.

Figure 3a shows the activity of the network at the beginning of the learning phase. At $t = 180ms$ the receptors corresponding to the pattern brain had just fired. Within $3ms$, 75 percent of the excited neurons reach the threshold and fire. The other neurons are only gradually excited and fail to fire. The network reaction to a receptor input at the end of the learning stage is shown in Fig 3b. Due to the acquired excitatory synaptic connections between neurons receiving input directly from the pattern brain (pattern neurons) the assembly reacts more synchronously and the fault level, given by the number of pattern neurons which fail to fire, nearly vanishes.

The success of the learning session is documented in Fig 4. The incomplete test pattern bra n is associatively restored by the network. The neurons representing the missing letter i react with a delay time of $1 - 3ms$, i.e. they fire nearly synchronously with the neurons excited by the test pattern.

The synchronization of the neural activity and the associative abilities of the network can be understood on account of the synaptic structure acquired during the learning session. Figures 5a,b show the afferent synapses of neuron (37,4) (presented by a star) after the training. All the neurons representing the pattern brain have developed saturated excitatory or inhibitory synapses to the reference neuron. During the association task the excitatory synapses saturated at a strength value S_u support the firing of the reference cell, whereas the inhibitory synapses saturated at $-S_l$ do not prevent the reference cell from firing. Afferent synapses of the reference cell (37,4) coming from a background neuron rest at the initial synaptic strength.
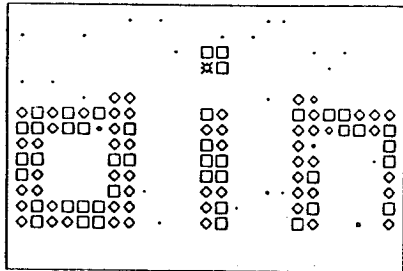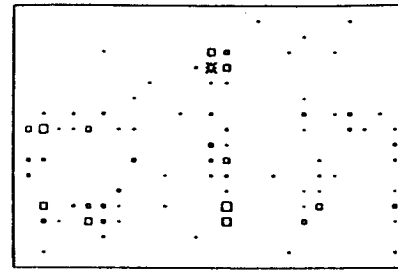
Afferent Synapses from Neuron (37,4)

Figure 5: The size of the squares and the diamonds encodes the changes $S_{ik}(t) - S_{ik}(0)$ which the excitatory and inhibitory synapses acquired during the learning session, respectively.

Due to fluctuations of the membrane potential which raise the sensitivity of the neurons the network can also learn a pattern which at any given time is only partially presented by the receptors. At each time interval the invisible fraction of the pattern (50 percent of the receptors) is chosen randomly. The uninstructed network has to learn the total pattern from the detected spike coincidences. The evolution of the synapses is demonstrated for the case of the afferent synapses of neuron (37,4) which represents the dot on the letter i. During the learning stage which lasts $3.7s$ the network has build up a synaptic structure which contains the information of the whole pattern (Fig 6). This simulation demonstrates that the synchronization of all pattern receptors at any given time is not a necessary condition for learning.

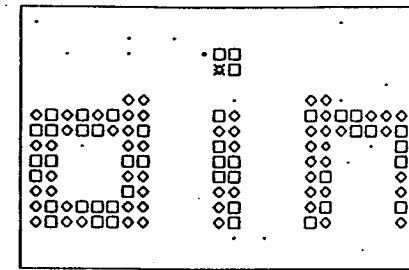Afferent Synapses from Neuron (37,4)   Afferent Synapses from Neuron (37,4)

Figure 6: Evolution of the afferent synapses of neuron (37,4) for the times $t = 1s$ (left) and $t = 3.6s$ (right) during the learning stage. 50 percent of the pattern brain is invisible.

CONCLUSION

We have presented a model neural network with a high level of endogeneous noise acting on the cellular potentials. This noise which is inherent in all biological neurons does not destroy the abilities of the network to learn and associatively reconstruct patterns. On the contrary, the noise controls the level of arousal and makes the network capable to react to a weak receptor input otherwise neglected. We argue that noise has a functional importance in neural systems. The explicit simulation of single spikes allows to test the influence of single neural events which are averaged over by mean spike rate models[8]. In addition the nonspecific influence of large neural nets (neural activity bath) on small neural assemblies can also be studied by the stochastic dynamics.

On the basis of the Hebbian rules which detect synchronicities between pre- and post-synaptic spikes a second condition for synaptic changes is introduced to protect the synaptic structure against destruction by spontaneous activity. The mean spike rates $\bar{\nu}$ of the pre- and postsynaptic neurons have to exceed considerably the spontaneous spike rate ν_s for an increase of the synaptic strengths. For a decrease of the synaptic strengths the postsynaptic spike rate must be considerably below ν_s. With this modified rules the network can also learn highly noisy patterns and patterns which are presented by a partially asynchronous receptor activity.

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