

# A Mosaic-Cycle Approach to Neural Computation\*

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*motto:*

Ainsi cette science qui devait tout m'apprendre finit dans l'hypothèse, cette lucidité sombre dans la métaphore, cette incertitude se résout en œuvre d'art. Qu'avais-je besoin de tant d'efforts? Les lignes douces de ces collines et la main de soir sur ce cœur agité m'en apprennent bien plus.

*Albert Camus, Un raisonnement absurde (Le mythe de Sisyphe)*

Keywords: mosaic-cycle, ecosystems, retina, adaptation, Turing machines

## Abstract

In this article I point at a broad analogy between observed behaviour of neuronal firing patterns and structures appearing in complex ecological systems, notably temperate pristine forests. Although such a connection seems at first remote, I will argue that the mosaic-cycle (patch dynamics) concept seems particularly apt at describing universal features common to a wide variety of selforganizing hierarchical structures. A simple approach where neurons are represented by connected probabilistic Turing machines will be introduced and related to the mosaic-cycle picture. At a more general level, I will try to sum up some of the generic properties an information processing machine like the brain must possess, including error correcting coding, optimal control, and universal computability. Finally, simple mathematical arguments are given to explain why perception and learning must be active, dialogue like processes.

## 1 Modeling: Difficulties and Perspectives

Since the last paper I wrote for the same Editor (Rujan, 1988) many of us, theoretical physicists, have lost our (neuro)biological innocence, and perhaps some of our well intentioned optimism as well. As the *Decade of the Brain* unfolds, it is not hard to see that in one respect we succeeded: the neurobiological community became keenly aware of the great potential of numerical simulations. And from buying shiny workstations to hiring Modelers there is only a small budgetary step: the Servers must be served.

What can biology expect from models and simulations? The few lines below might spare you long hours of heated discussions:

1. You can model only something you understand in some detail.
2. A model does not solve problems: a good model generates more questions than it answers.
3. Define carefully the level of description: even the biggest computers of today (or tomorrow) are Lilliputian compared to the processing power of a single cell.
4. Numerical simulations are model based specific controlled experiments.

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\*to be published in *Neural Bases of Learning and Memory*, J. Delacour (Ed.) World Scientific, 1992

5. In the long run, each particular problem might need appropriate methods: the results, not the methods are important.

A weekend easy reading like Edelman's *Topobiology* (Edelman, 1988) does not exactly enhance one's confidence in the simplified models we, physicists, garden with affection.

Modeling is thus not more (and not less) than an educated mathematical formalization of our **knowledge**, our **assumptions**, and our **expectations** about a system. Modeling teaches us how to discipline our thoughts and how to organize our knowledge. A good model has a large data compression rate: it succeeds with a small number of free parameters. There is pedantic but naive approach to science, which takes for granted that one comes closer to reality by improving a model's complexity. Fitting a set of points with more and more parameters will, of course, reproduce a given data set better and better. It will also fail completely in predicting the behaviour of a similar but slightly different case. Science is the art of finding the unifying features lying below the apparent disparity and variety of phenomena seen around us. In this respect, as Camus rightly saw it, science is dangerously close to art. A scientific discovery is — by definition — a rebellion against the status quo.

One often tends to forget that (scientific) progress is based on technology (de Solla Price, 1986). Either experimental or theoretical in nature, technology ultimately divides what is desirable from what can be done. This is particularly true for neurobiology. Contrary to Harvey's discovery of the blood circulatory system, where the analogy heart  $\Leftrightarrow$  mechanical pump worked perfectly well as metaphor **and** as mathematical model, the analogy brain  $\Leftrightarrow$  computer is rather weak. Nevertheless, we are bound to it in many intricate ways, if only because there is no better one.

Perhaps the biggest theoretical development in the last years is the wide acceptance of connectionism, the idea that the brain processes information in a delocalized, parallel, and asynchronous way, storing information in the relations (synapses) between the neurons rather than in the processing units themselves. The advantage of such a collective computation is evident to somebody trained in statistical physics. Perhaps that explains why 'connectionism' has its roots in cognitive psychology (Rumelhart and McClelland, 1986) and not in physics. Being a *consequent* connectionist, is not easy, however. We keep sliding away in the direction of Grand-Mother Neurons as soon as we take a semantic approach to 'high level' neuronal processing.

Looking from the theorist's side, the basic problem in neurobiology is the strong interaction between morphology, (electro)physiology, and collective function. Assume that we look at the vertebrate retina, a supposedly well studied part of the brain. There are discordantly many morphological types of detectors and neurons, all different in size, shape and connectivity patterns. Nevertheless, it is evident that the retina has a highly regular layered structure. Measuring intra-cell activity of single neurons, one can classify them according to their electric responses to stimulus form, light intensity, and color. Should morphology correlate with the electrophysiological data? One would expect so, but the number of distinct electrophysiological types is much less than the number of morphological types. Let us not forget that the neurons have also their — let's say — private life. They have their own metabolism, they are busy synthesizing different proteins needed for producing neurotransmitters, keeping their ion channels navigable and their structure fit. And what about the logistic system feeding the whole network with energy, transporting materials where they are needed, and organizing holidays for the tired workers?

The basic problem is that we do not know what the different cells are supposed to contribute to the collective function of the brain. In vertebrate retina, for example most cells act as filters and their rôle is more or less evident. Other cells, like the amacrine cells, are more puzzling. There is evidence for specific *morphological* adaptation of synapses to dark already at the detector level (Weiler and Wagner, 1984). Feedback from upper levels in form of dopamine

from interplexiform cells acts on the electrical junctions between horizontal cells, shutting off the color vision network and enhancing at the same time the white-black sensitivity. Is it the form of the single cell, or only the network structure influenced by the need of such regulation? In other words: to what extent are the higher functions of the brain regulating the morphology at a lower level?

Saccades, drift-like eye movements, and visual pursuit play an important rôle in the interpretation of visual images. But how is an image reconstructed after the eye muscles have positioned it on a spatially far away location on the retina? How is this feat done so fast and flawlessly that we are not even aware of it (Carpenter, 1988; Ditchburn, 1973)? I find this question at least as puzzling as the nowadays fashionable segmentation and binding problems (Damasio, 1989; von Malsburg and Schneider, 1986). When the whole structure is set up during development to what extent are these higher level functions influencing the whole neural architecture? How are the bio-chemico-physical constraints during development (Purves and Lichtman, 1985; Purves, 1988) defining the biological technology: what can be done and what not? And where and how is the long time scale evolutionary trail leaving its mark?

In physics, an elementary particle, for example, might contain in its mass part of the interaction fields. This is the case of 'renormalizable' theories, a 'soup' where one can still see the 'bread' pieces. There are even denser 'soups', where there is no way to separate the bread crumbs from the soup itself. Were the brain such a strongly interacting system, what chance do we stand in understanding it?

It might be that the basic computational units of the nervous system are very different from the single neuron cells. Besides and parallel to the already complicated neuronal network, there is a very intricate additional network of interacting neurotransmitters, receptors, intercell messengers, and other chemicals, being able to diffuse into the intracellular space and regulate in an indirect way the actual interaction between neurons and their sensitivity (Faber and Korn, 1988; Gally *et al* 1990). In this respect, neurons and their prolific networks seem to me like the highways, storage rooms, and logistic facilities of an invisible army.

In fact, the main theme of this article is that the brain can be viewed as a very complex ecological system and might be modeled in similar ways.

## 2 The Mosaic-Cycle Concept

The mosaic-cycle concept of ecology was developed in 1938 by Aubreville (Aubreville, 1932). It was revived recently by H. Remmert and D. Mueller-Dombois, several contributions to this topic have been published recently in a sleek volume (Remmert, 1991). The mosaic-cycle, or patch-dynamics concept, proposes a dynamic form of organization best exemplified by temperate primary forests in Europe and North America (Bormann and Likens, 1979). The mosaic is formed by different, asynchronously developing patches (stones) of vegetation. Within one stone, the trees have roughly equal age and belong to similar species. Their lifecycle determines the basic characteristic frequency of the patches. The strong correlations within one patch are provided by the so-called dieback phenomenon (Mueller-Dombois, 1991).

As the dominant organisms of the patch are getting older, they become more sensitive to different forms of environmental stress (sunburn, winds, mineral accumulation, parasites). When one or more old trees die, the whole patch becomes unstable and dies out. Once a patch becomes free, a fierce competition between soil vegetation, different bushes, and young trees of different species follow. In this period of the cycle, the number and the variety of different species reaches its peak. After some time, the dominant specie(s) trees become tall enough, the other trees die out and the stone matures. The 'young titans' are coming from similar species and have similar age.

Thus, this type of structure requires two basic underlying mechanisms: a characteristic life cycle, defined basically by the dominant species, and the dieback effect, which synchronizes from time to time the individual cycles over large spacial regions (a kind of local 'reset' command). Environmental fluctuations ensure that the size and the position of the patches shifts in time, allowing for a close to optimal exploitation of different resources. There is no, or very weak interaction between the different patches.

Roughly, this form of organization corresponds to a set of weakly inhibitorily coupled oscillators (young trees cannot compete with taller, older ones and can develop only on patches where the old trees died out). The size and the form of these stones might vary from a few tree fall-gap (tropical forest) to many square kilometers (taiga-type forests). Fig. 1 shows a typical patch structure in a Yugoslavian (?) virgin forest, Fig. 2 explains the basic elements of the underlying cycle. Similar structures can be observed in many social, political, and economical systems as well. What about the brain?

Three years ago, W. Singer's research team at the Max Planck Institute für Hirnforschung discovered by measuring the local field potential of assemblies of neurons in the visual cortex of the cat that typical frequencies of around 40 Hz. (Gray *et al*, 1989; Gray and Singer, 1989) build up when stimuli with given global properties are presented to the retina. Later, evidence has been found that assemblies of neurons coding for specific features of a stimulus (the contours of a moving object, for example) become synchronized not only over a single vertical cortex column but also over different cortex areas with the same receptive field (Eckhorn *et al*, 1988; Gray and Singer, 1989). The synchronization between different groups of neurons can be directly forced by the stimulus, followed by a period of more regular, collective oscillations. A very impressive finding is that when a stimulus was a moving bar, such synchronization in frequency occurred, while for two bars moving in different directions, it was absent (Eckhorn *et al*, 1990).

These experiments have been interpreted as evidence of a linking code, suitable for the representation (labeling) of spatially distributed features of an object. Such a procedure, known in object oriented programming as binding, has been suggested by von Malsburg and Schneider (von Malsburg and Schneider, 1986).

In other words, the local information conveyed by different groups of neurons is linked into more abstract global quantities by the common frequency of their synchronous oscillation. If such a coding indeed exists, it would require some decoding apparatus, some higher level interpretation of this frequency labeling, or at least a mechanism using extensively this feature. Thus, it is not only logical but rather necessary to assume that such a code must be a general feature of the neuronal code and is not restricted to the cat's visual cortex only. Many biologists are perhaps not willing to go that far, others dismiss it lightly as 'artifact'.

When trying to assess the merits of such a bold hypothesis one should consider several aspects. One is concerning experimental details: are the phenomena described here really universal and not specific for the visual cortex of the cat? Similar effects in the olfactory bulb (Freeman and Skarda, 1985) seems to support the universal character of these findings. However, one has to clarify to what extent are these observations reproducible: do they happen *always* in similar situations, or are there some cases when they happen, some cases when not? It is not my task to evaluate this type of evidence. On the theoretical side, assuming that the interpretation is correct, one has to answer more general questions: What are the advantages of such a coding? How complicated becomes to interpret it? How does the system's performance scale up, does it deliver in principle the required precision at the required speed, is it stable against fluctuations? Does it allow for a reasonable, fast neuronal computation?

Let me consider the analogy forest  $\Leftrightarrow$  brain. Assume that the basic cycle of the patches in the different primary forests of our brain is defined by the mean activity of the dominant type of neurons. Rejuvenation is related in this analogy to the arrival of different signals, competing

for attention. Although the electrical activity of the patch (assembly of neurons) might not be particularly strong, a large variety of different chemico-physical processes run in the background. The incoming stimuli compete for the resources of the patch. Some of them become dominant and suppress all others, the assembly 'maturates'. At this stage the mature neuronal assembly fires in a rather regular way. As the stimulus grows 'older', however, it becomes unstable due to channel conduction, to the overuse of local energy-material resources or because it is shut off by the lateral diffusion of particular chemical substances (Faber and Korn, 1988; Gally *et al* 1990).

The mosaic-cycle concept emphasizes the presence of desynchronized, cyclic patches of activity at the expense of complicated feedback mechanisms. In order to be a valid metaphor for the brain, it requires that the 'dieback' of neuronal-patch activity to be an essential ingredient of the neuronal regulation. In turn, this assumes the active participation of the whole intracellular local 'environment' in the neuronal computation process.

In this view, the neurons form basically a complicated network of communication channels, whose capacity, sensitivity, and traffic is directed by local chemical agents, acting from the intercellular space. For example, it has been recently suggested that a gas like NO might play a very important role in long-term potentiation (Gally *et al* 1990). Such an intercellular messenger could be exported by a postsynaptic neuron in order to increase the amount of released transmitter on the presynaptic side (Böhme *et al*, 1991; O'Dell *et al*, 1991; Haley *et al*, 1991; Schuman and Madison, 1991). Taking into account the fact that at the same synaptic junction many different chemical channels are present, it is not hard to see that including different interacting local chemical networks, possibly also lateral diffusion, is necessary if one wants to understand how and what information is transmitted over the neuronal network. In view of available new immunological methods of identifying specific neurotransmitters and neuroreceptors, keeping track of the mass balance of certain key materials might provide a lot of unexpected information on the low level dynamics of neural networks.

### 3 A Simple Mosaic Model

In order to proceed with such ambitious projects, I will introduce in this Section a deceptively simple phenomenological model of a neuron in terms of a probabilistic Turing machine. This means that instead of concentrating on the details of single neuron dynamics, I will accept the main features of such models *as they are*, leaving open the option of making the model as realistic as desired in later versions. This type of model can be easily structured and, in addition, some of the problems related to detection, preprocessing, error correcting coding, universal computation capabilities, communication, memory, learning, and optimal control can be discussed in a general framework. The goal of this paper is to introduce the main ideas and to present a valid working hypothesis: more details will be published elsewhere.

From a dynamic point of view we are interested in two main classes of processes:

1. how the membrane potential of neurons behaves as function of the input voltages, the distance from the synaptic input, and perhaps the average chemical state of the environment (single neuron dynamics),
2. how long-term potentiation is induced and preserved (synaptic dynamics).

Very good reviews for neuronal cable equations (non spiking neurons and electrical synapses) (Rall, 1989), compartmental models (Segev *et al*, 1989), or simplified variants of the Hodgkin and Huxley model (integrate and fire models) (Abbott and Kepler, 1991) are readily available.

The general form of the dynamic equations of any neuron has the form (Hodgkin and Huxley, 1952)

$$C \frac{dV}{dt} = -I_{ion}(t) + I_{inp}(t) + \kappa \Delta V \quad (1)$$

where  $C$  is the cell capacitance,  $I_{ion}(V, W_1(t), \dots, W_k(t))$  the membrane current and  $I_{inp}(t)$  the sum of external and synaptic currents entering the cell, measured over unit membrane area. The membrane current  $I_{ion}$  is the net effect of the conductance over different type of channels ( $W_i$  describes the open fraction of  $i^{th}$  channel type) between the interior of the cell and its environment. Space dependent inhomogeneity can be accounted for by the Laplace operator. Usually, ion diffusion through the different channels result on a highly nonlinear function of the membrane potential as a function of the potential  $V$  and time. A typical spike produced by a long enough positive external current  $I$  looks roughly as in Fig. 3. What is important is that if the capacitively time-integrated input signal overcomes some threshold value, then the neuron is depolarized and fires. Equally important is that shortly after firing the action potential is hyperpolarized and a given refractory period is needed before the membrane potential relaxes to the resting state. Mathematically, one has to deal with a set of more than three nonlinear differential equations. As expected, the whole menagery of period doubling Hopf bifurcations, intermittence, chaos, etc. shows up. Analyzing this complex dynamics is a profession in itself (Degn *et al*, 1987). My problem with this type of approach is that it produces fine-tuned parametric effects, many of them unstable against thermal or other external fluctuations. It is also not clear to what extent are these features actually used in neuronal computation.

Let us look again at Fig. 3. One could simply take a phenomenological approach and model this type of behaviour with a finite state machine. Let us choose some typical values of the input current and define probabilistic rules describing the different types of expected behaviour as a function of the input current. For example, assume that one allows only two different possible input values for the external current  $I_{inp} = 0, 1$ . Likewise, the membrane potential might take only on four states  $V = q, f, r, h$  defining the type of membrane excitation as quiescent, firing (or depolarized), rehyperpolarizing (or relaxing), and hyperpolarized. These four states are indicated in Fig. 3 by arrows on the  $t$  axis. Obviously, one could increase the number of states to the extent we feel necessary. Assume that every  $\tau$  time interval a new  $I_{inp}$  is entered in our probabilistic automaton. The effect of capacitive integration can be modeled by allowing the automaton to respond, say to the last 5 input values. We assume thus that the next state of the automaton will depend on the last five input  $I_{inp}$  signals and on its previous state:

$$V(t+1) = f(S(t), I_{inp}(t), V(t)) \quad (2)$$

where

$$S(t) = \sum_{k=0}^{k=5} I_{inp}(t-k) w_k \quad (3)$$

$\{w_k\}$  being some fixed set of weights. In the simplest case we could compare  $S(t)$  with some threshold value and decide whether the total input is large enough to produce a spike. Denoting this binary variable with  $\sigma(t) = 0, 1$ , we can define a probabilistic table with the elements

$$P_{i,j} = p(V(t+1)|\sigma(t), V(t)), \quad i = \{1, 2, 3, 4\}, \quad j = \{0, 1\} \{1, 2, 3, 4\} \quad (4)$$

where  $P_{i,j}$  is one element of the transition matrix  $\mathbf{P}$ . Assume for example, that the system is in the state  $V(t) = q$ . Thus, unless  $\sigma(t) = 1$ ,  $V(t+1) = q$ . If  $\sigma(t) = 1$ , then  $V(t+1) = f$ . Not all rules have to be deterministic. For example, we could introduce a time-delay by defining a probability smaller than one for the transition  $\sigma = 1, V(t) = q \rightarrow V(t+1) = q$ . Similarly, we could prolong at will the average time needed for the signal to relax from state  $r$  to state  $h$  and back to state  $q$ . In the present discretization, the transition matrix has  $4 \times 8$  elements.

These parameters are easily fitted to the real measurements and do not contain any further hidden assumption about the dynamics of the system. Once the transition matrix is defined, the dynamics of the system is described by the usual master equation

$$v(i, t + 1) = \sum_V P_{i, V \sigma} v(V, t) \Sigma(t) \quad (5)$$

where  $v(i, t)$  is the probability that at the time  $t$  the membrane potential is  $i$  and  $\Sigma(t)$  denotes the probability that in the last five time steps the input current was in average larger than a given threshold.

The current leaking through the different ion channels can be easily incorporated in this type of model. Even better, the effects of different kinds of neurotransmitters and neuroreceptors are easily modeled within this framework. Similarly, we could place this ‘neuron’ into a given environment, connect it to other neurons, model its local environment and adaptive changes resulting from different kind of chemical and/or electrical interactions.

It is perhaps worthwhile to stress again that this type of model is purely phenomenological: it assumes one already **knows** or one is able to **measure** the typical behavior of a given neuron under different environmental conditions. Note that I am not *explaining* the time dependent behavior starting from some higher principles (Ohm law, Laplace equation for ion diffusion, nonlinear dynamical effects, etc.) but I take the experimental information for granted and I model it in the simplest possible form. If later it turns out that some specific feature of the model is critical in determining the collective properties of the network, one must go back and develop a more detailed description of the underlying mechanisms.

Since we are interested in the collective properties of whole networks, this type of approach has many advantages. First, the behavior of a single computational unit is easily compared (perhaps even fitted) to electrophysiological data. Second, once the transition matrix is set, the neuron is very effectively simulated on a computer, allowing for large scale simulations. Third, the probabilistic approach allows one to work in a model where fluctuations are automatically included and controlled. Fourth, there is only a question of data availability and patience to incorporate different types of units connected in a more or less realistic way. In fact, together with J. Ammermüller and W. Möckel, we are presently setting up such a stochastic model for the outer plexiform part of the carp retina. It is rather amazing how difficult is to gather and fit together the needed data, even for such a well known system.

### 3.1 A Two-Dimensional Model

For the sake of simplicity, let us assume only one time step memory, as well as a square lattice structure of the neurons, each interacting (both ways) only with its neighbors.

The dieback is modelled as an extracellular interaction between spatially neighboring neurons. The basic hypothesis assumes that in the membrane activation cycle *there is* a state regulated by local mean potentials. For example, we could postulate the accumulation of some laterally diffusing intercellular messenger shortens the refractory period if some neighboring cells are already in the rest state. Thus, a neuron is ‘encouraged’ to move from state  $h$  to state  $q$ , once some of the neighbors have already done so.

We represent such a dynamics in the form of a probabilistic Turing machine, summarized in the the Table shown below. In the original formulation the variable  $\sigma$  is ‘read’ from some tape. Here  $\sigma$  is a environment binary variable summing up the effect due to interactions with other neurons. The internal cycle of a neuron is represented by the four internal states introduced above. In output, the neuron sends a  $\sigma'$  binary output variable to its postsynaptic partners.

$\sigma, s$	0 q	0 f	0 r	0 h	1 q	1 f	1 r	1 h
0 q	$1 - p_0$	$p_0$	0	0	0	0	0	0
0 f	0	0	0	0	0	0	1	0
0 r	0	0	$1 - p_1$	$p_1$	0	0	0	0
0 h	$1 - p_2(\Sigma)$	$p_2(\Sigma)$	0	0	0	0	0	0
1 q	0	1	0	0	0	0	0	0
1 f	0	0	0	0	0	0	1	0
1 r	0	0	$1 - p_3$	$p_3$	0	0	0	0
1 h	$1 - p_4(\Sigma)$	$p_4(\Sigma)$	0	0	0	0	0	0

Here  $\sigma(t) = \Theta(\sum_{nn} \sigma(t-1) + I_{inp}(t) - \theta(t))$ . The excitation variable  $\sigma = \{0, 1\}$  codes for the activation state of the neuron,  $\Theta(x) = 1$  if  $x \geq 0$  and 0 otherwise, the sum runs over all output  $\sigma$  values of afferent neurons,  $\theta$  is a (possibly time dependent) threshold.  $s$  is the internal state with the four possible values  $q, f, r, h$ .  $p_i, i = 1, \dots, 4$  is the probability that state  $i$  will change over the next one.  $p_0$  is the quiescent fire rate. The overall variable  $\Sigma$  takes into account the dieback effect: the value of switching from state  $h$  to state  $q$  is influenced (via intercellular messengers!) by how many neurons in its immediate physical neighborhood are already in state  $h$ . Note that this effect does NOT require a synaptic connection between the neurons.

Thus our model functions like a clock and has a definite time direction. After the system is wired up (synapses in both directions are set up), it is initialized at random, and a stimulus sequence is prepared. The stimulus is entered in a predefined way on specified areas of the network at predefined time steps. In the simulation described below, only nearest neighbor connections were allowed between neurons and  $p_4$  was increased by a given amount when one or more of the four neighbors was in state  $h$ .

A simulation cycle has two basic steps:

1. The input  $\sigma$  variable is calculated from the actual system state and the actual external stimulus input. The global  $\Sigma$  variable denotes how many spatially nearest neighbors are in state  $h$ .
2. The new internal states and the output state  $\sigma'$  are calculated using Table I.

The firing pattern of a neuron is influenced thus not only by the weighed sum of the input current coming from different afferent synapses but also by a local field building up in the intercellular space between spacially neighboring cells. In the absence of any stimulus, similarly to what happens in a forest simulation (Wissel, 1991), one sees distant, desynchronized patches which just happen to have roughly the same age (excitation state). The amount and the correlation between the random firing in the quiescent state (no external inputs) can be regulated by tuning appropriately the threshold. Snapshots of the activity patterns reveal a structure similar to Ising clusters above the critical temperature. The patterns are not strongly correlated and patches of relatively small size are evenly distributed in the system.

Applying an external input on one or two columns of the system has drastic consequences: a strongly oscillating signal takes over the whole lattice (large patch), as the system were driven suddenly well below the critical temperature. In a relatively small number of iteration steps, while keeping constant the external input, the picture changes from a single patch dynamics to a rather intertwined, dynamically changing net of filaments. Thus, the system desynchronizes (mixes) rapidly, in spite of a high average firing rate. After removing the external signal the system relaxes back to the kind of small, independent patch dynamics discussed above.



In summary, the picture emerging from this simulation can be interpreted as following: an unstimulated neuron layer forms small, uncorrelated patches of spontaneous activity. The activity level is easily regulated by chemicals acting on the average threshold. When a certain amount of external stimuli enters the system, a short period of strong, synchronized activity follows. Keeping the stimulus on will maintain the high level of activity but not the synchronization. As the input impulse is disconnected, the system relaxes back to the spontaneous activity level. In the simulation we see thus an amplification of the input signal coupled to a strong but temporary frequency locking.

In order to function as vehicles for segmentation and feature binding, neuron oscillators must be able to synchronize and desynchronize their activity rather rapidly. Recently, a large number of papers have been published on the synchronization properties of coupled oscillators (Golomb *et al*, 1991; Hall, 1991; Schuster and Wagner, 1990; Sompolinski *et al*, 1990a; Sompolinski *et al*, 1990b; Winfree, 1967). There are several problems with the synchronization of noisy oscillators. Usually, the phase locking is mediated by long-range (mostly excitatory) connections. While phase locking happens rather fast, desynchronization is much too slow. Recently, it has been suggested that using nonlinear neurons in their chaotic phase will speed up desynchronization (Hansel and Sompolinski, 1992). Another problem is related to the fact that due to the neuron-neuron couplings a phase delay must occur, a fact not seen experimentally.

A simple explanation of several puzzling facts found in synchronization of several distant patches of neurons might be related to the mosaic-cycle picture. The synchronization of different patches in distant cortical areas might simply reflect the fact that cycles characteristic for neuron-clocks were initiated at the same time. This is mostly evident, as expected, when one averages over local populations of neurons. It also gives a simple explanation for the zero phase lag synchronization measured in remote cortical columns with similar receptive fields (Gray and Singer, 1989).

## 4 Some Computational Properties

Usually, formal models of neurons (especially McCulloch—Pitts binary neurons), implement easily any logical function. However, cellular automata like models, similarly to the neuron model introduced above, are not automatically able of universal computation. In order to show that this is indeed the case, one either has to map the automaton on a known model with universal computation capability, or one has to show that using a particular coding, the dynamics of the network will implement in special cases a complete set of boolean functions, like say the functions NOR, OR, and AND.

The model introduced above was a kind of (probabilistic) clock, which under stimulating circumstances will go around its state cycle. If one connects such neurons in a linear chain and one assumes for the sake of simplicity that each neuron has four internal states, under appropriate condition they satisfy the so called next-state majority rule (Goles and Martinez, 1989). Since in this model the variables form a linear lattice, each has two neighbors. The variables are updated synchronously, and switch to the next state (mod 4) only if at least one of the neighbors is in that state. Otherwise, it remains in the old state. This type of interaction is expected also within the stones (patches) of the system, where the activity of neurons is highly correlated.

Already, this one-dimensional model has several interesting properties (Goles and Martinez, 1989). First, it decreases locally the value of the Potts Hamiltonian,

$$H_i = - \sum_j \delta_{l_i, l_j} \quad (6)$$

where  $j$  is a nearest neighbor of  $i$ ,  $l_i$ ,  $l_j = 0, 1, 2, 3$  are 4-state Potts variables and  $\delta$  the Kronecker-delta, which is a natural measure of the degree of local synchronization between variables. In fact, it seems very appealing to assume that the behavior of a neuron is controlled by its tendency to minimize some *local* cost (energy) function, to which the long-range interneuron interactions contribute only on an indirect way, via the release and take-up of neurotransmitters. Secondly, the model is able of universal computation. This can be seen by showing that the model can implement a complete set of boolean functions (NOR, OR, AND). It also contains so-called gliders, finite configurations moving in the cellular automaton space. Gliders can be used to code binary variables. Together with the logical gates, they provide the possibility of simulating any boolean function in the one dimensional cellular automata space.

One of the most appealing aspects of neural network models is the fact that information is represented in the form of certain firing patterns of a whole assembly of neurons, and is stored in appropriate synaptic connections. The process of learning is thus related to the dynamics of synaptic strengths. Exactly how long-term potentiation is induced is still under debate. There is some evidence that the synaptic changes are induced by a special network of neurons. Even so, it seems hard to believe that synaptic connections can remain so accurately tuned for years. The very existence of long term memory implies the existence of an extremely accurate regulatory system. This suggests that along with electrophysiological measurements it is highly desirable to follow also the transport of different neurotransmitters and intercellular messengers.

Although the main communication pathways in the brain are more or less hardwired, it is difficult to imagine that the information gathered from different sensory channels is synthesized into one single coherent percept without allowing for a very flexible intertwining of the different paths. Thus, although many paths are possible, every signal seems 'to know' in what specific direction it has to go. I imagine this process as somebody climbing upstream a mountain river, jumping from stone to stone. Similarly, an input signal might use the active patches of a given layer in order to move in a given direction. This type of 'content dependent' communication must be an important feature of the brain. If this is the case, then how to make sure that the messages are not corrupted during communication?

In the two dimensional model discussed above external signals lead to an amplification of the average fire rate together with a temporary synchronization of the signal over a relatively large area. One possible interpretation of this dynamic behavior is related to error free communication. Error correcting coding is a procedure of introducing redundancy in the signal such that after transmission through a noisy channel, the original signal (or the information it contains) can be reconstructed with high accuracy. One simple example is sending over a binary channel a bitstring consisting of 0's and 1's. During transmission, the bits are flipped with a small probability,  $p$ . One simple way of improving retrieval is to send each bit three times, for example. In arrival, one takes as valid only the majority of the three variables (McEliece, 1977). Looking from this perspective, short time oscillatory synchronization might be a simple way to repeat the same message several time. The integrate and fire process of the receiving neuron shows indeed similarity to a majority rule decoding. Since some axons form rather long (and thus noisy) communication channels, such a simple strategy might be very useful for error correcting coding.

There are, of course, more efficient ways of error correcting coding which use statistical mechanical methods (Sourlas, 1991)). Such algorithms for visual (Poggio, 1990) and speech recognition tasks (Derouault, 1987).

## 5 Perception as Optimal Queries

Another basic principle seen in every biological system is *control and correct*. By this I mean that from the molecular level up to the highest perception levels, the constant dialog with environment triggers adaptation and correction processes. In visual perception, for example, we are constantly inquiring our environment with finely tuned eye movements. Similarly, our cochlea is emitting sound at the frequencies most used by our auditory apparatus (Zurek, 1985). Thus, perception is not a static process, as sometimes naively assumed, but more like to an ask and answer game with our environment.

It is worthwhile perhaps to close this article with a simple example showing how the generalization abilities (the prediction power) of a single neuron can be improved by allowing it to formulate questions such that the environment's answer has a maximal degree of information. This type of game has been worked out by W. Kinzel and myself for the simple case of a single formal neuron (a Perceptron) (Kinzel and Ruján, 1990). In our model we had one neuron using Hebbian learning rules with no memory of the past examples except for its weight vector (actual synaptic strengths). After giving ourselves a few examples generated from a target linear separable function, we allowed the neuron to formulate questions on whose outcome he was most insecure. Giving the right answer to these questions, the Perceptron in question was able to learn much faster the target function than in experiments where the Perceptron was allowed to learn only randomly generated examples. This is shown in Fig. 4.

This type of reasoning may be applied to more complex networks as well: by measuring the 'questions' of the network can we guess something about its structure? This comes close enough to what psychology is supposed to do. Assuming that our motoric control is optimized, can we use our mathematical models to infer some specific neuronal structure of the motoric centers? Some impressive implementations of robot insects (Beer, 1990) seem to support this view.

## 6 Instead of Conclusions

I think it is rather clear that the type of approach suggested in this article suffers from the same weaknesses as its more complicated predecessors: although hopefully retaining some of the biological properties of the real neurons, it does not provide an automatic answer to the following questions:

1. Given an input signal, how are the characteristic features of this signal computed and coded by a network of 'neuronal' units?
2. What should be considered as a meaningful 'pattern': a given firing pattern of the neuron assembly at a given time or perhaps some more sophisticated space and time dependent (average) firing frequency configuration?
3. How are the correlations of such a pattern stored so that the pattern can be retrieved later by an appropriate input signal? What are the possible biological mechanisms behind such long term memory? Can such a mechanism store ten of thousands of pictures (Standing, 1973)?
4. How is an external stimulus transmitted to the desired brain region(s) and what kind of error correcting code is used in order to avoid noise induced failures?
5. Is there a specialized 'teacher' neuronal network controlling learning? What are the basic learning algorithms used by the brain?

6. What kind of time — and space — labelling scheme is used in order to synchronize information arriving from different input channels?
7. If one considers the brain as having long range neuronal networks coupled to local chemical interactions: which is doing what?

There are many partial and tentative answers to these questions but there is fair to say that we do not know.

The complexity of biological structures is in many ways depressing for the theoretically minded. The road to a biologically acceptable model seems rather long (and winding). The data and knowledge gathered with patience by biologists is not yet enough to provide sharp clues where and what to look for. Should then we give up the quest of understanding how we learn, remember, and think? Should one decide that the world in general and the brain in particular defies any logical (rational) explanation, as many people believe? Is there a critical level of complexity above which, even in principle, no possible mathematical description exists and thus, no theoretical prediction can be made? Is life and our ability to look at it only a single, particular accident in the Earth's, or perhaps even in Universe's history?

My answer is NO. The most unbelievable aspect of living beings IS their predictability, the unparalleled ingenuity of Nature to reuse the SAME basic structures in so many different forms. The stability of complex organisms and ecological systems defies that of their mathematical models. In many ways, a fish or an ant is a far more accurate mechanism than any atomic clock. It is this type of strong determinism, so evident during development, which indicates that the living world HAS generic functional principles. These are the Mount Everests of the next generations.

## 7 Acknowledgments

I am grateful to Prof. Delacour for the possibility to write and publish this article.

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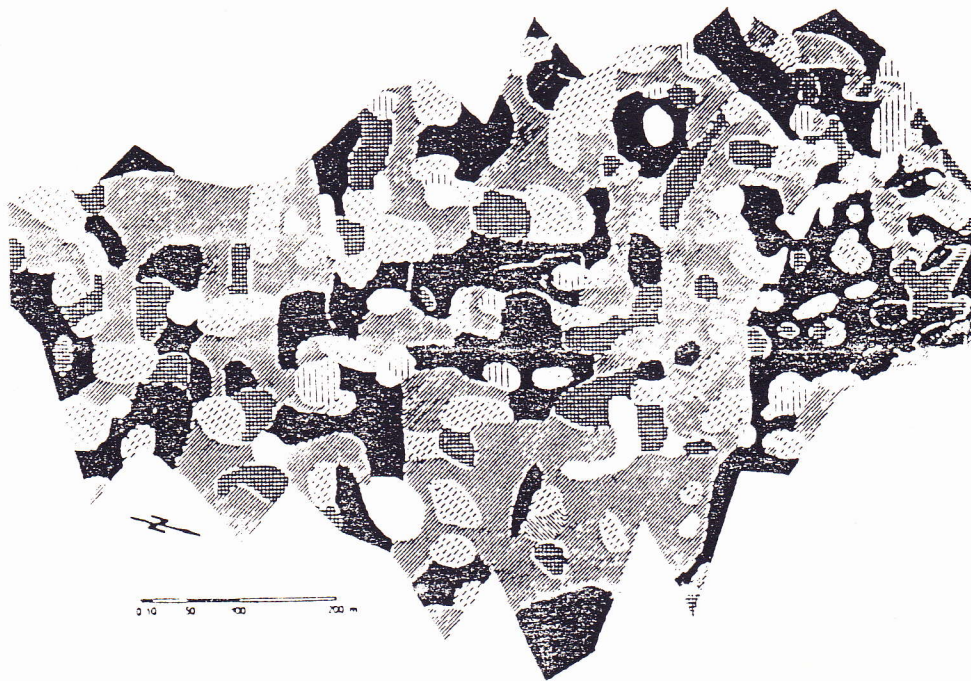


Figure 1: *The spatial distribution of an undisturbed 1-km wide central core area of a Yugoslavian virgin forest. Shading reflects age structure (after Remmert, 1991).*

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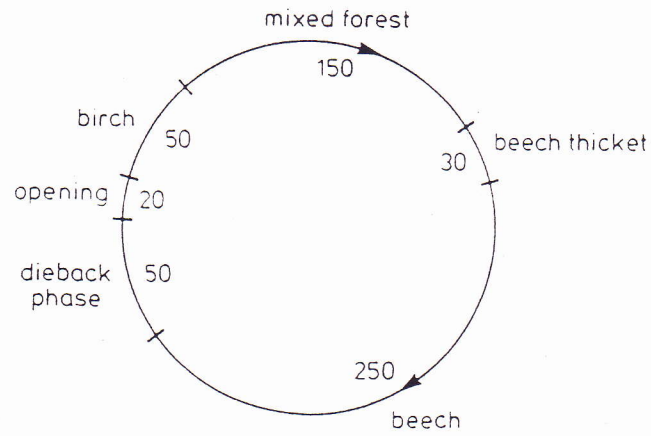


Figure 2: Schematic representation of the forest cycle (after Remmert, 1991).

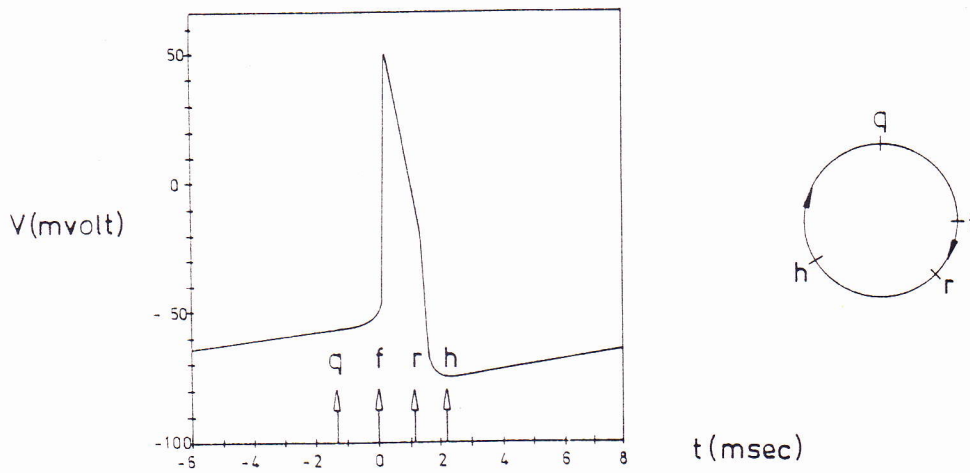


Figure 3: A typical action potential spike as a function of time. The arrows denote the four different states used in our model.

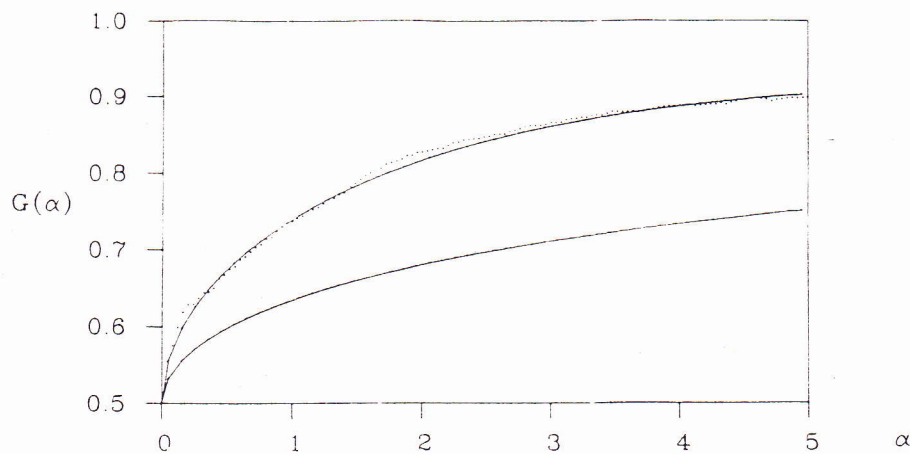


Figure 4: Generalization probability of a Hebb-trained Perceptron.  $\alpha$  is the ratio between the number of presented examples per number of input units, the lower curve is the result obtained from passive learning, while the upper curve is the result obtained when the Perceptron is allowed to ask questions with a maximal information content.