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Neural Network Model Carrying Phase Information with Application to Collective Dynamics

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A network of periodically bursting model neurons is proposed. Its unique feature is a complex representation of the cell variables and also of the synaptic matrix. In the strong-coupling limit, the model recovers the traditional neural network model of simple on-off units, while in the weak-coupling limit it reduces to the network of smooth phase oscillators. In the special case of all-to-all excitatory coupling, some numerical and analytical evidence is provided for the occurrence of global phase locking. More complicated collective behavior such as clustering is also discovered numerically. Stimulus-evoked collective oscillations as observed in the cat primary visual cortex are explained within the present framework.

Recent electrophysiological studies revealed that neuronal responses with oscillatory modulation of the spiking frequency occur in various parts of mammalian brain.[1),2)] It has hence been suggested that multiple sensory inputs with some mutual similarity could be linked through the phase locking of this sort of neural oscillations, and that such linking could be crucial to the early stage neural information processing.[3)] Unfortunately, however, traditional neural network models composed of the simple McCulloch-Pitts elements or of their continuous analogues do not seem to provide a suitable framework for testing the above hypothesis nor for evaluating the computational power of oscillatory neural networks in general. This is because such models ignore from the outset the basic fact that a single neural cell itself can behave as an oscillator and hence carry phase information. It is not only meant here that cell membranes are capable of generating a periodic train of action potentials. More importantly, they are known to exhibit quite commonly the periodic bursting, i.e., alternation between the period of rapid spiking and that of quiescence.[4),5)] This second form of neural oscillation is of our main concern in the present paper.

Recently, some researchers studied oscillator networks in relation to sensory information processing in neural fields.[6),7)] They used relatively simple oscillators such as the phase oscillators which are quite unlike the processing units postulated in the usual network models. It may be wondered if it is not too busy for a neuron to behave in one case like an Ising spin as in the Hopfield model[8)] while in the other case like a smooth phase oscillator or even like an XY spin as in Sompolinsky et al.'s recent model.[6)] Is not there any way of integrating these seemingly conflicting aspects of a neuron into one simple dynamical unit? Once this has been done, one may be able to construct a new network model possessing high dynamical flexibility,

yet being simple enough to permit extensive mathematical and numerical analyses. This is exactly what we attempt in this paper. An earlier attempt along the same line was due to Abbott.[9] The main difference between his and the present approaches lies in the way of modeling the internal oscillatory dynamics of a cell. His model is based on a reinterpretation of the FitzHugh-Nagumo dynamics in such a way that the respective stable branches of the sigmoidal manifold are associated with the active and silent phases of a bursting neuron. The bursting mechanism assumed by Abbott is similar to that in Chay and Keizer's ordinary-differential-equation model[4] for insulin secreting β-cells of the mammalian pancreas. In that case, the slow dynamics of the variable controlling the burst generation is basically relaxational but strongly influenced by the action potential itself. In contrast, what we do in the present paper amounts to an extreme simplification of Plant's model[5] for pacemaker neurons of the Aplisia abdominal ganglion for which the slow subsystem forms an autonomous independent oscillator. As was discussed by Rinzel and Lee,[10] the above gives the two representative mechanisms for bursting. From a mathematical viewpoint, the model based on the second mechanism seems to be more naturally related to the phase oscillator model, and is easier to analyze especially for collective behavior.

We present our network model by contrasting it with the conventional one. For comparison's sake, the latter is expressed in terms of an auxiliary variable x_i in the form

$$S_i(t)=F(x_i(t)-h)\,, \tag{1a}$$

$$x_i(t+1)=\sum_j J_{ij}S_j(t)\,, \tag{1b}$$

where $S_i(t)$ represents the firing rate of the i-th cell averaged over some short interval about time t. The output function $F(x)$ is an increasing function of x changing abruptly between 0 and 1 near $x=0$, so that h represents the threshold value of x_i for depolarization. The summation in Eq. (1b) extends over all cells connecting to the i-th cell.

Besides x_i, we postulate in each cell an extra degree of freedom y_i, and this pair of cell variables defines a *complex potential* $z_i(t)$ with amplitude $r_i(t)$ and phase $\phi_i(t)$. Our model then takes the form

$$S_i(t)=F(\mathrm{Re}\, z_i(t)-h)\,, \tag{2a}$$

$$z_i(t+1)=z_i^*(t)e^{i\Omega}+\sum_j C_{ij}S_j(t)\,, \tag{2b}$$

where $z_i^*(t)\equiv r^*\exp(i\phi_i(t))$ is the reduced potential with fixed amplitude r^*. In aforementioned Plant's model,[5] x_i may be interpreted as intracellular free calcium concentration, and y_i as the slow conductance for calcium. Intrinsic oscillatory dynamics of the cell is implemented by the first term on the right-hand side of Eq. (2b). The last term in the same equation has the usual form of synaptic coupling, except that the synaptic efficacies C_{ij} are supposed here to be complex, i.e., $C_{ij}=|C_{ij}|\exp(i\alpha_{ij})$.

The dynamical rule in Eq. (2b) tells that the transformation $z_i(t)\to z_i(t+1)$ can be decomposed into three elementary steps as $z_i(t)\to z_i^*(t)\to z_i^*(t)\exp(i\Omega)\to z_i^*(t)$

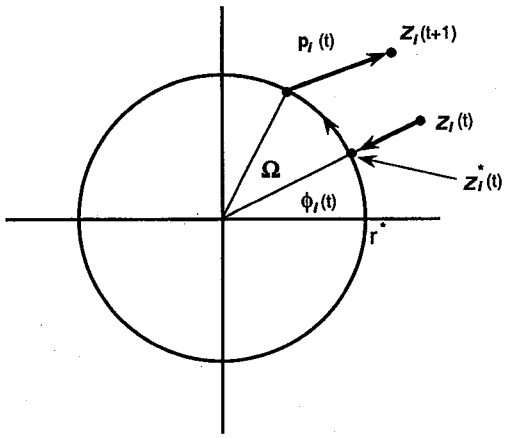

Fig. 1. Dynamical rule producing $z_i(t+1)$ from $z_i(t)$ in the complex plane. Three elementary steps indicated by the arrows are involved.

$\times\exp(i\Omega)+p_i(t)\equiv z_i(t+1)$, where $p_i(t)=\sum_j C_{ij}S_j(t)$ (see Fig. 1). The first step corresponds to the rapid relaxation of z_i to its native attracting cycle which is a circle of radius r^* in the complex plane. Here the phase of z_i is preserved, which is consistent with the general fact that autonomous oscillators lack restoring force against phase disturbances. The second step corresponds to the rotation of z_i along the circle by an angle Ω, reflecting the oscillatory nature of z_i. Finally, the input p_i kicks z_i out of the circle to give $z_i(t+1)$. Note that p_i generally changes the phase of z_i as well as its amplitude.

An alternative way of modeling the internal oscillatory dynamics would be to assume the form

$$z_i(t+1)-z_i(t)=\epsilon\{(1+i\Omega)z_i(t)-(1+ic)|z_i(t)|^2z_i(t)+\sum_j C_{ij}S_j(t)\}\,, \tag{3}$$

or its time-continuous version ($\epsilon\to 0$), namely, coupled Ginzburg-Landau oscillators. The two models are expected to lead to similar behaviors. In the present study we will work with the first form (2b).

Our model can be generalized in a number of ways. For instance, h, r^* and Ω may become cell-dependent parameters, and some random perturbations or more general external stimuli may be included in Eq. (2b).

There are two trivial limits in our model. Suppose first that the oscillation amplitude of z_i is vanishingly small or, equivalently, the total input p_i is strong enough to mask the oscillatory nature of z_i. Then, by putting $r^*=0$, the conventional model is recovered where $x_i(t)$ is identified with $\mathrm{Re}z_i(t)$ and J_{ij} with $\mathrm{Re}C_{ij}$ ($=|C_{ij}|\times\cos\alpha_{ij}$). If seen from our *complex synaptic matrix* viewpoint, a given cell j is excitatory or inhibitory according to $|\alpha_{ij}|<\pi/2$ or $|\alpha_{ij}|>\pi/2$, respectively. As another limit, consider single-cell dynamics by switching off the coupling. Our model then reduces to

$$S_i(t)=F(r^*\cos\phi_i(t)-h)\,, \tag{4a}$$

$$\phi_i(t+1)=\phi_i(t)+\Omega \tag{4b}$$

or $\phi_i(t)=\Omega t$ for Eq. (4b). For simplicity, let $F(x)$ be represented by the unit step function $\theta(x)$, where $\theta(x)=1$ for $x\geq 0$ and 0 otherwise. Then, under the condition $|h|<r^*$, active phase ($S_i=1$) and silent phase ($S_i=0$) alternate. A periodically bursting neuron has thus been modeled in a simplest way. Whether this condition for bursting is satisfied or not, $\mathrm{Re}z_i$ oscillates smoothly like $\cos\Omega t$. Such smooth oscillations of membrane potential are commonly known under the name of *slow waves*. They can persist even when tetrodotoxin is used to suppress the spike, and hence some

researchers postulated an autonomous oscillator functioning independently of the action potential.[5] We follow exactly the same idea.

The synaptic coupling is now switched on. Since too strong coupling leads to the usual network model as noted above, our main concern below will be the case of relatively weak coupling such that the oscillatory nature of the cells could hardly be masked. What we expect then is that the cells exchange phase information and, through mutual adjustment of the timing of bursts, a coherent temporal pattern of activity be created in the network. In order to demonstrate this possibility, some numerical simulations were carried out for Eqs. (2a, b). Without loss of generality, we put $r^*=1$ hereafter. Our numerical study is restricted to the special case of all-to-all excitatory coupling, so that we put $C_{ij}=N^{-1}C(1-\delta_{ij})$, where $C=|C|\exp(i\alpha)$, $|\alpha|<\pi/2$ and N denotes the population size. The same form of coupling in the conventional model will be of little dynamical interest, except possibly for the occurrence of collective bistability. In the actual simulation, Ω was replaced by cell-dependent quantities Ω_i, the latter being assumed to obey a Lorentz distribution $L(\Omega-\Omega_0;\ \gamma)\equiv\gamma[2\pi((\Omega-\Omega_0)^2+\gamma^2)]^{-1}$. This form of frequency distribution, as well as the assumption of all-to-all excitatory coupling, is particularly convenient when our numerical results are compared with analytical ones. We also assumed $F(x)=\theta(x)$. Recent extensive studies on large populations of coupled phase oscillators clarified that collective oscillations easily arise in the populations of globally coupled oscillators with frequency distribution.[11),12)] Our particular concern here is whether the same is true for our network model.

Figure 2 shows a typical case in which a domain of mutually synchronized cells develops out of the state of complete incoherence. Initially, the phases of bursting are totally random, while in a period of a few oscillations a major part of the entire population comes to behave in a coherent fashion.

The degree of global synchronization in sufficiently large populations may be quantified by the average of the reduced potential, i.e., $Z(t)\equiv N^{-1}\sum_j z_j^*(t)$. A slightly more convenient measure will be the order parameter R defined by

$$R=\overline{X\dot{Y}-Y\dot{X}}\,, \tag{5}$$

where X and Y are the real and imaginary parts of Z, the bar denoting a long-time average, and the dots should be understood as $\dot{A}(t)=A(t)-A(t-1)$. Since collective oscillations imply a closed orbit in the complex Z-plane, their absence/presence may be indicated by vanishing/non-vanishing value of R; such a simple property is not shared by Z except for highly symmetric cases.

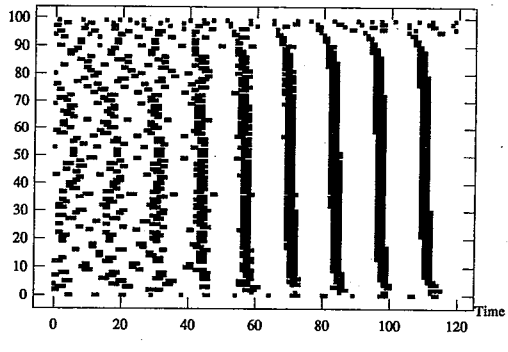

Fig. 2. Formation of a phase-locked domain exhibited by 100 globally coupled cells obeying Eqs. (2a, b) with distributed Ω. The cells are numbered vertically in order of increasing natural frequencies. Active phases are indicated by dark horizontal bars. Parameter values: $h=0.90$, $\Omega_0=0.50$, $\gamma=0.02$, $|C|=0.70$, $\alpha=0.50$.

Figure 3 shows how R changes with the synaptic strength $|C|$. As is expected, a critical value of $|C|$ for the onset of collective oscillations seems to exist.

There are some striking similarities of our numerical results to what we know about the populations of the phase oscillators studied earlier. We suspect that the present model itself might be reduced to the network of phase oscillators under suitable conditions. The reduction is in fact possible when the synaptic coupling is sufficiently weak, as we see below.

Since the deviation of $|z_i|$ from $r^*(=1)$ may still be neglected when the coupling introduced is weak, we retain Eq. (4a) as valid. The only remaining problem is therefore to find an approximate form of the coupling term to be added to the right-hand side of Eq. (4b). Comparing the phases of the respective sides of Eq. (2b) with each other, and putting $r_i=1$, we obtain to the lowest order in the coupling strength

$$\phi_i(t+1)-\phi_i(t)=\Omega+\sum_j|C_{ij}|\sin(\alpha_{ij}-\Omega-\phi_i(t))S_j(t)\,. \tag{6}$$

Substituting Eq. (4a) for S_j, and using new phase variables ψ_i defined by $\psi_i=\phi_i-\Omega t$, we rewrite Eq. (6) in the form

$$\psi_i(t+1)-\psi_i(t)=\sum_j|C_{ij}|\sin(\tilde{\alpha}_{ij}-\Omega t-\psi_i(t))F(\cos(\Omega t+\psi_j(t))-h)\,, \tag{7}$$

where $\tilde{\alpha}_{ij}=\alpha_{ij}-\Omega$. Since the time-variation of ψ_i should be sufficiently slow, the left-hand side of Eq. (7) may be approximated by $d\psi_i/dt$ and, moreover, the right-hand side may be time-averaged over the oscillation period $2\pi/\Omega$ under fixed ψ_j's over the same period. In this way, we get

$$\begin{aligned}\dot{\psi}_i&=\sum_j|C_{ij}|\frac{1}{2\pi}\int_0^{2\pi}d\lambda\,\sin(\psi_j-\psi_i+\tilde{\alpha}_{ij}-\lambda)F(\cos\lambda-h)\\&=\sum_j K_{ij}\sin(\psi_j-\psi_i+\tilde{\alpha}_{ij})\end{aligned} \tag{8}$$

or

$$\dot{\phi}_i=\Omega+\sum_j K_{ij}\sin(\phi_j-\phi_i+\tilde{\alpha}_{ij})\,, \tag{9}$$

where

$$K_{ij}=\frac{|C_{ij}|}{2\pi}\int_0^{2\pi}d\lambda\,\cos\lambda\,F(\cos\lambda-h)\,. \tag{10}$$

Equation (9) gives the standard form of the phase model. When the natural period of bursting changes from cell to cell as we assumed in our numerical simulations, one may simply replace Ω by Ω_i in Eq. (9). A little careful examination shows that such replacement is consistent with our perturbative reduction only when the width of the distribution of Ω_i is much smaller than the average frequency Ω_0. This condition is assumed to be satisfied below.

Remember that our numerical simulations assumed: (a) all-to-all coupling, (b) $\alpha=\Omega_0$ so that $\tilde{\alpha}_{ij}\simeq\alpha-\Omega_0=0$, (c)$F(x)=\theta(x)$ and (d) Lorentz distribution $L(\Omega-\Omega_0;\,\gamma)$ for Ω_i. Under these conditions, Eqs. (9) and (10) are reduced to

$$\dot{\phi}_i=\Omega_i+\frac{K}{N}\sum_j\sin(\phi_j-\phi_i) \tag{11}$$

and

$$K=\begin{cases}|C|\sqrt{1-h^2}/\pi\,, & (|h|<1)\\ 0\,, & (|h|\geq 1)\end{cases} \tag{12}$$

respectively. Previously, the phase model of the form of Eq. (11) with Ω_i obeying the Lorentz distribution $L(\Omega-\Omega_0;\ \gamma)$ was solved exactly for Z.[12] The result is $Z=\sqrt{1-(2\gamma/K)}\exp(i\Omega_0 t)$ for $K\geq 2\gamma$ and 0 otherwise. Thus R becomes

$$R=\begin{cases}\Omega_0\left(1-\dfrac{2\gamma}{K}\right), & (K\geq 2\gamma)\\ 0\,, & (K<2\gamma)\end{cases} \tag{13}$$

where K is given by Eq. (12). This approximate result for R is now compared with our numerical curve for the same quantity, which is shown in Fig. 3. Their agreement is satisfactory at least not far from the critical point.

Although still preliminary, further numerical study revealed the occurrence of more complicated collective dynamics when the coupling becomes stronger. Figure 4 shows an example of such behavior for a population *without* frequency distribution, where the system is decomposed into two subpopulations with different timing of bursts. The states of three, four and more clusters were also found under different conditions. This kind of clustering behavior was first discovered by Kaneko[13] for globally coupled chaotic units and then by Golomb et al.[14] for globally coupled non-smooth phase oscillators.

The effect of an external stimulus I is now considered, and we add this term to the right-hand side of Eq. (2b). For simplicity, I is assumed to be real positive, as weak as $|C|$, constant in time and cell-independent. We will show below that the collective dynamics is very sensitive to I when the threshold parameter h is near its

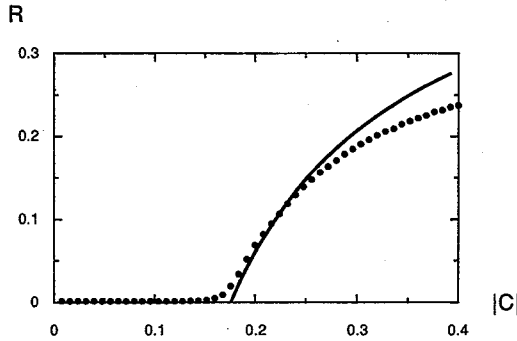

Fig. 3. Order parameter R versus coupling strength $|C|$. Circles indicate the data from computer simulation on Eq. (2a, b) with 100 globally coupled cells. The analytical result in Eq. (12) valid in the weak-coupling limit is indicated by a solid curve. Parameter values: $h=0.7$, $\Omega_0=0.5$, $\gamma=0.02$, $\alpha=0.5$.

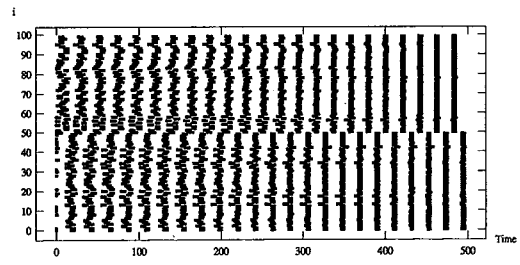

Fig. 4. Temporal pattern of bursts exhibited by a population of 100 globally coupled cells without frequency distribution (displayed in a way similar to Fig. 2 except that the cells are renumbered suitably). Parameter values: $h=0.51$, $\Omega=0.31$, $|C|=0.70$, $\alpha=2.20$.

critical value 1. In such a situation, we are no longer allowed to neglect the small amplitude-deviation of z_i from 1, but instead we have to come back to the original expression (2a) for $S_i(t)$ with

$$\mathrm{Re}z_i(t)=\cos(\phi_i(t)+\Omega)+\sum_j|C_{ij}|\cos\alpha_{ij}S_j(t)+I\,. \tag{14}$$

In the 0-th order approximation in which the last two terms were neglected, $S_j(t)$ is vanishing for the most period on account of the assumption $h\lesssim 1$. This implies that in Eq. (14) the coupling term is negligible compared to the input I. Thus, in the first order approximation, we may use in Eq. (6) the expression

$$S_j(t)=F(\cos(\phi_j(t))+I-h)\,. \tag{15}$$

On the other hand, the new term $-\sin(\phi_i(t)+\Omega)I$ which appears on the right-hand side of Eq. (6) vanishes on averaging. Consequently, the previous result (9) without external stimulus remains valid if we change the definition of K_{ij} as

$$K_{ij}=\frac{|C_{ij}|}{2\pi}\int_0^{2\pi}d\lambda\cos\lambda\,F(\cos\lambda+I-h)\,. \tag{16}$$

It is clear that the effective coupling K_{ij} is strengthened by I, so that if the system is originally near and below the threshold for collective oscillation, the external stimulus may easily bring it into the oscillatory regime. Stimulus-evoked collective oscillations of neural populations have been observed in the cat primary visual cortex.[1] This phenomenon is thus naturally understood if we suppose our idealized population with all-to-all excitatory coupling to represent one orientational column in the visual cortex. It is also suggested from the experiments that each neuron gives rise to at most a few spikes per oscillation period. This is consistent with our assumption $h\lesssim 1$, namely, the assumption of brief active period.

We conclude this paper with a few additional remarks. First, the reduced dynamics in Eq. (8) bears some resemblance to the relaxational XY spin dynamics. In fact, if $\tilde{\alpha}_{ij}=0$ and if at the same time the synaptic matrix is symmetric, then Eq. (8) derives from the hamiltonian $H=(1/2)\sum_i\sum_j K_{ij}\cos(\phi_i-\phi_j)$ via $\dot{\phi}_i=-\partial H/\partial\phi_i$. Generalization to finite temperature XY spins would also be easy to achieve by including random noise in Eq. (2b). Secondly, the form of the phase coupling in Eq. (9) implies that a given pair of cells favors in-phase/out-of-phase configurations if the corresponding $|\tilde{\alpha}_{ij}|$ is smaller/larger than $\pi/2$. On account of the difference between α_{ij} and $\tilde{\alpha}_{ij}$, the above condition for $|\tilde{\alpha}_{ij}|$ does not correspond precisely to the excitatory/inhibitory condition. This difference Ω_0 is unimportant, however, as far as the typical period $2\pi/\Omega_0$ of bursting remains, as usual, much longer than the minimum interval between successive spikes which is $O(1)$. Thirdly, our proposed mechanism leading to phase locking seems to be quite efficient energetically because it can work when the synaptic coupling is too weak to change the level of activity to a meaningful extent. In the framework of the conventional neural network model with global coupling, the only way to produce oscillations will be to assume a feedback loop between excitatory and inhibitory neuron populations.[15] This of course presupposes strong synaptic coupling. Finally, our extension of synaptic

efficacies to complex numbers suggests the possibility of a new learning scheme based on the *orientational* plasticity of C_{ij} in the complex plane. In this connection, very suggestive is the fact that $\tilde{a}_{ij}$ (i.e., essentially the phases of C_{ij}) are important parameters in determining phase wave properties associated with a space-continuous version of the phase equation (9).[16] Possible roles of the phases of C_{ij} in neural information processing are yet to be clarified.

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