Dynamics and kinematics of simple neural systems

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The dynamics of simple neural systems is of interest to both biologists and physicists. One of the possible roles of such systems is the production of rhythmic patterns, and their alterations (modification of behavior, processing of sensory information, adaptation, control). In this paper, the neural systems are considered as a subject of modeling by the dynamical systems approach. In particular, we analyze how a stable, ordinary behavior of a small neural system can be described by simple finite automata models, and how more complicated dynamical systems modeling can be used. The approach is illustrated by biological and numerical examples: experiments with and numerical simulations of the stomatogastric central pattern generators network of the California spiny lobster. © *1996 American Institute of Physics.* [S1054-1500(96)02103-9]

The dynamics of neural systems is an important biological discipline where physical and mathematical modeling can potentially provide a great deal of insight. Elaborate models can and have been developed based on considerations of the detailed neural structures; however, the approach taken in this work was to use a simple model to attempt to understand the basic purpose served by specific patterns of neural synchronization. A relatively simple neural system was explored here, namely the central pattern generators (CPG) that control the rhythmic activity in invertebrates. Experimental results for CPG neuron firings for the California spiny lobster were successfully modeled via a simple finite automata approach, without having to resort to complicated systems of ordinary differential equations.

I. INTRODUCTION

A. Levels of description

This paper is based on a short course of lectures in which biological experiments on small neuronal systems were presented along with physical approaches to the modeling of such systems. One of our aims has been to discuss the idea of neural system modeling at different levels, illustrated by concrete examples. It is well known that even a single, isolated neuron is a strongly nonequilibrium system with many degrees of freedom. Therefore a system of coupled neurons is inherently even more complicated. However, when considering them only on a relatively large timescale (only one order less than the characteristic time of the rhythmic patterns they produce), it is possible to use another level of description and model neurons as relatively simple dynamical systems. Such modeling of neuronal generators of nontrivial patterns offers the possibility of using the same approach as for circuits of any coupled generators (for example, electronic generators) and to answer some very important neurophysiological questions by using the experience of artificial network analysis in physics and engineering. In particular we consider two questions in detail: (i) stationary (ordinary) and adaptive behaviors of neural systems, and (ii) the simplest ways of modeling the ordinary behavior, so that the model not only makes correct ''predictions'' but is useful also for understanding the role of different types of coupling between neurons.

B. Motor control

How does a small insect like a centipede move all its legs in a highly coordinated manner, such that it may successfully overcome different obstacles without considering what leg to place in what position? How does a small mollusc like *clione*, the sea angel, move its wings to swim? And how does a tongue move so that we seldom bite ourselves, yet we are able to speak, whistle, chew, etc.? With different answers, perhaps, then are different versions of the same question related to the rhythmic movements of living organisms. How can such activities be performed, and what are the features of the nervous system that allow a living creature to produce rather complicated but well coordinated movements?

The vertebrate examples all involve the action of a huge number of neurons, and that is why they are hard to analyze in detail (at a low level, i.e. taking into account every neuron and every synapse). The control of rhythmic activity of invertebrates is performed by relatively simple neuronal sys-

tems called central pattern generators (CPG). The issues we address in this paper are the analysis of these small neuronal systems and the explanation of the way they operate by modeling them as simple dynamical systems.

Neurobiologists have made enormous progress in understanding the neuron's behavior at the cellular level—how neurons are organized, how they generate action potential, how neurons influence each other through synapses, how special chemicals—neuromodulators—act on neurons and change their behavior, etc. At the same time it has been studied how these cellular processes and their alterations are correlated with actual behavior of animals. However there is a gap between these two levels of analysis. Nonlinear dynamics may help to link cellular levels to behavioral levels by explaining how a network of neurons can produce the behavioral patterns (electrical signals that are sent to muscles to control them) observed in nature (see, for instance the review by Abarbanel *et al.*[1]).

The problem is extremely difficult for complex organisms (vertebrates). We actually know almost nothing about the detailed microcircuitry of the brain and the physiological differences between individual neurons. There are no experimental techniques presently available which can provide biologists with the details of brain circuitry, and assumptions about how neurons in the cortex, cerebellum, etc. are actually wired up must be rather speculative.

The production of behavioral sequences, as well as the processing of sensory information, requires coordination of many neural circuits. There has to be flexibility in the ways in which such circuits can be tuned and orchestrated as well as the way in which circuits can be combined because sensory neurons, central neurons (interneurons) and motor neurons can participate in various combinations during different behavioral patterns. Using the simplified nervous systems of invertebrates (simplified at least in terms of number of neurons), neurobiologists have overcome many of the obstacles which lie in the way of a detailed analysis of brain circuitry.

C. CPG networks

The structure of many small systems like CPGs are very well known (see Sec. II and examples therein). Biologists know how neurons connect with each other in detail. The question that arises is: What kind of mathematical objects are required to model CPGs? Because of the huge amount of experimental data, very detailed models can be (and actually have been) constructed. If one needs to know all the details of the transition processes, neuronal responds to the action of neuromodulators, a detailed model that consists of dozens of ordinary differential equations (ODEs) may be useful. If, on the other hand, one wants to understand only roughly why some neurons are synchronized in a particular fashion, much simpler logical considerations may be enough.

A nice example of the latter case is the swimming CPG of the mollusc *clione*. We would like to discuss this example because it is very simple and it gives the right basic idea how a CPG works. This small ''beast of prey'' swims with the aid of its wings. The CPG controlling the muscles driving *clione* wings has been investigated in detail.[2] This pattern generator is shown schematically in Fig. 1. Roughly speaking this generator consists of five neurons only (actually, there are more neurons but some of them behave so synchronously that they may be regarded as one). Neurons *a* and *b* are coupled by mutual inhibitory coupling, i.e., excitation of one neuron inhibits the other one, and by excitatory and inhibitory couplings with neuron *c*. When the latter is active it excites *a* and inhibits *b* neurons, i.e. helps *a* and *b* fire in antiphase. Moreover, *a* and *b* not only excite the motoneurons MN1 and MN2 (that contract and relax muscle) but each of them inhibits the motorneurons excited by the other. This construction allows MN1 and MN2 to work in antiphase with very high reliability, as needed for the coordinated work of muscles. The considerations presented above are very simple, however they allow one to make certain assumptions about how the system works without complicated mathematics but from general qualitative principles: mutual inhibitory coupling tries to enforce antiphase synchronization, and electrical or mutual excitatory coupling produces in-phase synchronization. These principles are based on experiments (e.g. Refs. 3 and 4) and confirmed by numerical simulations (e.g. Ref. 5).

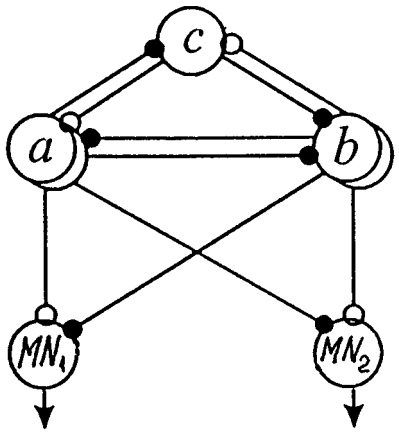

FIG. 1. The scheme of *clione* swimming CPG. Filled circles denote inhibitory synapses, empty ones—excitatory. MN1 and MN2 are motoneurons.

Thus, in some cases it is possible to give a logical description of the stable, ordinary behavior of a network.[6] Sometimes this is enough if one wants to understand the structure of a very simple CPG. Of course, for the study of details of neuronal dynamics, transition processes, etc., complicated models like systems of nonlinear differential equations should be used. However, if one is interested only in the general features of the stationary operation of a small neural system (i.e. in its kinematics, because in this case one considers only the motion on an attractor of the dynamical system, so the trajectory is fixed), verbal description or models like simple finite automata can be helpful.

Nerve impulses (spikes) are brief, with a relatively constant size and shape. Consequently, the information is contained in trains of several spikes that form a burst. From this point of view, the modeling of a neural network by symbolic

dynamics is correct and convenient. However, as it was shown in many model experiments, the dynamics of the fast and slow motions are connected and important interactions occur between these two types of motion. For example, the bursting period and phase lag between neurons may depend on the strength of coupling. A symbolic (and especially logical) approach is not an efficient description of these phenomena, because when we take into account all important information about dependence on the synaptic current of the form of spikes, the distance between them, etc., symbolic models become more complex and harder to simulate numerically than typical ODE models.

II. ARCHITECTURE AND OPERATION OF SMALL NEURAL SYSTEMS

One type of neural pattern particularly amenable to analysis is the centrally produced cyclic motor pattern. This pattern consists of rhythmic bursts of impulses in the appropriate motor nerves. When these bursts reach and activate the muscles, sequential behaviors like locomotion, chewing, flying, etc., are generated. Such behaviors are produced by small groups of synaptically coupled neurons—central pattern generators, an important characteristic of which is that their activity is autonomous, i.e. produced without the need for sensory feedback so that the portion of the nervous system which is responsible for their generation can be removed from the animal and the CPG can be studied in isolation. That is why CPG is a very convenient neuronal system for investigation. Of course sensory feedback does exist and may play an important role in regulation of motor patterns but is not responsible for their existence.

Here we consider one example of a CPG—lobster stomatogastric CPG (see Refs. 7 and 8). The stomatogastric CPG of the California spiny lobster *Palinurus interruptus* innervates its foregut. The stomach contains three ossicles which function as teeth, two lateral and one medial. They are controlled by the gastric mill CPG located in the stomatogastric ganglion which sits in an artery on top of the stomach. The ganglion contains about thirty neurons which have large cell bodies and are clearly visible. The neurons can be identified, removed from ganglion and cultured. There are four different regions of foregut: the esophagus, the cardiac sac, the gastric mill containing teeth and pyloric region where food particles are filtered and sent to more caudal regions of the gut. The stomatogastric ganglion can be divided into two networks—gastric and pyloric that control gastric mill and pyloric filter respectively. Much is known about the neural basis of their behavior, less is known about the esophagal and cardiac sac behavioral patterns.

The pyloric behavior consists of a dilation of the pyloric region due to PD (pyloric dialator) and VD (ventricular dialator) cells firing. Then there is a sequential constriction of the region from front to back as the LP (lateral pyloric), IC (inferior cardiac) and PY (pyloric) cells fire. This pattern can be observed at a frequency of about 0.5 Hz in both isolated and intact systems. Because there are so few neurons involved, all of the neuron types for each CPG can be recorded simultaneously with intracellular microelectrodes. The electrical activity consists of slow oscillatory potentials with attenuated action potentials riding on the depolarizing phase of each burst. All of the neurons are motor neurons with the exception of the AB (anterior burster) neuron which is an interneuron, i.e. it sends information only to other nerve cells.

The basic functional circuitry for the stomatogastric ganglion was worked out by recordings from pairs of identified stomatogastric neurons. Fortunately, intracellular recordings from the cell bodies of these neurons display subthreshold activity so that monosynaptic connections between cells can be established. By passing current between neurons, connections between them can be easily determined. This has led to a complete description of the two pattern generators in the terms of their synaptic connectivity as shown in Fig. 2. For convenience we have also pictured a scheme of the pyloric network separately in Fig. 3. Note that the majority of the connections are inhibitory and that in addition to the pattern generating neurons, some of the identified neuromodulatory and sensory cells are also included. Although the two networks function more or less independently of each other and of sensory inputs, the extensive connections between two groups and sensory input may play a crucial role for an animal in some non-standard situations (see Refs. 3, 7–11 and others for experiments).

III. THE MODELS ONE MAY NEED TO DESCRIBE CPG DYNAMICS

A. ''Adequate'' models

The state of a neuron is determined by nonequilibrium diffusion of different ions through its membrane. Consequently its activity should, in general, be modeled using a kinetic description.[12] However, there is no need for such a description as long as we are interested in the neuron only as a generator of low-frequency electric pulsations. Furthermore, the cellular membrane can often be considered as an equipotential surface, so the variables describing the state of a neuron (membrane potential, ionic concentrations, etc.) are functions of time only. Therefore, for the construction of a reasonable model it is sufficient to apply ordinary differential equations for the dynamical variables: membrane potential, macroscopic ionic currents and concentrations. Here we exploit the fact that the characteristic time scale T of the electric activity of the neuron is much larger than the characteristic time of the kinetic processes. A neuron can be regarded as a nonlinear electric circuit. The energy sources for operation of this dissipative system are biochemical processes of metabolism. Thanks to the feedback that open and close ionic channels in the membrane at the respective phases of electrical activity, the state of neuron corresponding to the resting potential may become unstable and the neuron becomes a generator. Such a generator may be regarded as a dynamical system in which microscopic kinetics is revealed only as small fluctuations.

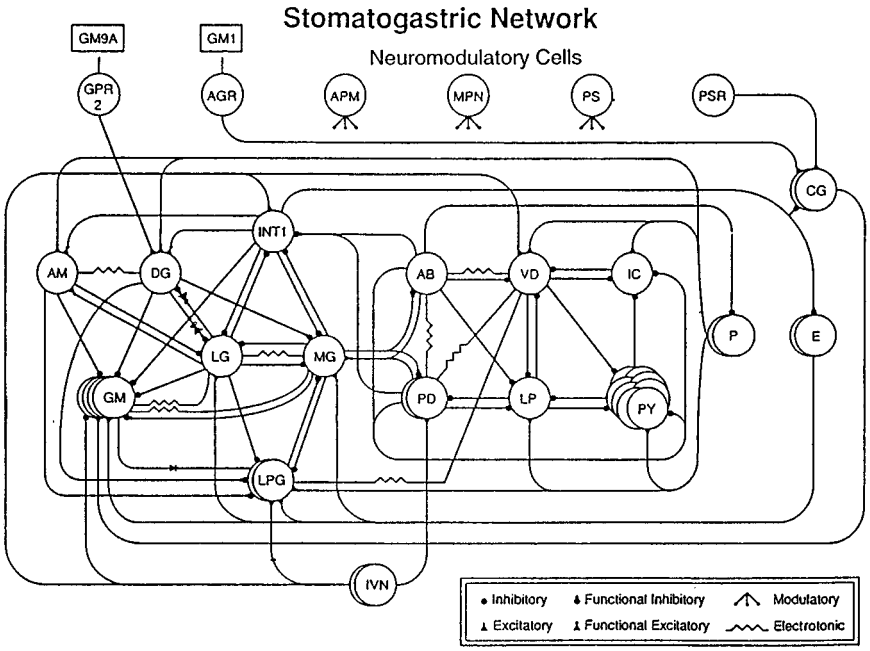

FIG. 2. The scheme of the lobster stomatogastric ganglion.

This dynamical approach is supported by nonlinear time-series analysis of the membrane potential—the calculation of embedding, correlational and Lyapunov dimensions, Lyapunov exponents, etc. Experiments performed in different laboratories indicate that the normal electrical activity of a single isolated neuron is dynamical chaos (e.g. Refs. 5 and 13). For example let us consider an isolated LP neuron of the lobster stomatogastric ganglion (see Sec. II).[5] In this preparation it is possible to obtain very long time series of membrane potential. The phase portrait of this behavior displayed in Fig. 4 has been reconstructed from data following the well-known Takens procedure.[14] It is a strange attractor that can be projected onto a three-dimensional space. The Lyapunov dimension of this time-series is about 2.8 and the embedding dimension obtained by the method of false nearest neighbors method is 3–4. (For a discussion of calculations of dimensions and identifying a dynamical system from observed data see, for example, Ref. 15.) Hence, the electrical activity of this neuron takes the form of a low-dimensional chaos, and the neuron can be modeled as a nonlinear dynamical system. The same situation arises with many other CPG neurons (see, for example, the work of Hayashi and Ishuzuka[13] and others).

B. Some examples of ODE models

In this section we discuss some examples of differential equations used for neuronal modeling. Neurons from many small neural circuits like CPGs are often described by conductance-based models—a generalization of the classical Hodgkin–Huxley formalism.[16] This generalization is based on taking into account different ionic channels and dependences of the membrane conductances (for different ionic

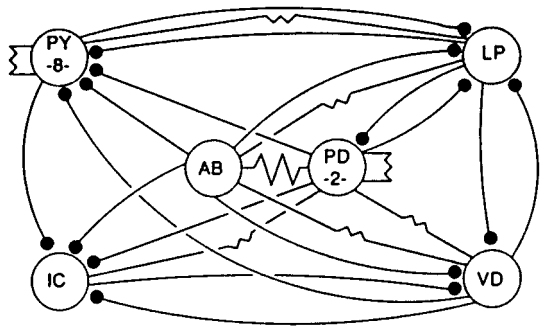

FIG. 3. The scheme of the pyloric network.

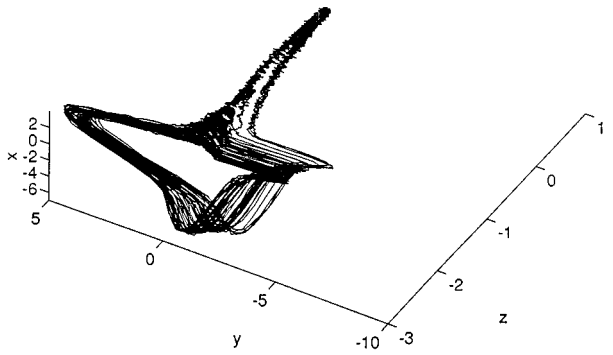

FIG. 4. An attractor reconstructed from the time series (see Fig. 5c) in three-dimensional phase space.

currents) on concentrations, membrane potential, etc. The general form of such models is

$$C\frac{dV}{dt}=I-\sum_{i=1}^{N} g_i a_i^{p_i}(t) b_i^{q_i}(t)[V(t)-V_i],$$

$$\frac{da_i}{dt}=(a_{\infty i}(V)-a_i)/\tau_{a_i}(V), \tag{1}$$

$$\frac{db_i}{dt}=(b_{\infty i}(V)-b_i)/\tau_{b_i}(V), i=1,2,3,\ldots,N,$$

where V is the membrane potential, C is the membrane capacity, and I is. Here i denotes different ionic species, and a and b describe states of ionic channels, $a_\infty(V)$, $b_\infty(V)$ and $\tau(V)$ are some sigmoidal functions. There are many models with $N\sim 10$ or even more. They describe neuronal behavior very precisely, but they are high-dimensional and contain a lot of often unknown parameters. As a consequence they are hard to analyze in detail (see, for example, Refs. 17–22). The other feature of this type of model (as well as of the living cells they describe) is that the actual behavior very often is a low-dimensional one. Therefore, the dynamical variables in system (1) are not really independent, rather they behave like master–slaves variables. Consequently, a lot of important effects (except those connected with interactions of particular ionic currents) can be described by low-dimensional ODE models (of conductance-based type[22] or of a phenomenological form).

One of the frequently used low-dimensional models is that of Rose and Hindmarsh[23]

$$\frac{dx}{dt}=y+ax^2-bx^3-z+I,$$

$$\frac{dy}{dt}=c-dx^2-y, \tag{2}$$

$$\frac{dz}{dt}=r[s(x-x_0)-z],$$

where x is a membrane potential, y represents fast currents (e.g. sodium and potassium), z represents a slow current (e.g. Ca^{2+}–current), and I is an external current. The Rose–Hindmarsh model and its modifications can describe many effects observed in neural systems.

The type of model that is most appropriate clearly depends on what details and features of the dynamics one needs to know. It is possible to simplify the models further and use two-dimensional systems of differential equations (like the conductance-based Morris–Lecar model[24] or phenomenological Wilson–Cowan model,[25] FitzHugh–Nagumo model[26,27]), integrate-and-fire, phase oscillators, Hopfield-like models and so on. Usually very simple models are used to analyze large, complex neuronal assemblies (like human cortex, that contains about 5×10^9 neurons), because these assemblies simply cannot be analyzed in practice by using complicated models with dozens of variables and hundreds of parameters. Furthermore, we have no reason to believe that the complexity in models will bring more accurate results, because we do not know the details of the cortical circuitry.

The same or similar simple models may be used (and actually are used) in modeling of small neural systems. When we consider a stationary regime and our aims are limited to understanding, say, how phase shifts between the potentials of various neurons are formed, we can use very simple models. In some cases, derivation of relatively simple models from more complicated and precise ones can be done with mathematical rigor, for example, by averaging of motions on different time-scales (e.g. see Refs. 28 and 29).

In the next section we will introduce a class of very simple models (finite automata) in order to describe basic features of CPG networks (in particular, phase shifts between spiking-bursting activity, the length of these phases, and the relative synaptic strength) qualitatively correctly. We will not perform a rigorous reduction from complex ODE models to simple finite automata. Actually, there is no reason for such a derivation because the phenomenological model helps to achieve our ''modest'' aims.

IV. FINITE AUTOMATA DESCRIPTION OF SMALL NEURAL NETWORKS

A. What are finite automata models useful for?

Here we introduce a simple finite automata model for neurons of CPG-like small systems. This phenomenological model is based on different experiments with different CPGs. The model is a three-state finite automata that captures spiking, bursting and resting activity, which is usually observed in CPG neurons.

The utilization of logical models and finite automata in modeling of neurons is not a new one (e.g. see Refs. 30 and 31). Simple models of neural systems have been used for many years. Starting with the work of McCulloch and Pitts,[32] a significant number of papers have appeared, but almost all of them have been dedicated to the problems of memory, learning and other phenomena in large neuronal ensemblies. A few papers have been dedicated to the study of CPGs, e.g. Ref. 33 — logical approach, Ref. 34 — Hopfield-like models. We want especially to mention the work of Thompson[35] who used a two-state probabilistic model of each neuron to describe the dynamics of the pyloric network, because we consider the modeling of the pyloric network too. Here we develop a new approach suggested by one of the authors.[36] It generalizes from two states to three in order to approximate the CPG neurons as much as possible without missing the transparency presented in these automata-type models. We show how starting from a particular set of dynamical rules belonging to each of these three states the characteristic behavior of some CPG is accomplished. To reproduce the experimental results with good agreement means in this case that the qualitative features utilized in finite automata are fundamental in the dynamics of these neurons.

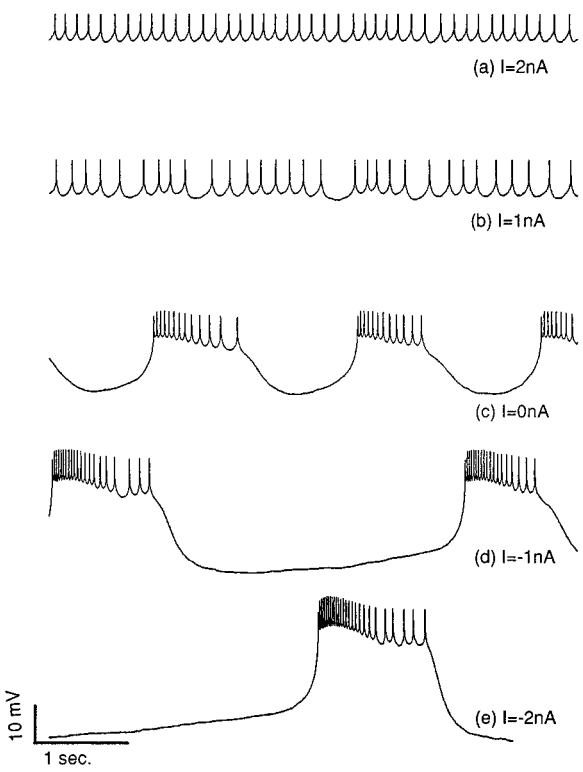

FIG. 5. (a)–(e) Membrane potential of isolated LP neuron, depending on the value of input constant current I.

Networks of such models can qualitatively correctly represent the length of different phases in neuronal behavior, phase shifts between different neurons, relative strengths of synaptic coupling. For example, they can be used for obtaining correct relative values of these strengths. They are much easier to use in this case than systems of ODEs, because they have fewer parameters and are simpler in numerical simulations. Therefore, such simple models are fairly convenient. They allow us to concentrate our attention on the main features of the network (types and architecture of connections) and network's behavior.

B. Spiking-bursting symbolic neuron

As we have noted in Sec. II, the characteristic behavior of a CPG neuron shows three well differentiated states: the resting, the bursting and the spiking state. Along with these three states we have to set an ordered switch between them according to one control variable functionally equivalent to the electric current I. Based on different experiments (see Sec. II, Fig. 5), the model assumes three states ($f=0,1,2$), regulated by a variable that represents the current:

$$\begin{aligned} &\text{spiking} \quad f=2, \quad I\geqslant 1,\\ &\text{bursting} \quad f=1, \quad 0<I<1,\\ &\text{resting} \quad f=0, \quad I\leqslant 0. \end{aligned} \tag{3}$$

Each state has its own set of rules and is represented by particular variables: f is a common variable for all states that denote these states, Φ_S is the phase of spiking state (describes fast motion), and Φ_B is the phase of bursting state (describes slow motion).

The resting state is trivial and has no rules for time evolution: $f=0$ is a constant in time.

The spiking state is characterized by an amplitude, $f=1+f_S$, and a phase, $\Phi_S \in [0,g_S(I,w_S)]$, where w_S is an integer that represents the nominal spiking period for a particular current $I=I_o$ [in the absence of any input current the oscillator spikes every $g_S(I_o,w_S)$ units of time]. The phase evolves in time as $\Phi_S(t+1)=\Phi_S(t)+1$ if $\Phi_S\leqslant g_s$, otherwise $\Phi_S=0$. f_S is written as

$$f_S=\begin{cases} 1, & 0\leqslant\Phi_S<rg_S(I,w_S),\\ 0, & rg_S(I,w_s)\leqslant\Phi_S\leqslant g_S(I,w_S), \end{cases} \tag{4}$$

and for the integer decreasing function $g_S(I,w_S)$ we use

$$g_S(I,w_S)=\begin{cases} w_S-\alpha I w_S, & w_S-\alpha I w_S<w_{limit},\\ w_{limit}, & w_S-\alpha I w_S\geqslant w_{limit}, \end{cases} \tag{5}$$

where typically $\alpha\sim 0.1$ and $r\sim 0.2$ is chosen.

The bursting state is described by a second phase $\Phi_B \in [0,g_B(I,w_B)]$, where w_B is an integer that represents a nominal bursting period for a particular nominal current $I=I_o$ analogously with Φ_S. We used the following form of g_B: $g_B=w_B(2-\beta I)$ with $\beta\sim 1$. The time evolution of the bursting phase is the same as that of spiking phase and

$$f=\begin{cases} 1+f_S, & 0\leqslant\Phi_B<m_B(I,w_B),\\ 0, & m_B(I,w_B)\leqslant\Phi_B\leqslant g_B(I,w_B), \end{cases} \tag{6}$$

in which $m_B(I,w_B)=Iw_B$ is chosen. Let us note again that these functions are a general qualitative approximation.

There is an additional subject of importance when we are talking about understanding and modeling of real neurons — noise. Noise exists in any real neuron, therefore the question arises: Is noise a principal part of neural life or not, and should we introduce noise in our model? The answer is affirmative. Numerical experiments have showed that there exist some fine attractive structures in the phase space of the model with very small basins of attractions. They correspond to pathological regimes that were never observed in experiments. Weak noise added to the model destroys such pathological regimes. We think that it is some kind of indirect argument that noise is also important for the normal operation of living nerve cells. In numerical simulations we introduce noise in the following fashion:

$$w_B=w_B^0+\sigma\eta(n), \tag{7}$$

where σ characterizes the amplitude of noise, which is about 5% of w_B^0 [$\eta(n)$ is normalized to one random Gaussian signal with zero mean].

Based on some experiments on the phenomena called rebound potential[37,38] it is reasonable to introduce the rule for nonlinear phase change as

$$\Phi_B(t+1) = \begin{cases} 0, & \Delta I(t) > C_{th}, \\ \Phi_B(t)+1, & -C_{th} \leqslant \Delta I(t) \leqslant C_{th} m_B(I(t), w_S), \\ \Delta I(t) < -C_{th}, \end{cases} \tag{8}$$

where C_{th} is the threshold at which the nonlinear phase change takes place, and $\Delta I(t)$ represents the incoming current received by the neuron from other neurons through synapses.

The symbolic neuron model may be summarized in the form

$$\begin{aligned} & I(t) = I_o + \Delta I(t), \\ & f(t) = \begin{cases} 2, \ I(t) \geqslant 1, \Phi_S(t) \in a(I(t)); & \text{or} \quad I(t) \in f(I(t)), \Phi_S(t) \in a(I(t)), \Phi_B(t) \in b(I(t)), \\ 1 \ I(t) \geqslant 1, \Phi_S(t) \in c(I(t)); & \text{or} \quad I(t) \in f(I(t)), \Phi_S(t) \in c(I(t)), \Phi_B(t) \in b(I(t)), \\ 0 \ I(t) \leqslant 0; & \text{or} \quad I(t) \in f(I(t)), \Phi_B(t) \in d(I(t)), \end{cases} \\ & \Phi_S(t+1) = \begin{cases} 0, & \Phi_S(t) > g_S(I(t)), \\ \Phi_S(t)+1, & \text{``rest''}, \end{cases} \\ & \Phi_B(t+1) = \begin{cases} 0, & \Phi_B(t) > g_B(I(t)) \quad \text{or} \quad \Delta I(t) > C_{th}, m_B(I(t)), \\ \Delta I(t) < C_{th}, & \\ \Phi_B(t)+1, & \text{``rest''}, \end{cases} \end{aligned} \tag{9}$$

where the intervals a, b, c, d, and f are given by

$a(I(t)):[0, r g_S(I(t)))$,

$b(I(t)):[0, m_B(I(t)))$,

$c(I(t)):[r g_S, g_S(I(t))]$,

$d(I(t)):[m_B(I(t)), g_B(I(t))]$,

$f:(0,1)$.

Now we will use this system to model neural networks.

C. Network modeling

Obviously, the modeling of synaptic connections is an important part of neural systems modeling. It is known that the transmission of information through synaptic connections is mainly due to spikes. A spike generated in the cell body arrives via the axon to the synaptic terminal and releases neurotransmitters that after a brief delay reach the postsynaptic membrane and change its permeability for different ions and a postsynaptic potential is generated. Another spike arriving a little bit later generates another postsynaptic potential which is added to the previous one. The synaptic connection in our model counts the number of spikes that arrive at the cell inside some time interval. The change in the current for the cell number i is

$$\Delta I_i(t) = -(f_i(t) - f_{rev}) \sum_{j=0}^{M} s_{ij} N_j(t - r w_S, t - \tau), \tag{10}$$

where M is the number of cells in the network, s_{ij} represents the synaptic strength, $N_j(t - r w_S, t - \tau)$ counts the number of spikes that were generated in j-cell from time $t - \tau$ to time $t - r w_S$, and τ is integer number (thus we have finite automata with τ-memory step iteration). The variable f_{rev} represents the reverse potential of the synaptic connection, $f_{rev} = 0$ for inhibitory and $f_{rev} = 2$ for excitatory synapses. This model applies for chemical synapses only. In fact they are the most widespread in CPGs. Still the modeling of electrical coupling is a minor point of our approach. We suppose that mutual excitatory coupling (which never occurs in CPGs, usually excitatory coupling is only in one direction) can model an electrical synapse. This is supported by analysis of different experimental data and by numerical simulations with more complicated models. Several authors (e.g. Refs. 3–5) conclude that electrical coupling and mutual excitatory coupling have the same functional role—enforcing of in-phase synchronization (while mutual inhibitory coupling tries to produce out-of-phase synchronization).

The model will be used to simulate the behavior of the lobster pyloric CPG described in Sec. II. Note that the AB and both PD neurons are strongly coupled by electrical synapses, and therefore are always synchronized in phase. The same situation applies to the group of eight PY neurons—they are strongly electrically coupled, always in-phase synchronized, and consequently behave like one unit. In order to simplify calculations we used a pyloric network composed of only five model neurons: VD, LP, IC, PY and AB/PD. The results of the calculations presented in Fig. 6 can be compared with direct experimental measurements[9] reproduced here in Fig. 7. (Particular values of parameters in this simulation were the following: nominal current I_0: 0.4, 0.5, 0.8, 0.4, 0.6; bursting period w_B: 437, 437, 583, 437, 500; spiking period w_S: 28, 46, 23, 20, 21, for PY, IC, VD, LP and AB/PD neurons respectively.) It is evident that our finite

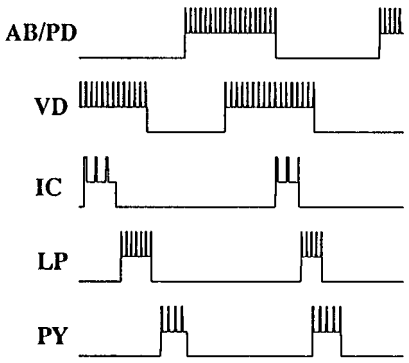

FIG. 6. The behavior of finite automata neurons of the simplified pyloric network (traces of the ''membrane potential'' f).

automata neurons are able to capture the two main features of behavior of the real pyloric network—the phase shifts between neurons and the period of the bursting phase. Based on the chosen parameters, we can infer the relative strengths of different synapses.

Let us note that the type of synchronization between some neurons is not obvious even though we know the structure of CPG. For example, the VD neuron is electrically coupled with PD and AB neurons. But they are synchronized out-of-phase. It may happen because the electrical coupling is weak enough. IC and VD neurons are mutually inhibitory coupled but fire in phase. One may suppose that in-phase synchronization will disappear if one will make the coupling stronger. It is not easy to prove in biological experiments. We performed numerical simulations with our model network and supported the supposition.

Such a manipulation with network parameters can be continued. By changing the synaptic coefficients s_{ij} in the model, we can predict what happens when neuromodulators (chemicals that modify the synaptic strengths) are applied to the stomatogastric ganglion. The usage of a finite-automata model is a simple job. One may easily change network parameters in order to understand their significance. To do it in a physiological experiment on a living CPG is a very hard or even impossible, in some cases, task. Therefore, the model described above is a nice tool for understanding the dynamics of the lobster pyloric CPG and other CPGs. We would like to note again that models like (4) are rather rough models of living networks so they may capture only rough features of the neuronal system's behavior.

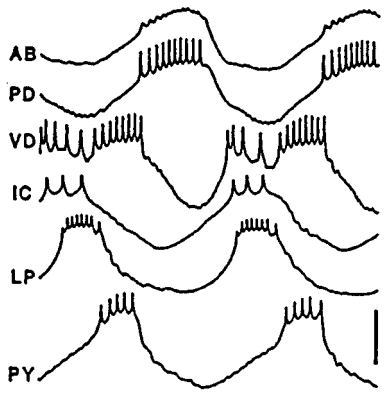

FIG. 7. The behavior of a living pyloric network (traces of the membrane potential) (Ref. 9).

V. CONCLUSION

The main role of a CPG is the production of rhythmic patterns. First of all, CPGs create motor patterns and only then modify them according to different needs (adaptation, control, etc.). To model this basic, fundamental function of CPGs, very simple dynamical systems like simple finite automata can be constructed. We use the word ''kinematics'' in the title of the paper meaning that we consider this stable unaltered motion of a living neural network, which is an inherent action (production of behavioral patterns) and is defined only by some key features of the neurons and types of coupling. It corresponds only to the motion on the attractor of the system and in this sense the trajectory of motion is fixed. We have demonstrated here, that models like simple finite automata can be very helpful for description of such a behavior. If one wants to understand alterations of these basic behaviors (i.e. to know how CPG can not only generate but also process information) more complicated dynamical systems are helpful. In this case it is more convenient to use ordinary differential equations, which one can naturally obtain in a rational manner from more detailed phenomenological approach.

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