Chaos and associative generation of information by networks of neuronal oscillators

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Networks of neuronlike elements described by a simple model are studied. For a single neuron, a number of bifurcations and chaotic attractors are observed. A mechanism of an associative production of information based on a transition *fixed point* \rightarrow *chaotic attractor* \rightarrow *new fixed point* is proposed and discussed. [S1063-651X(96)00308-X]

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Recent progress [1] in investigation into neurocomputing is based to a large extent on the seminal paper of Hopfield [2] who studied networks composed of binary, all-or-none, formal neurons [3] having symmetric interneuronal (synaptic) couplings. Among brain functions Hopfield concentrated only on associative memory (AM), i.e., on a system that should retrieve an entire stored pattern if sufficient partial information (a noisy pattern or a part of the original pattern) is presented to the system. Obviously, it is important to go beyond the limits of this approach towards (i) models more close to real nervous systems and (ii) other types of information processing performed by them. In this paper we attempt to advance in both directions in order to attain the two goals described as follows.

First, it is desirable to have a model in which a neuron can exhibit a rich variety of dynamical regimes including not only such classical phenomena as oscillations, threshold behavior, etc. (see, e.g., [4-6]) but also chaotic motion. There are experimental observations that a real nerve cell can demonstrate chaotic behavior [7] and chaotic modes can be of great importance in brain activity as discussed in a number of papers (see, e.g., [6,8,9]).

Second, we would like that a system of neuronlike elements can function both as AM and an associative generator of information. Following the Hopfield approach [1,2], we assume that any piece of information (pattern) μ is encoded by a fixed point (FP) ξ^{μ} of the network dynamics in such a way that close, in a sense, patterns are mapped into close points of the phase space. Then an AM machine should operate so that if its initial state falls in a neighborhood of a FP ξ^{μ} then the state converges to ξ^{μ} . Analogously, by associative generation of information (AGI) we mean the following: if the appearance of a pattern encoded by a FP ξ^{μ} is adversely evaluated for some reason by the system (or by an external supervisor) then a new FP $\tilde{\xi}^{\mu}$ should be produced in the vicinity of the former, yielding a new pattern. AGI is important for elaboration of plans of behavior, creative thinking, forecasting, and even for such mental processes as perception and recognition of patterns [10].

Plainly, AGI machines could be designed by making use of a random generation of numbers. However, this way can obviously be too exhaustive and seems to be not plausible for real nervous systems. On the other hand, it is well known that chaos is characterized by divergence, in certain directions, of close phase trajectories and hence a chaotic attractor (CA) can, in principle, be viewed as a generator of some information (see, e.g., [9]). It is natural therefore to look for dynamical mechanisms of AGI on the basis of chaotic phenomena in neural networks. Thus our second, main, target mentioned above closes to the first.

As a reliable ground of research into computational abilities of neural networks, one may take the known Hodgkin-Huxley equations [11], which provide a good quantitative description of essential properties of nerve cells [4–6]. However, they are too complicated. As a simplification that reproduces principal features of the Hodgkin-Huxley equations, the FitzHugh model [12] is often accepted [5]. The state of a neuron is determined, in this model, by two variables and therefore dynamical equations for them do not admit chaotic solutions [13]. For this reason, we study a simple generalization of the FitzHugh model in which the state of a neuron is described by three dynamical variables x,y,zwhose time evolution is governed by the differential equations:

$$\dot{x} = a_1 x + a_2 y + a_3 z + a_4 - \lambda x^3 + J,$$

$$\dot{y} = b_1 x + b_2 y + b_3 z + b_4,$$
(1)

$$\dot{z} = c_1 x + c_2 y + c_3 z + c_4.$$

Here $a_{\mu}, b_{\mu}, c_{\mu}, \mu = 1,2,3,4$, are constants, λ is a positive constant, and *J* is a current entering the neuron. Equations (1) can also be treated as a reduced variant of the Hodgkin-Huxley equations or as a partly linearized version of the model of Hindmarsh and Rose [14]. They are a straightforward generalization of the van der Pol–Duffing equations [15] as well. A biological motivation of Eqs. (1) and interpretation of the variables and parameters can be different depending on the point of view at the equations [16].

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FIG. 1. Phase portraits of two CA's projected on the *x*-*y* plane for a neuron (1) appearing at different values of the bifurcation parameter $a_1:(a)$ $a_1=3.9$; (b) $a_1=3.96$. (Values of the other parameters are given in the text). Estimated values of the largest Lyapunov exponent λ and the Lyapunov dimension d_{λ} are (a) $\lambda=0.24$, $d_{\lambda}=2.009$; (b) $\lambda=0.92$, $d_{\lambda}=2.033$.

{PA, PA, PA, PA, CA}, {PA, PA, CA, CA, CA, CA}. A number of bifurcations are observed such as changes of the stability of FP's, period doubling, intermittency explosions, interior and boundary crises, and various changes of the symmetry of CA's and PA's including homoclinic connections for cycles of different complexity.

As a typical example of a sequence of bifurcations, we mention the following chain: $FP \rightarrow P1 \rightarrow FP \rightarrow 2FP \rightarrow$ $2PD \rightarrow 2C \rightarrow 2I \rightarrow 2P3 \rightarrow 2PD \rightarrow 2C[P3] \rightarrow C \rightarrow I \rightarrow P3$ & $2C \rightarrow 2PD \rightarrow 2P1 \rightarrow 2FP.$ This scenario appears as the coefficient a_1 is varied and Eqs. (1) are symmetric under reflection (i.e., when $a_4 = b_4 = c_4 = J = 0$; values of the parameters are the following: $a_k = \{a_1, -3, 3\},\$ $b_k = \{-55, 0, 15\}, c_k = \{18, -18, -2.5\}, k = 1, 2, 3, \text{ and } \lambda = 1;$ this set of values is used in what follows for illustrations, if other values are not given). Here P1, P3, and P5 designate a PA comprised of one, three, and five "turns" respectively; P3&P3 corresponds to a symmetric cycle similar in form to two cycles P3; PD and I are the marks of period doubling and intermittency, respectively; the expression C[P3] means that the CA resembles, in a sense, a cycle P3 [see, e.g., Fig. 1(a)]. The number 2 before a designation of an attractor indicates that there are actually two attractors symmetric under reflection. Phase portraits of two CA's are given in Fig. 1. A bifurcation diagram is given in Fig. 2 [17].

One can also mention such a phenomenon as a tight intertwining of coexisting attractors observed at some values of parameters. This property implies that the shape of a single spike of oscillations does not depend practically on the



FIG. 2. A bifurcation diagram for a neuron (1) as the coefficient a_1 is varied in magnitude along the horizontal axis. Values of the *x* coordinates of points in the Poincare section defined by intersections of the phase trajectory with the plane y=0 are plotted along the vertical axis.

attractor but samples of such impulses are different for different attractors. In particular, the frequency of impulses as a function of a current entering a neuron has a form that depends on the attractor. Another feature, revealed for coexisting CA's, is a sensitivity of the sample of impulses, for a particular attractor, to a value of the bifurcation parameter a_1 .

The main conclusion here is that Eqs. (1) exhibit both the classical properties of a neuron [as it should be because Eqs. (1) yield a generalization of the FitzHugh model] and practically all types of chaotic behavior revealed in other threedimensional nonlinear systems so that our first target is achieved in plenty. Unfortunately, there are no relevant neurobiological data to carry out detailed comparisons with our findings.

Now we consider the behavior of a system composed of N identical elements described by Eqs. (1). The interaction between the neurons results in the currents J_1, \ldots, J_N entering them. The commonly accepted equation is $J_i = I_i + \sum_{j=1}^N T_{ij} w_j$, where I_i is an external current received by the neuron i and the synaptic efficacy T_{ii} determines the strength of the influence of an output signal w_i of the *j*th neuron on the state of the *i*th neuron. As usual, we set $w_i = f(x_i)$ where $f(\cdot)$ is an input-output transfer function of a neuron assumed to be a monotone sigmoid (for a discussion of such a representation see, e.g., [6]). A typical simple choice is $w = \tanh(gx)$, g = const. A more accurate representation is the Freeman parametrization of experimental data of the form [6,19] $w = \Phi(x) \equiv w_0 (\alpha - \exp\{-[\exp(\beta x) - \gamma]/\kappa\}),$ if $x > x_0$, otherwise w = 0. Here $x_0 = \beta^{-1} \ln(\gamma - \kappa \ln \delta)$ and $w_0, \alpha, \beta, \gamma, \delta$, and κ are constants. We used both the equations although most intensive studies were carried out for the linear approximation of the transfer function, w = hx, h = const, because this enables us to look for appropriate values of the network parameters with the aid of an analytic technique [17,18].

Following the Hopfield approach [1,2], the matrix of interneuronal couplings *T* should be adjusted so that the set of given *N*-dimensional vectors ξ^1, \ldots, ξ^p encoding memorized patterns (MP's) be FP's (as usually, we consider the case p < N). Therefore the matrix *T* must obey the system of equations $\Sigma_j T_{ij} v_j^{\mu} + u_i^{\mu} = 0$. Here vectors $\mathbf{v}^1, \ldots, \mathbf{v}^p$ and $\mathbf{u}^1, \ldots, \mathbf{u}^p$ are defined so that $v_i^{\mu} = f(\xi_i^{\mu})$ and $\begin{aligned} u_i^{\mu} &= a_1 \xi_i^{\mu} + a_2 \zeta_i^{\mu} + a_3 \eta_i^{\mu} + a_4 - \lambda (\xi_i^{\mu})^3 + I_i^{\mu}, \text{ where } \zeta_i^{\mu} \text{ and } \\ \eta_i^{\mu} &\text{ are solutions of the linear equations } \\ b_1 \xi_i^{\mu} + b_2 \zeta_i^{\mu} + b_3 \eta_i^{\mu} + b_4 &= 0 \text{ and } c_1 \xi_i^{\mu} + c_2 \zeta_i^{\mu} + c_3 \eta_i^{\mu} + c_4 \\ &= 0 \quad (\mu = 1, \dots, p, i = 1, \dots, N). \end{aligned}$

The general solution *T* is expressed through a particular solution *R* and a solution *H* of the corresponding homogeneous equation so that T=R+BH where *B* is an arbitrary matrix. To obtain the matrices *R* and *H*, we will use in what follows the projection learning rule [20,21], which yields the equation $R_{ij}^{\mu}=R_{ij}^{\mu-1}-(\sum_k r_k^{\mu-1}v_k^{\mu})^{-1}(u_i^{\mu}+\sum_k R_{ik}^{\mu-1}v_k^{\mu})r_j^{\mu-1}$ and an analogous formula for H_{ij}^{μ} . Here $\mu=1,\ldots,p$ and vectors \mathbf{r}^{μ} are defined so as to satisfy the conditions $\sum_k r_k^{\mu}v_k^{\alpha}=0, \alpha=1,\ldots,\mu$ and $\sum_k r_k^{\mu}v_k^{\mu+1}\neq 0$.

To compute the vectors \mathbf{r}^{μ} we exploit outer products [22] of vectors $\mathbf{v}^1, \ldots, \mathbf{v}^{\mu}$ as follows [21]. Let us consider the components $V_{i_1\cdots i_m}$ of an axial skew-symmetric tensor $V:V_{i_1\cdots i_m} \equiv \sum_{1}^{N} \varepsilon_{i_1\cdots i_m k_1\cdots k_{\mu}} v_{k_1}^1 \cdots v_{k_{\mu}}^{\mu}$. Here $m \equiv N - \mu$ and $\varepsilon_{j_1\cdots j_N}$ are components of the fully antisymmetric tensor. For the case of linearly independent vectors $\mathbf{v}^1, \ldots, \mathbf{v}^{\mu}$ treated in what follows, there exists at least one nonzero component of the tensor V. Let $V_{n_1\cdots n_m} \neq 0$, $n_1 < \cdots < n_m$. Then take arbitrary values as the coordinates b_{n_1}, \ldots, b_{n_m} of a vector **b** and determine the other coordinates by the equation

$$b_j = (V_{n_1 \cdots n_m})^{-1} \sum_{i=1}^m b_{n_i} V_{n_1 \cdots n_{i-1} j n_{i+1} \cdots n_m}$$

where $j \in \{1, 2, ..., N\} \setminus \{n_1, ..., n_m\}$. Every such vector **b** is orthogonal to the vectors $\mathbf{v}^1, ..., \mathbf{v}^{\mu}$ and therefore we can set $\mathbf{r}^{\mu} = \mathbf{b}$. Thus the synaptic matrix *T* is determined not uniquely but, instead, through the set of free parameters and they are explicitly given. It is of interest to examine how the behavior of a network is changed as these parameters are varied while the FP's encoding MP's are retained.

We studied the learning and performance of networks when the number of neurons N=4, 6, or 8 and number of MP's p=2. For any set of MP's, such synaptic matrices were constructed that produce all types of stability. As the synaptic matrix has been adjusted so that MP's are stable FP's, the system can be treated as autoassociative memory [see Fig. 3(a) for an illustration] [23].

Of special interest here is the case when a FP encoding a MP loses its stability because the synaptic couplings are modified due to a change of parameters of the above learning rule [24,25]. The type of motion most often observed now is periodic oscillations. It happens that they are preceded by irregular transient processes. As parameters of the learning algorithm are varied, transient processes can become stable and CA's appear. They may either be placed around the unstable FP or consist of two such symmetric wings that one of them is located around the FP. The phase portraits of the CA's are similar to that shown in Fig. 1(b).

Inverse transmutation of a CA into a stable FP can obviously be done by an appropriate change of parameters of the learning rule. Another way is control of chaos, which can be caused by feedback with time delay [26] appearing in neural networks in a natural way. We used two models to investigate such a phenomenon. Namely, we replaced the



a network composed of N=4 neurons for the case when FP's are $\xi^1 = (-2.8, -3.1, -7.4, 5.7), \ \xi^2 = (-2., 5., 1.7, 7.1)$. The transfer function is $f(x) = \tanh(0.2x)$; $a_1 = -8$; values of the other parameters of a neuron are given in the text. (a) The synaptic matrix T has such values (see below) that ξ^1 and ξ^2 are stable FP's and the network operates as AM. (b) The synaptic matrix has such values that ξ^1 and ξ^2 are nonstable FP's and CA's occur around them. (c) The synaptic matrix is the same in (b) but time delay is introduced at time instant t = 450 ($\Delta t = 5$, $\tau_{21}^* = 0.05$, $T_{21}^* = -14$.) so that the current J'_i is used instead of J_i . (d) The current J''_i is used instead of J_i , the synaptic matrix is the same as in (b) and (c). Time delay is introduced at t=450 ($\Delta t_d=5$, $\tau_{21}^*=0.04$). [Values of elements of the synaptic matrix are the following (k=1,2,3,4): (a) $T_{1k} = (95.5068; 638.88; -665.71; -266.658), T_{2k} = (-699.411;$ $-882.021;1120.13;175.973), \quad T_{3k} \!=\! (652.134;627.987;-409.855;$ -105.62), $T_{4k} = (-66.2163;976.012; -887.861; -146.882);(b)$, (c),(d) $T_{1k} = (-176.792; -127.611; 167.126; -33.339), T_{2k} =$ (-176.364; -56.2778; 192.292; 33.9942), $T_{3k} = (653.519;$ $T_{4k} = (-545.998; -301.375;$ 638.28; -420.736; -109.835), 503.557;229.581)].

above equation for the neuron current J_i either by $J'_i(t) = \sum_j T_{ij} f(x_j(t)) + \sum_j \widetilde{T}_{ij} f(x_j(t - \tau_{ij}))$ or by $J''_i(t) = \sum_j T_{ij} f(x_j(t - \tau_{ij}))$ (we set the external current I_i to zero). For simplicity, only one term with time delay was usually introduced in fact. The corresponding quantity τ_{ij} (usually τ_{21} or τ_{11}) was linearly increased from zero to a fixed value τ_{ij}^* (taken to be in the interval 0.03, . . . ,0.15) during a time interval Δt_d (we chose as a rule $\Delta t_d = 5$ but a value of Δt_d was increased up to 25 to test the stability of effects observed). The additional synaptic couplings \widetilde{T}_{ij} were linearly changed from zero to fixed values T_{ij}^* (taken to be of the order $0.1T_{ij}$).

Usually, such a stabilization of a CA yields a PA but there exist values of parameters that a stable FP $\tilde{\xi}^{\mu}$ occurs. Depending on the parameters' values, this FP $\tilde{\xi}^{\mu}$ either coincides with the FP ξ^{μ} from which the CA has appeared or is a new FP close to ξ^{μ} . For the linear transfer function f(x)=x, the time interval Δt_s between switching on the time delay and stabilization of the FP $\tilde{\xi}^{\mu}$ was found to be $20, \ldots, 50$. This time can be divided into two phases, $\Delta t_s = \Delta t_{s1} + \Delta t_{s2}$, so that during Δt_{s1} the behavior of the system seems to be irregular while the second regime is an oscillatory relaxation towards the FP $\tilde{\xi}^{\mu}$. Duration of the first phase $\Delta t_{s1} = 10, \ldots, 20$ was found to be practically independent of the time delay parameter τ_{ij}^* whereas the quantity Δt_{s2} has a minimum at some $\tau_{ij}^* = \tau_{ij}^{*m}$ (of the order of 0.05). This minimal value of Δt_{s2} is equal to $10, \ldots, 30$.

The maximal value of distance $d(\xi^{\mu}, \tilde{\xi}^{\mu})$ between FP's ξ^{μ} and $\tilde{\xi}^{\mu}$ was about 2.7 whereas the distance $d(\xi^{\mu}, \xi^{\nu})$ between FP's encoding different MP's was varied from 0.1 to 30; usually $\max_{\mu} d(\xi^{\mu}, \tilde{\xi}^{\mu}) = \delta \max_{\mu,\nu} d(\xi^{\mu}, \xi^{\nu})$, where $\delta = 0.3, \ldots, 0.8$. For all the cases studied, the new FP $\tilde{\xi}^{\mu}$ lies inside the region in which the CA was located before introducing time delay.

The main properties of the transformation of the CA into a FP $\tilde{\xi}^{\mu}$ observed in the case of the current J'_i and the linear transfer function hold also if the current J''_i is used or the function $\tanh(gx)$ or $\Phi(x)$ is taken as f(x) [18,23,27]. Figures 3(c) and 3(d) illustrate this.

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Thus a stabilization of a CA due to feedback with time delay yielding a new FP $\tilde{\xi}^{\mu}$ in a neighborhood of the original FP ξ^{μ} leads to a transition *fixed point* $\xi^{\mu} \rightarrow chaotic attractor$ $\rightarrow new fixed point \tilde{\xi}^{\mu}$, which can be treated as a dynamical mechanism of AGI. It is worth noting that this scenario has been found in a simple enough model. Indeed, only three variables are used to describe the state of a neuron (that is the minimal number to admit CA's), only one simple nonlinear term λx^3 is included into their dynamical equations (1), even linear interactions between neurons [with the transfer function f(x) = hx] can be taken and even four neurons can constitute a relevant network. Obviously that such a complex system as the real brain can exploit the above dynamical mechanism of AGI under wide conditions.

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number of the free parameters equals to N-1, N-2, and N^2 , respectively). The most important case is when only one parameter is varied (usually, a component of the vector \mathbf{r}^1 was changed). The effect of destabilization of FP's can be observed under such variation.

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