Model for a neural network structure and signal transmission

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We present a model of a neural network that is based on the diffusion-limited-aggregation (DLA) structure from fractal physics. A single neuron is one DLA cluster, while a large number of clusters, in an interconnected fashion, make up the neural network. Using simulation techniques, a signal is randomly generated and traced through its transmission inside the neuron and from neuron to neuron through the synapses. The activity of the entire neural network is monitored as a function of time. The characteristics included in the model contain, among others, the threshold for firing, the excitatory or inhibitory character of the synapse, the synaptic delay, and the refractory period. The system activity results in ''noisy'' time series that exhibit an oscillatory character. Standard power spectra are evaluated and fractal analyses performed, showing that the system is not chaotic, but the varying parameters can be associated with specific values of fractal dimensions. It is found that the network activity is not linear with the system parameters, e.g., with the numbers of active synapses. The details of this behavior may have interesting repercussions from the neurological point of view. [S1063-651X(97)16308-5]

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I. INTRODUCTION

The investigation of the dynamics of neural networks in living organisms is of ever-increasing interest for obvious reasons. In all practices we consider a neural network to be a collection of a large number of neurons. The neuron is the elementary functional unit of the central nervous system (CNS). It is an excitable cell that can receive and transmit signals to other cells in the network to which it is connected, which may be local or distant. The structure of a neural cell is relatively simple, made of the soma (the cell body), the axon, and a large number of dendrites. The axon length varies in a wide range, from some mm to more than a meter, and it is covered by a sheath of partially electrically inactive material, the myelin. In most cases the axon of a cell is used to transmit a signal to other units. The dendrites are much shorter in length, and their function is to make a large number of connections between the cells, the synapses. The spatial conformation of the dendrites is very complex with various levels of arborization, resembling some well known fractal structures. On the surfaces of the dendrites lie some type of microscopic formations called dendritic spines. The CNS neurons possess different degrees of branching, and they are classified accordingly as simple unipolar, bipolar, pseudo-bipolar, and multipolar. Anatomical studies provide well known information as to what type of neurons make up the various sites of the brain.

The membrane of the neuron is used for the signal transmission. The inner side of the membrane is electrically negative while the outer side is positive. This potential difference is about -90 mV. This is due to the difference in the concentrations of the various ions (Na^+, K^+, Cl^-, HCO_3^-, Ca^{2+}, Mg^{2+}, etc.) inside and outside the membrane. If an electrical stimulus is applied to a point on the membrane, nothing happens until the potential difference reaches values greater than -60 mV. At this threshold value the membrane becomes permeable to Na^+ ions, which enter massively in the cell, making the potential difference positive to about $\sim +30$ mV. This critical difference of potential is the threshold θ of the membrane. At the same time, a very complex interchange of ions between both sides of the cell membrane occurs via specific channels. This leads to the generation of a signal or action potential. This change lasts less than one msec in time, and propagates through the entire neuron. A stimulus that generates an action potential is excitatory. If a stimulus makes the inner side of the membrane more negative, then it is called inhibitory, as it brings the cell potential further away from the threshold for firing. When this happens, the neuron, in order to maintain the ion concentration at acceptable levels, must activate some energy-consuming mechanisms for reestablishing the previous equilibrium state.

The excitatory or inhibitory character depends on the neurotransmitter in use. A synapse may alternate between the two in response to various stimuli. Usually many synapses must fire at the same time in order to pass a signal to the target neuron. The probability of activation of the target neuron is given by the algebraic sum of the excitatory and inhibitory contributions.

In the brain the signals are passed from neuron to neuron through the synapses. On the terminal branches of the axons there are small knobs called presynaptic terminals. They are microscopic enlargements of the end point and contain (among other organelles) the synaptic vesicles, the stock of the neurotransmitter molecules. At this point the distance between the two membranes is very small (~ 200 Å). This space is called synaptic cleft. The activation of the synapse releases the neurotransmitter. The time that the signal needs to pass to the next neuron is 1000 times greater than the time the signal needs to propagate the same distance along the neuron membrane. The synapse acts as a delay point in the transmission. How this delay affects the brain function is not clear.

Another important property of the synapse is its dynamic adaptation. When a synapse is used very frequently for sig-

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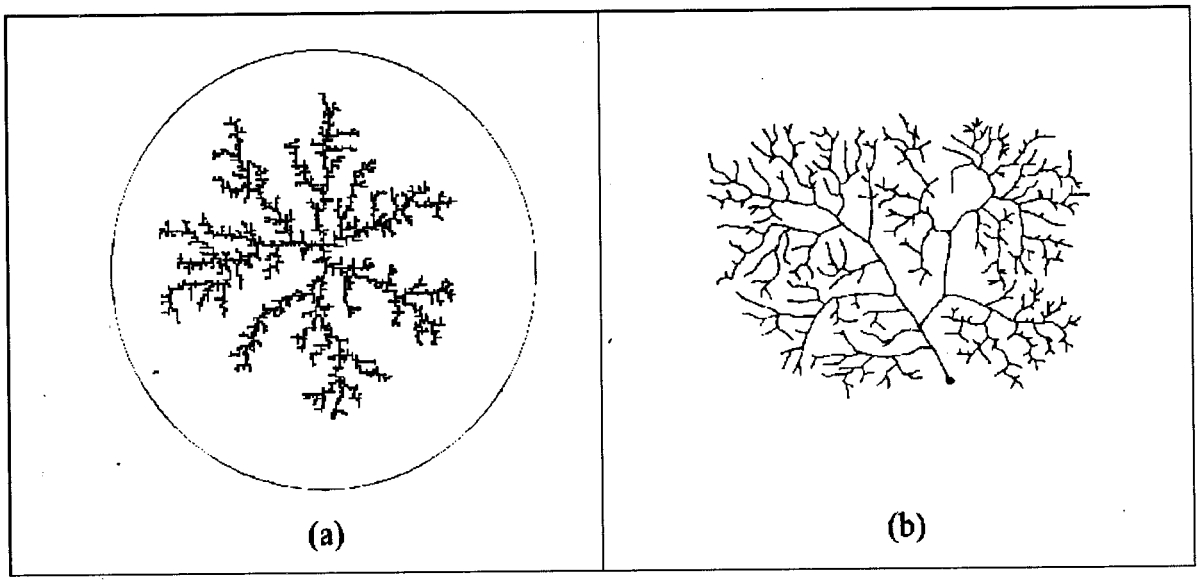

FIG. 1. (a) A simple isolated DLA cluster. (b) A camera lucida drawing of a typical Purkinje neuron [4].

nal processing within a short period of time then its threshold is proportionally reduced. This is known as the Hebbian rule, and it is a remarkable example of adaptive behavior in living organisms.

A synapse may be formed between an axon and a dendrite (axo-dendritic), between an axon and the cell soma (axo-somatic), between two axons (axo-axonic), or between dendrites (dendro-dendritic). For every neuron there is, on the average, a number of 10^4 synapses.

The main characteristics of the structure of the various brain sites are well known. In the brain cortex, for example, a piece of tissue 1 mm^3 under the microscope shows six layers with a known type of cells and known orientation of their fibers. The cerebellar cortex has three layers, where we know exactly the type of cells and the fiber conformation in each one of them. What is not known is how a small number of neurons are mutually interconnected. The interconnections are very dense, and local closed loops of neurons are formed. These loops may function as a feedback system. The inhibitory synapses play an important role in these loops. Without them a loop can carry a signal indefinitely, and this may lead to neuron death by a process known as excitotoxicity. The inhibitory synapses regulate the flow of information in the local or distant neural networks.

All these connections are continuously remodeling as a function of various stimuli. This remodeling consists of ex novo formation of new synapses and changes in existing synapses or destruction of synapses. These processes need a high quantity of energy, so the brain with a mean weight of 1.2 kg (less than 2% of the total body weight) consumes 20% of the energy of the entire body.

The brain function comprises various sensorial, motor and cognitive tasks. All of them are harmoniously integrated in a structured behavior. Every task, such as speech or object recognition is accomplished in specific sites of the brain. In most parts, the function of every site is well known. The specialization of various parts of the brain reflects a different microscopic structure of various types of neurons and with various connections between them.

One possible means to further enhance our understanding of the brain is to create a computational model for the activity of a network. It is true that such a model can exist at different levels of complexity, starting from the ingredient molecules and moving on to the cell membrane, the neuron, clusters of neurons, neural nets, etc. At every level one is limited by the available knowledge. The first mathematical approach of this type was the perceptron, a model proposed by McCulloch and Pitts [1] in 1943. It was the first effort that used a neural network as an ensemble of a large number of units, but acting in a collective manner. A second monumental model was proposed by Hodgkin and Huxley in 1952 [2], which focused on the mechanism of the ion exchange through the cell membrane that leads to signal propagation through the neurons. There is an enormous amount of work that emanated from these two pioneering efforts.

One of the parameters that has not been considered in these and subsequent models is the spatial conformation of the neuron with its complex arborizations, the position of the neurons in space, and the complicated interconnections between them. A model that includes all these parameters might be useful in the study of these properties, and their role in the entire brain function. One would hope to include as many characteristics as possible, from the present state of knowledge. Nevertheless, up to now very few models exist. For example, in most cases the neuron is treated as the smallest unit of the system while we know that this is far from the truth. In the present work we propose a model in which we have included as many characteristics as possible.

One model for the neuron structure that has recently been used as a prototype of a neural network is borrowed from condensed matter theory and is related to crystal growth, the so-called diffusion-limited-aggregation (DLA) model [3]. This model represents a highly ramified structure characterized by an abundance of dendrites, similar to the neural dendrites. A typical picture of such a DLA structure is given in Fig. 1(a), while the details are described later. Figure 1(b) gives a camera lucida [4] (which is a tracing out from slices of neural tissue, under the microscope) drawing of a typical

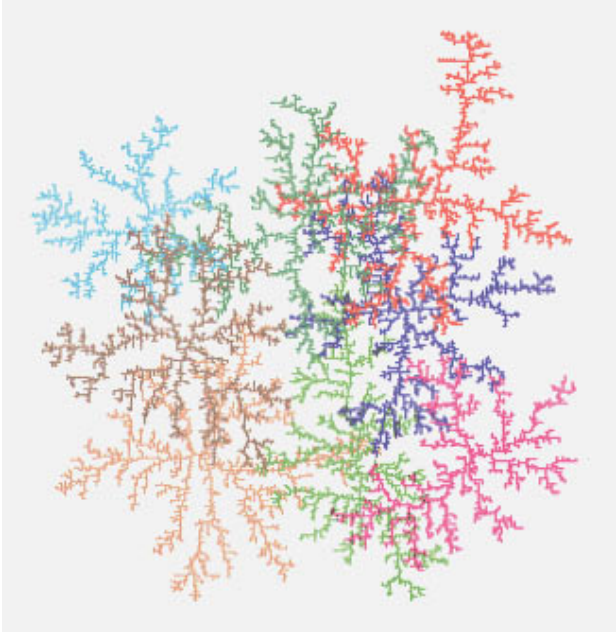

FIG. 2. (COLOR) A collection of eight DLA clusters built on a 350×350 lattice. The cluster mean size is 2200 sites. In this structure $\rho = 0.12$.

Purkinje neuron. The resemblance between the two is striking. Such a model was recently proposed by Caserta *et al.* [5], for which the fractal dimension was calculated. What we propose is to use a collection of a large number of these structures so as to construct an entire neural network. In a neural network the overlap of the dendrites of different neurons represents the synapses. Similarly here the different DLA's overlap and produce an interconnecting pattern. Such a network is shown in Fig. 2, where there are eight neurons present.

In the present model the neuron is not treated as the smallest unit of the network, as it is usually done, but its internal structure is utilized. In particular, we investigate here how a signal is transferred inside a ramified structure, and then transferred from one unit to the adjacent units through the synapses. Thus, one neuron is made of several thousand ''building blocks,'' which we will address as neuron sites, representing a realistic neuron structure. It is at this point that the DLA structure is particularly useful, as each such cluster is made of a large number of building blocks, with the overall picture resembling a real neuron. The details of the DLA structure are given in Sec. II, while the dynamics in such structures is given in Sec. III. Our results are given in Sec. IV, the signal analysis section. Finally, the conclusions and summary are given in Sec. V.

II. STRUCTURE OF THE NEURAL NETWORK

A neural network is constructed of a large number of units (neurons). Each neuron structure is a DLA cluster. Briefly, a DLA cluster structure is constructed in the following way. We start with a seed located in the center of a symmetrical object, such as a circle or a square, in two dimensions. The underlying space is a two-dimensional grid (lattice). Similar objects would apply in three dimensions. A particle is positioned in the circumference of the circle, and it is allowed to perform a random walk inside the circle. If it happens that its position at some time during the walk is adjacent to the original seed, then the motion stops and the particle gets attached to the seed, so that a cluster of two particles is now formed. If it happens that the particle moves outside the circle, i.e., away from the seed, then this particular particle is ignored, and we go on to the next particle. The process continues with a number N of particles being attached to the cluster. The number N depends on the desired size. A complete DLA structure is shown in Fig. 1(a). The interesting and curious observation in DLA is the peculiarity of the structure formed, which is far from compact. The characteristic property that prevails is that after the initial formation of the cluster by the first few particles, subsequent particles have a very low probability of reaching and attaching to the original seed, or to a site close to it, but a high probability of attaching along the dendrites, further increasing the dendritic character of the overall shape. An added advantage of the DLA model is that it is a regular fractal structure, i.e., it is self-similar under magnifications or reductions. It has a fractal dimension $d_f = 1.60$. While a DLA is a highly random structure, and statistical in nature, this characteristic d_f provides a useful quantitative measure to the entire system.

A number of neurons of size N is first formed independently and then placed together on a two-dimensional square lattice, in random positions. Thus the sytem has a density ρ, which is given by the (total number of neuron sites)/(number of system lattice sites). The placing is done by randomly choosing the positions of the seed of each neuron. It is not allowed for two seeds to be closer than a minimum distance r_0. This is done so that the overlap is mainly on the dendrites and not on the neuron soma or axon. The overlap sites are first identified as the sites occupied by dendrites belonging to different neurons. These sites are labeled as the system synapses.

At the ends of the lattices cyclic boundary conditions are employed, so that one unit touching the lattice boundary may be allowed to continue on the other side.

III. NEURON DYNAMICS

The signal is initiated in a neuron site. Both the site and the neuron are randomly chosen. This initial site is characterized as active. One time unit is the time it takes for the signal to be transmitted to its neighbor site, which is in the same neuron. This is the smallest time increment in the model. There are available up to three neighbor points. This is because the signal at each instance preferentially does not return to the site where it originated, but is spread forward throughout the network. This is done by characterizing the original point as passive for the subsequent two time units so that no signal can propagate through it for two moments. This restriction is removed after two time units. The newly visited points are also characterized as active. This procedure is repeated for a large number of time units. This mode of transfer resembles a spreading wave.

When the signal reaches a synapse site, it may be carried over to the adjacent neuron that it is in contact with. In order for this to happen the value of the signal must be greater than the neuron threshold θ. Here θ is given as a parameter. If the signal is smaller than θ then it is not lost but accumulated at

the synapse together with other arriving signals. At this point there is an option for adjusting the length of time over which accumulation takes place so that this time is used as a parameter. When the signal is greater than the threshold θ, then it can be carried over to the adjacent neuron. This procedure is not instantaneous, but the transmission is delayed for a certain time, called the synaptic delay (SD). The reason for this is because it is known that the signal transfer in the synapse is of the order of 1000 times slower than the transfer inside a neuron. If during the synaptic delay a new signal reaches the synapse it is ignored. After firing, the synapse goes into a refractory period (RP). We define the refractory period as the time period that follows the activity of a synapse, during which period this synapse cannot be active any more but must necessarily remain passive.

All synapses operate only one way, the direction being chosen at random, but staying the same for the entire duration of the calculation. When a traveling signal first reaches a synapse, it checks to find out the directionality of that synapse. If the direction of propagation is the same as the directionality of the synapse then the process proceeds as described. If, on the other hand, the synapse directionality is opposite, then the signal cannot be transmitted. In this case the synapse is placed in a refractory period. All signals possibly present in that synapse are annihilated.

All synapses are characterized as either excitatory or inhibitory. The fraction of each (out of the total number of synapses) is f_e and f_i, respectively. Thus, $f_e+f_i=1.0$. The identity of each synapse is determined at random with a probability according to that fraction. Generally, the excitatory (inhibitory) characterization describes the property that brings closer (further away) the synapse signal value to the synapse threshold. In particular, this is implemented here using the following way: For an excitatory synapse the signal is transmitted in its full value, as previously described. For an inhibitory synapse the signal becomes negative, and similarly it gets transmitted through the synapse to the next neuron, but its value is decreasing with time. The rate of decrease is constant, following a simple first-order rate law. The constant k is also given as a parameter. The same constant is used throughout this paper; it is such that it decreases the signal by $\frac{1}{20}$ of its previous value. When a negative signal reaches a synapse it causes the synapse threshold to increase by an amount equal to the signal value. This lasts for a certain amount of time and then the threshold returns to the normal value. Here we used $t=50$ time steps for all calculations. Negative signals traveling inside a neuron cannot be transmitted to other neurons.

In the present model the number of existing synapses depends on the density of the DLA structures. Thus, we introduce the parameter f_s, which represents the fraction of the synapses that can be used to transfer the signal. The rest of the synapses are present but the signal cannot be propagated through them. This is a way to increase or decrease the overall signal transmission. Individual synapses are chosen randomly, according to probability f_s, regarding their activity.

IV. SIGNAL ANALYSIS

The behavior of a neural network such as the one described here is monitored via the network activity that it exhibits, as a function of time. The activity a is defined as the fraction (out of the total) of the active units at a given time. In most cases we consider as a unit the entire neuron, even though (see later) we also examine the case of the synapse constituting a unit. Thus $a=$(neurons that are active)/(total number of neurons in the system). Thus, it is always $0<a<1$. In Figs. 3–8 we show the response of the neural network to a change in the values of the parameters discussed. In Fig. 3 we have a neural network with a refractory period RP$=300$, a synaptic delay SD$=800$, and a fraction $f_e=0.8$ of excitatory synapses. We vary here f_s, the fraction of synapses used, from $f_s=0.2$ to $f_s=1.0$. We immediately observe that the activity exhibits an oscillatory behavior. It starts from a zero value, increases during the first 10 000 steps, and then reaches a constant, ''equilibrium'' value. This constant value is higher for the larger fraction of active synapses, as expected, since there are more pathways by which the signal can spread throughout the entire network. Thus, it ranges from about $a=0.2$ (for $f_s=0.2$) to $a=0.4$ (for $f_s=1.0$). We notice that the relation is far from linear, but actually sublinear, implying that in a system such as the present one, due to the large number of units and interconnections, one needs only a small fraction of active synapses for the signal propagation. Conversely, if a large number of synapses is destroyed over time, this does not lead to catastrophic consequences for the operation of the neural net.

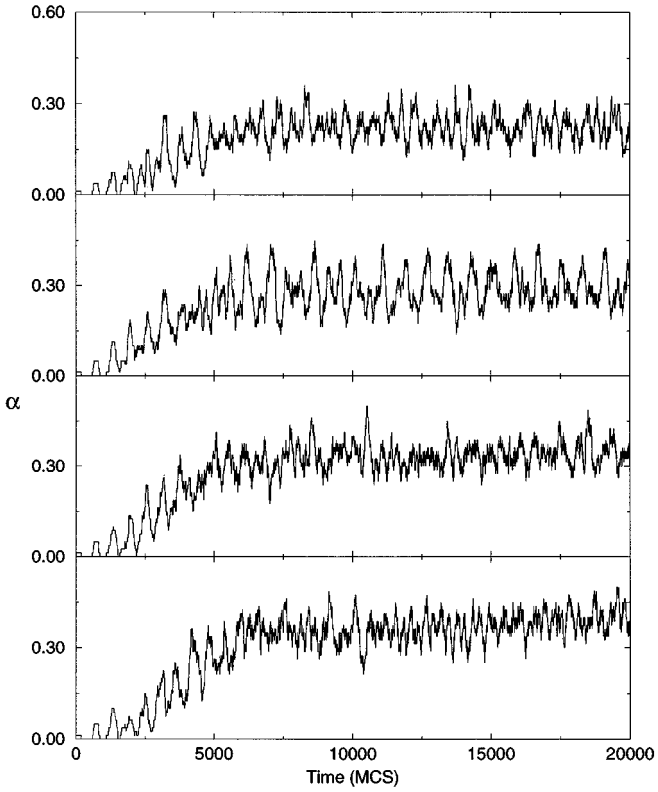

FIG. 3. Normalized (neuron) activity vs time [in Monte Carlo steps (MCS)] RP$=300$, SD$=800$, $f_e=0.80$. The four diagrams depict the percentage of the synapses that is used, f_s, which is (bottom to top): $f_s=1.0$, 0.75, 0.50, 0.20.

The oscillatory behavior is encountered (and also in the subsequent figures) because of the constraint of the refractory period imposed on the neural activity, which decreases

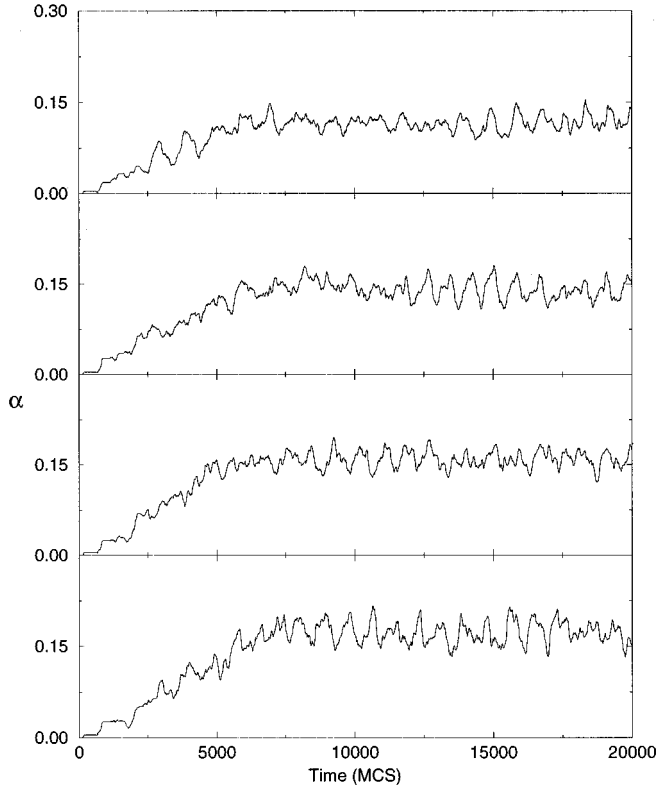

FIG. 4. Normalized (synapse) activity vs time (in MCS). RP=300, SD=800, $f_e=0.8$. The four diagrams depict the percentage of the synapses that is used, f_s, which is (bottom to top): $f_s=1.0$, 0.75, 0.50, 0.20.

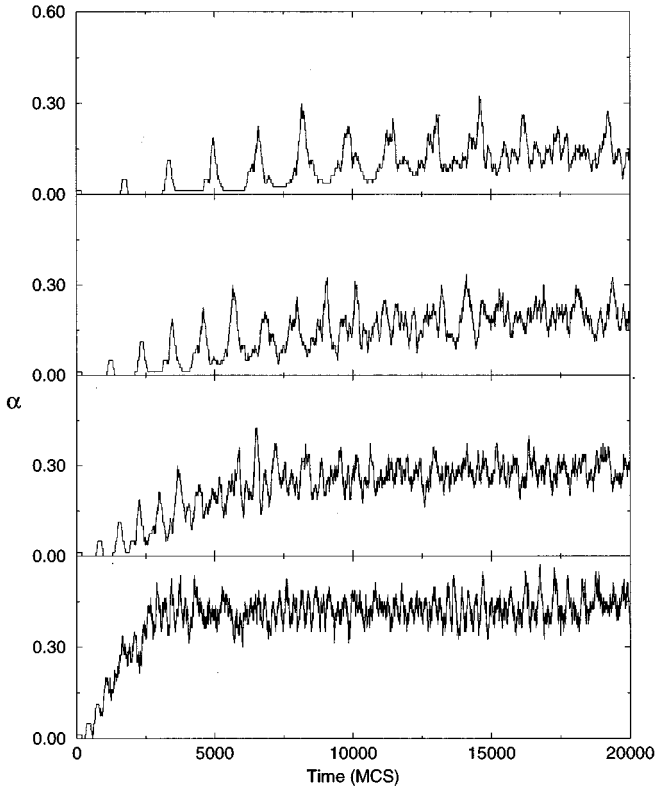

FIG. 5. Normalized (neuron) activity vs time (in MCS). RP=300, $f_e=0.8$, while the fraction of the synapses that are used is $f_s=0.50$. The four diagrams depict the SD, which is (bottom to top): 200, 600, 1000, 1500.

the activity to zero after firing. This is a very realistic feature. First, it shows that the activity time series is not fully periodic. However, there is obviously some ''approximate'' periodicity as the distance between the peaks is almost constant. This means that some new information may be revealed in it, and this is presented after the discussion of the figures with the variation of the system parameters.

Figure 4 shows the same data as in Fig. 3, but now we plot the activity a of the synapses, i.e., only for this figure, for comparison purposes, a is the fraction $a=$(synapses that are active)/(the total number of synapses). We observe similar behavior as in the activity of neurons of the previous figure.

Figure 5 shows the variation of the length of the SD. Here SD goes from 200 to 1500, and we notice that the activity of neurons a goes from $a=0.10$ to $a=0.45$. This increase is also expected, because the short SD allows for faster signal transmission and propagation.

Figure 6 shows the variation of f_e, the fraction of excitatory synapses. A small fraction leads to an almost zero activity, and vice versa. Of course, this is due to the fact that a small fraction of excitatory synapses means that typically these synapses are well below the threshold value.

In these calculations the general question arises as to what effect randomness has in the observed behavior, and also to the relation of randomness to the choice of the specific values of the parameters used. For this reason we performed calculations for the parameter values of Fig. 6 under several different variations of randomness. Thus, we used different starting points of the signal. This implies that all signals would follow totally different paths. We also used totally different DLA clusters with the same stipulations as before. The results are shown in Figs. 7 and 8. In these figures we use the same parameter values as in Fig. 6. In Fig. 7 we have the case of a small fraction of excitatory synapses, $f_e=0.3$. We notice that we get a large variety of signals, ranging from zero to periodic signals, but all have the characteristic that they are small, and consistent with the small values of Fig. 6. In Fig. 8 we performed the same calculations, but for a large fraction of excitatory synapses, $f_e=0.8$. Here we see that the variation of the signal is minimal, implying that for large parameter values the system behavior is always the same. We conclude that randomness in these systems does not drastically affect their behavior, but simply shows the statistical character of the process.

A first impression about the nature of these signals can be obtained by taking the power spectrum of these, at least to exclude the possibility that there is only white noise present. Thus, in Fig. 9 we take the power spectrum of the curve of Fig. 3(a), and plot it in log-log form. We see a straight line with the slope of -1.81. Different statistical realizations of this curve produced a slope of -1.83 (average of 10 different realizations). This implies a f^{-2} law, and some underlying structure in the signal.

In order to quantitatively interpret the periodicity of the activity signals presented above, we performed an analysis of several of these signals, according to the method of Grassberger and Procaccia [6]. In this method the signal is treated

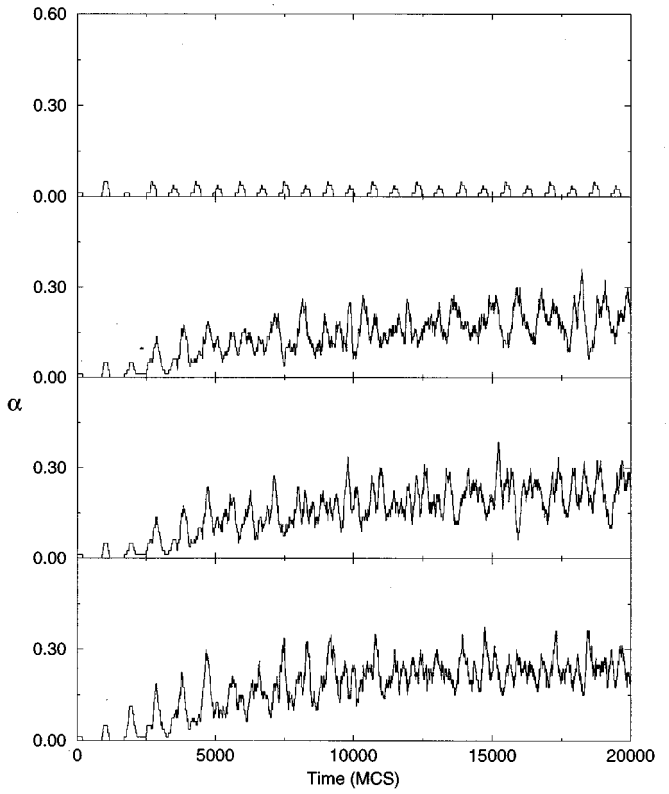

FIG. 6. Normalized (neuron) activity vs time (in MCS). RP=300, SD=800, $f_s=0.5$. The four diagrams depict the change in the fraction of the excitatory synapses, which is (bottom to top): $f_e=0.80$, 0.60, 0.50, 0.30.

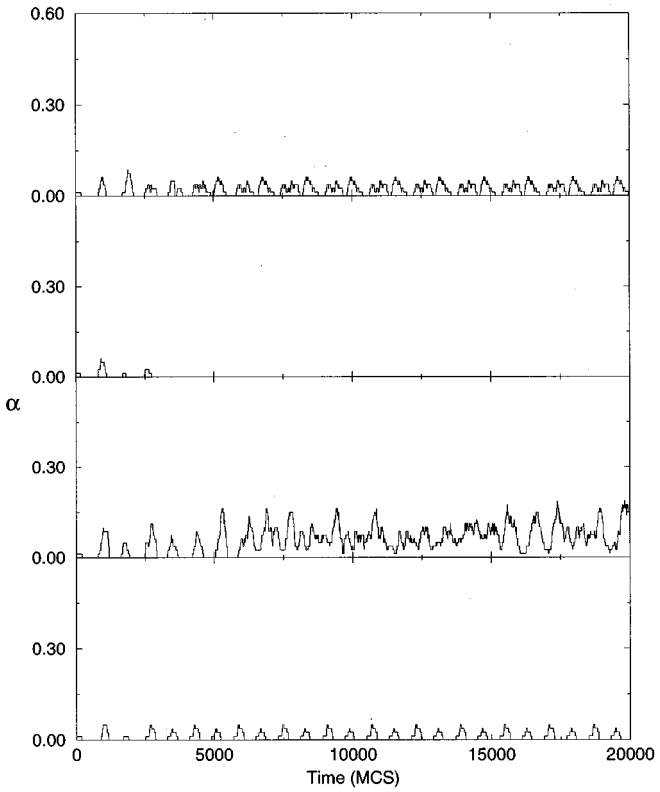

FIG. 7. Normalized (neuron) activity vs time (in MCS). RP=300, SD=800, the fraction of the synapses that is used is $f_s=0.5$, while the fraction of the excitatory synapses is $f_e=0.30$. Each plot is produced for a different signal starting point (different random number generator seed).

as a time series for which a possible characteristic fractal dimension can be derived. This is done in the following method. We consider a time series to be made of the signal of any of these in Figs. 3–8. We must ignore the section of the series which gives an initial rise, and keep only the section for which the signal varies around a mean value, at which point it is a steady-state signal. For example, for the signal of Fig. 5(c) we consider the time domain $10\,000<t<50\,000$. Let us call this signal $x_0(t)$. We would like to reconstruct the dynamics of the system solely on our knowledge of $x_0(t)$. We consider the phase space spanned by the variables $k=0,1,2,\ldots,n-1$, where k are several variables that take part in the dynamics of the system. For our problem these are the parameters of refractory period, synaptic delay, etc. At a given time a state of the system is a point in phase space, while a sequence of states in time gives a trajectory. If the dynamics of the system obey some dissipative deterministic laws, then the trajectories converge to an attractor. We thus form this attractor from the $x_0(t)$ series, by successively shifting the original time series by the same amount in time Δt, and forming n such series as

$$x_0: x_0(t_1), x_0(t_2), x_0(t_N),$$

$$x_1: x_0(t_1+\Delta t), x_0(t_2+\Delta t), \ldots, x_0(t_N+\Delta t),$$

$$x_2: x_0(t_1+2\Delta t), x_0(t_2+2\Delta t), \ldots, x_0(t_N+2\Delta t), \quad (1)$$

$$\vdots$$

$$x_{n-1}: x_0(t_1+(n-1)\Delta t), x_0(t_2+(n-1)\Delta t), \ldots,$$

$$x_0(t_N+(n-1)\Delta t).$$

These variables are expected to be linearly independent if the Δt shift is properly chosen. We chose several different Δt values, but in the subsequent calculations we use $\Delta t=1000$ time units. Notice that $x_0(t)$ is a vector made of the set of points, as given in Eq. (1). A general notation for it is x_i. We now choose a reference point in x_i and compute all the distances $|x_i-x_j|$ from the $(N-1)$ remaining points. This way we get the total of all points x_i in phase space. Doing this for all i we get

$$C(l)=\frac{1}{N}\sum\left[\frac{1}{N}\sum\ \Theta(l-|x_i-x_0|)\right], \quad (2)$$

where Θ is the Heavyside step function, $\Theta(x)=0$, if $x<0$ and $\Theta(x)=1$ if $x>0$. $C(l)$ is the correlation function of the attractor, since it shows how a point in the vector x_i affects the positions of other points. Thus if the attractor is a d-dimensional manifold, then we expect

$$C(l)\sim l^d, \quad (3)$$

with its dimensionality given by the exponent d. In Fig. 10 we plot in log-log form $C(l)$ vs l for several different m values. We see that for small m the slopes of the ensuing curves increase, but after $m=5$ (approximately) the slopes

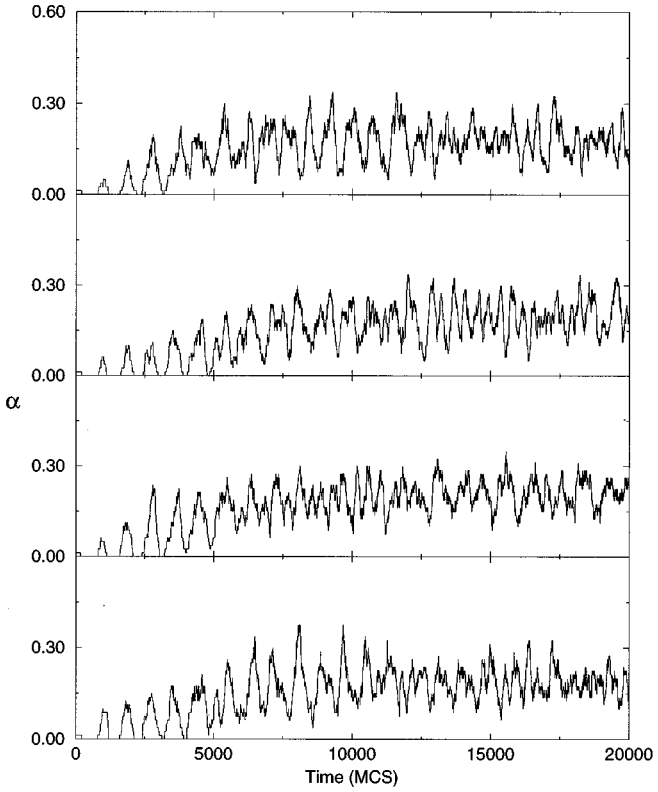

FIG. 8. Normalized (neuron) activity vs time (in MCS). RP=300, SD=800, the fraction of the synapses that is used is $f_s=0.5$, while the fraction of the excitatory synapses is $f_e=0.80$. Each plot is produced for a different signal starting point (different random number generator seed).

become constant. In Fig. 11 we plot the slope vs m, producing the fractal dimension of the system. We do this for the four signals of Fig. 5, which are very different in their complexity. The eventual limiting slope is in the range of 4.0–5.0, while we observe that the higher-frequency signal gives the highest slope and as the frequency signal decreases, the fractal dimension also decreases monotonically. Thus, this method supplies a good quantitative criterion, that connects

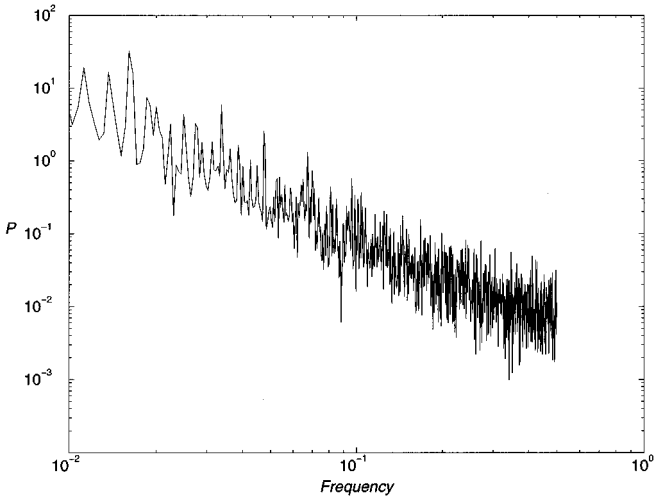

FIG. 9. Power spectrum of the signal of plot of Fig. 3(a), plotted in logarithmic scale. The frequency is in inverse MCS.

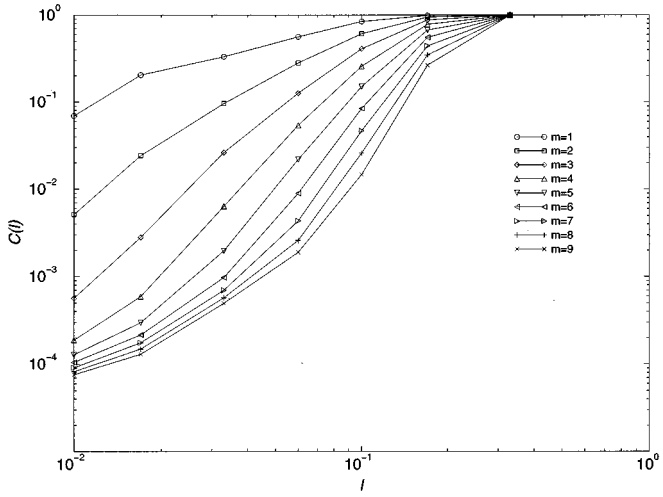

FIG. 10. The correlation coefficients $C_2(l)$ vs l for the signal of Fig. 5(b).

the neural net activity to the fractal dimension picture. Of course, if the signal contained only white noise, then we would not observe the saturation to a finite slope, as in Fig. 11, but the slope would increase linearly with m in the entire domain. We note here, however, that the estimation of the slopes in the curves of Fig. 10 (that are used to produce Fig. 11) is very precarious, because our data do not have long straight line sections, as one would, in principle, expect theoretically. Since this is common with experimental signals, we used only the straight sections of these curves, but in a consistent manner.

In Fig. 12 we plot the Kolmogorov entropy K vs m for the value of l=0.004. K is defined as

$$K=\frac{\ln C_m}{\ln C_{m-1}} \tag{4}$$

and we observe that in the same range K is also reaching a constant value, as expected, from the fractal dimension picture.

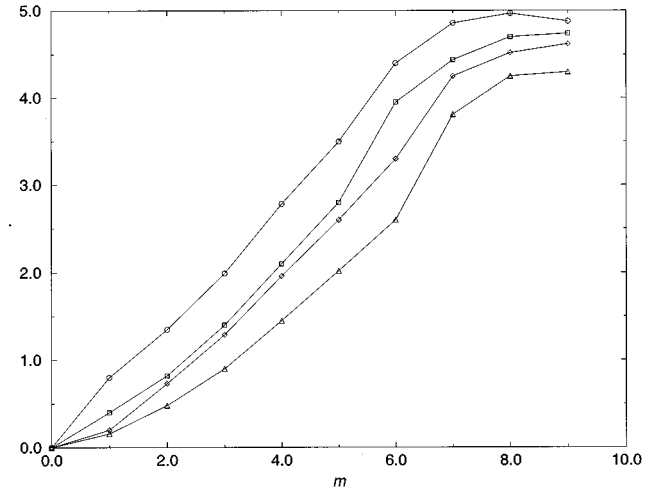

FIG. 11. Slopes of the curves of Fig. 10 vs m.

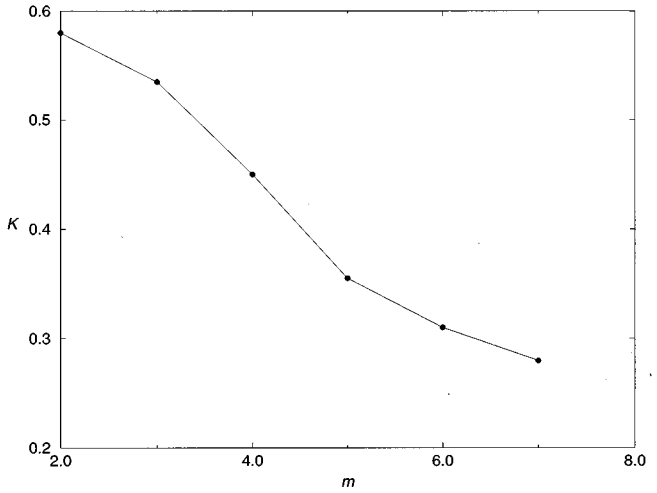

FIG. 12. Kolmogorov entropy K vs m for the data of Fig. 10.

We performed the same analysis for some more such signals, resulting is similar curves, and similar fractal dimensions. The implication of this analysis is that for our system the minimum embedding dimension is approximately in the range of $m=5-6$, signifying that this is the number of parameters that is necessary to describe the system. This number most probably maps the total number of parameters used in the simulation, and thus it includes the refractory period, the synaptic delay, the percentage of synapses used, and the percentage of excitatory synapses (total of four parameters).

V. SUMMARY AND CONCLUSIONS

In the present work, we have attempted to build a primitive model for the activity of neural networks that incorporates the characteristics of the neural activity of the CNS, with several conceptual parameters, for which it is known from physiology that they are very basic in the dynamics of neural networks in living organisms. The prime difference of our model is that it does not treat the neuron as the smallest unit of the neural network, but each neuron is made of thousands of building blocks, with a ramified dendritic structure, which plays an important role in the signal transfer.

We have shown that the activity of a net made of DLA structures produces a very complex, ''chaotic'' looking signal, which, upon elaborate analysis, is found to contain information about the magnitude of the system parameters. This signal is the response of the system to the random impulse presented. A detailed approach shows that it is far from random or white noise, but it quantitatively gives a measure of the ability of the neural net to sustain its activity. Every set of initial conditions that is used leads to a characteristic fractal dimension, which is always monotonic and within a given range. The numerical value of the fractal dimension gives information about the signal magnitude and other characteristics, making it a useful quantity to monitor.

An important point is that the brain is a very complex feedback system. The biological systems in nature consist of millions of elementary feedback subsystems, which control each other in a very precise hierarchical structure. In the present simulation feedback subsystems exist created in random order. In these subsystems both inhibitory and excitatory synapses exist. We have included a parameter that defines the ratio between the excitatory and inhibitory synapses. We observed that a major number of inhibitory synapses corresponds to a lower activity of the system expressed in the number of active units at the given time. This situation is observed in biological systems as well, especially when we supply the organism with substances that enhance the inhibitory synapses.

Also of importance is the ''spatial'' aspect of the signal transmission. In the brain the signals are transported by bundles of fibers, a structure that does not exist in our model, because of the small number of neurons used. We will deal with this in the future.

The brain, as an autonomous system, operates under various internal or external conditions. If it loses a small number of neurons and/or synapses, it can achieve its target without a serious problem. In various diseases, such as in Parkinson's disease, it is well known that before the first symptom appears, a specific region of the brain (substantia nigra) loses more than 60–70 % of its neurons, and consequently the relevant synapses. Similar observations pertain in all degenerative diseases. This condition is treated in the present study, as the parameter in the system given by the ratio of active synapses. We saw that the system activity is decreased as the number of active synapses is decreased. A desired quantity would be the exact point that this degeneration first appears. However, we have not dealt in detail with this quantity in the present study.

In conclusion, we see that incorporating many complex features in neural networks based on information from neuroscience leads to interesting conclusions, some expected, while others give, at least qualitatively, the trends of what happens when the number of synapses and the excitatory to inhibitory ratio are decreased.

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