

Directional operations in the motor cortex modeled by a neural network of spiking neurons

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Abstract. A neural network with realistically modeled, spiking neurons is proposed to model ensemble operations of directionally tuned neurons in the motor cortex. The model reproduces well directional operations previously identified experimentally, including the prediction of the direction of an upcoming movement in reaching tasks and the rotation of the neuronal population vector in a directional transformation task.

1 Introduction

A particular direction of movement involves the activation of a large ensemble of directionally tuned cells in the motor cortex (see Georgopoulos et al. 1993 for a review). An ensemble measure of cell activity, the neuronal population vector, has proved to be a useful, robust, and accurate measure of the directional tendency of the neuronal ensemble (Georgopoulos et al. 1983, 1986); for example, it predicts the direction of an upcoming movement during the reaction time (Georgopoulos et al. 1984, 1988), during an instructed delay period (Georgopoulos et al. 1989a), and during a memorized delay period (Smyrnis et al. 1992). The population vector is a weighted sum of vectorial contributions of individual cells in the ensemble: if C_i is the unit preferred direction vector for the i th neuron, then the neuronal population vector \mathbf{P} is:

$$\mathbf{P}(t) = \sum_i V_i(t) C_i \quad (1)$$

where the weight $V_i(t)$ is the activity (firing rate) of the i th neuron at time bin t . During the period of time at which the movement is being planned, as well as while the movement is executed, the neuronal population vector reveals nontrivial, definite, and well-reproduced dynamical behavior; namely, a relatively long-lasting (up to 500 ms) stability of its direction in reaching tasks

(Georgopoulos et al. 1988, 1989a); a rotation between two fixed directions with subsequent stabilization in the direction of the upcoming movement in a transformation task that requires a movement to be made at a given angle from a stimulus direction (Georgopoulos et al. 1989b; Lurito et al. 1991); and a continuous change in direction which predicts with a lead of approximately 150 ms the change of direction of the movement in drawing tasks (Schwartz 1993).

Two basic hypotheses could be proposed regarding these phenomena. The first is that the observed effects are straightforward consequences of processes taking place somewhere outside the motor cortex. Although other brain areas are active during reaching, including the premotor cortex (Caminiti et al. 1991), parietal cortex (Kalaska et al. 1983), and cerebellum (Fortier et al. 1989), there is no evidence that a higher-order coordinating center really exists. The second hypothesis is that, although converging extrinsic inputs can initiate the changes in activity in the motor cortex and contribute temporarily or constantly to its ongoing activity, the dynamics of the motor cortical network is essentially governed by the interactions among cells within the motor cortex rather than being imposed externally (Georgopoulos et al. 1993). In this paper we describe a biologically relevant neural network that models the motor cortical activity during the intended movement in the framework of the second hypothesis above. The key idea is that a correlation between synaptic connection strengths and preferred directions of the neurons involved in the connection could ensure the observed stability and transformations of the neuronal population vector.

Although simulations of the dynamical behavior of the neuronal population vector by modeling neurons as simple input-output devices have been carried out (Lukashin 1990; Eisenman et al. (1991); Georgopoulos et al. 1993; Lukashin and Georgopoulos 1993, 1994); it is still unclear where the border has between important features and incidental details in the modeling of motor cortical cells. Hence, more realistic models are desirable. As a first step in that direction, we simulate in the present study an artificial network that consists of 48 heavily

interconnected, directionally tuned neurons modeled at a reasonable level of complexity. Real motor cortical neurons produce trains of action potentials, or spikes, and it is the timing of these spikes that carries information about the directional tendency. In this study we sought to determine the extent to which an ensemble of spiking neurons can carry out the directional operations of the neuronal population vector.

2 Model

To describe a neuron and the interactions between neurons, we use a biophysical model of intermediate complexity. A neuron is represented as a one-compartment cell equipped with a standard set of active channels with kinetics governed by the Hodgkin–Huxley type of equations (Hodgkin and Huxley 1952; Koch and Segev 1989). Each cell has voltage-dependent sodium and potassium channels and a calcium-dependent potassium channel that generates the late-phase afterhyperpolarization. The intracellular level of free calcium is the sum of two calcium currents: the first corresponds to the influx through a voltage-gated calcium channel, and the second to calcium that enters the cell through the NMDA channel. The depolarizing current resulting from the influx of calcium ions has been ignored. Specifically, the system of equations listed in Ekeberg et al. (1991) was used to calculate the ionic currents, calcium level, and corresponding voltage- and time-dependent degrees of activation/inactivation of the channels. The relevant parameters are presented in Table 1. [The exponential

expressions for the rate constants of activation/inactivation were described using 28 parameters exactly as listed in Table 1 of Ekeberg et al. (1991); these parameters are not reproduced here.]

All neurons are connected with each other, and changes in synaptic conductance are used to model conventional excitatory and inhibitory synaptic interactions. The contribution of the synaptic currents into the total current entering the cell is calculated by a linear summation over all synapses. The ionic current resulting from activation of a synapse due to the arrival of a presynaptic action potential is calculated as the product of the time-dependent synaptic conductance times the difference between the reversal potential of the synapse and the membrane potential of the cell. The synaptic conductance decreases exponentially with a decay time constant. If another presynaptic spike arrives while a residual synaptic current still remains, the conductance is not allowed to increase above the level assigned for a single spike (Ekeberg et al. 1991). The model is capable of producing action potentials with a realistic shape and in a realistic (for motor cortical cells) firing frequency range between 10 and 70 impulses per second.

To make the model specific to a description of the motor cortical network, two additional factors are introduced. First, each i th neuron receives an extra amount of intracellular current. This extra current, E_i , serves to assign the preferred direction of the neuron. We chose an expression for the current E_i such that it reproduced quantitatively the directional tuning curve, that is, the changes in cell activity related to changes of the externally given direction of the movement. The experimentally observed tuning curve can be approximated as a linear function of the cosine of the angle between the preferred direction of a cell and the movement direction (Georgopoulos et al. 1982; Schwartz et al. 1988). Within the model, this condition leads to the following relation:

$$E_i = b + a \cos(\theta - \alpha_i) \quad (2)$$

where the angle θ corresponds to the externally given direction, the angle α_i is the preferred direction angle of the i th neuron, and b and a are adjustable parameters. The observed tuning curves are reproduced if the following values of the parameters are used: $b = 3.0$ nA, $a = 2.2$ nA. Second, the value of synaptic conductance corresponding to the connection between the j th (presynaptic) and i th (postsynaptic) neurons shown in Table 1 is multiplied by an additional parameter, the connection strength w_{ij} , and the type of synapse (excitatory or inhibitory) is determined by the sign of the w_{ij} value (positive or negative, respectively). A specific form of the correlation between the connection strengths w_{ij} and the preferred direction angles α_i is chosen in accordance with the experimental findings (Georgopoulos et al. 1993). It was shown (Georgopoulos et al. 1993) that the weights of functional connections between directionally tuned neurons tend to be negatively correlated with the difference between the preferred directions. A first obvious approach to model this correlation is to

Table 1. Parameters used for the simulations

Parameter	Value
<i>Cell</i>	
Leak current equilibrium potential	−70 mV
Sodium equilibrium potential	50 mV
Potassium equilibrium potential	−90 mV
Calcium equilibrium potential (voltage-gated)	150 mV
Calcium equilibrium potential (NMDA-gated)	20 mV
Membrane capacitance	0.05 nF
Membrane leak conductance	0.01 μ S
Sodium conductance	1.00 μ S
Potassium conductance	0.50 μ S
Calcium-dependent potassium conductance	0.02 μ S
Calcium accumulation rate (voltage-gated)	0.0040 $\text{mV}^{-1}\text{ms}^{-1}$
Calcium accumulation rate (NMDA-gated)	0.0005 $\text{mV}^{-1}\text{ms}^{-1}$
Calcium decay rate (voltage-gated)	0.0300 ms^{-1}
Calcium decay rate (NMDA-gated)	0.0030 ms^{-1}
<i>Excitatory synapses</i>	
Reversal potential	0 mV
Conductance after spike	0.003 $\cdot w^a \mu$ S
Decay time constant	100 ms
<i>Inhibitory synapses</i>	
Reversal potential	−85 mV
Conductance after spike	0.015 $\cdot w^a \mu$ S
Decay time constant	20 ms

^a Here, w is the strength of synaptic connection (see text)

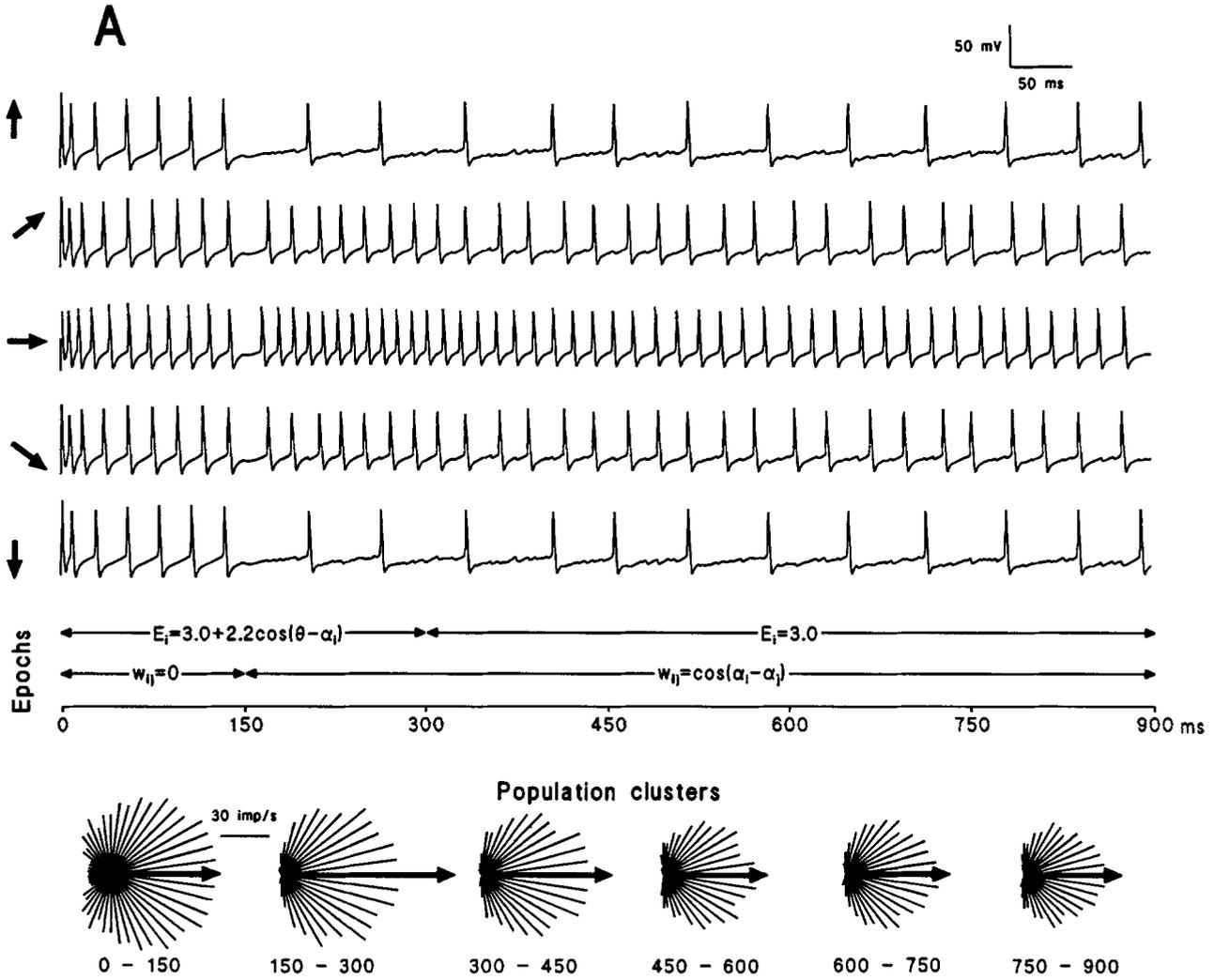


Fig. 1A. (Continued on p. 82)

represent an unknown function $w_{ij}(\alpha_i - \alpha_j)$ as the sum of two harmonics:

$$w_{ij} = d \cos(\alpha_i - \alpha_j) + c \sin(\alpha_i - \alpha_j) \quad (3)$$

where d and c are adjustable parameters that determine the relative contribution of symmetric and antisymmetric components, and $d > 0$. The correlation between the weight of the synaptic connection w_{ij} and the angles α_i , α_j is the main feature of the suggested model.

3 Simulations and discussion

The results of simulations in the framework of the above model are shown in Fig. 1. The dynamics of the network is governed by the system of 384 coupled differential equations (8 equations per each neuron). Expressions for the extra currents E_i and for the synaptic connection strengths w_{ij} are described by (2) and (3). The preferred direction angles α_i are uniformly distributed on the inter-

val $[-\pi, \pi]$. For a given set of parameters, the system of differential equations is solved using a fourth-order Runge-Kutta formula with a time step equal to 0.005 ms. To calculate the neuronal population vector (1), we estimate the instantaneous frequency of cell discharge as the number of spikes during each 150-ms time bin.

The spike trains shown in Fig. 1A at the time interval between 0 and 150 ms reflect the activity of the network without synaptic connections, that is, $w_{ij} = 0$. A directional tendency of the network emerges due to the fact that neurons become directionally diverse by the action of the externally imposed currents E_i . The direction of the population vector coincides with the externally given direction $\theta = 0$ deg. At the instant of time 150 ms, the symmetric component of the synaptic connections is switched on, that is, $w_{ij} = \cos(\alpha_i - \alpha_j)$ [see (3)]. After this moment, the directional tendency becomes even stronger because of an inhibition of the firing of neurons whose preferred directions are far from the externally given direction of 0 deg. The most interesting dynamical

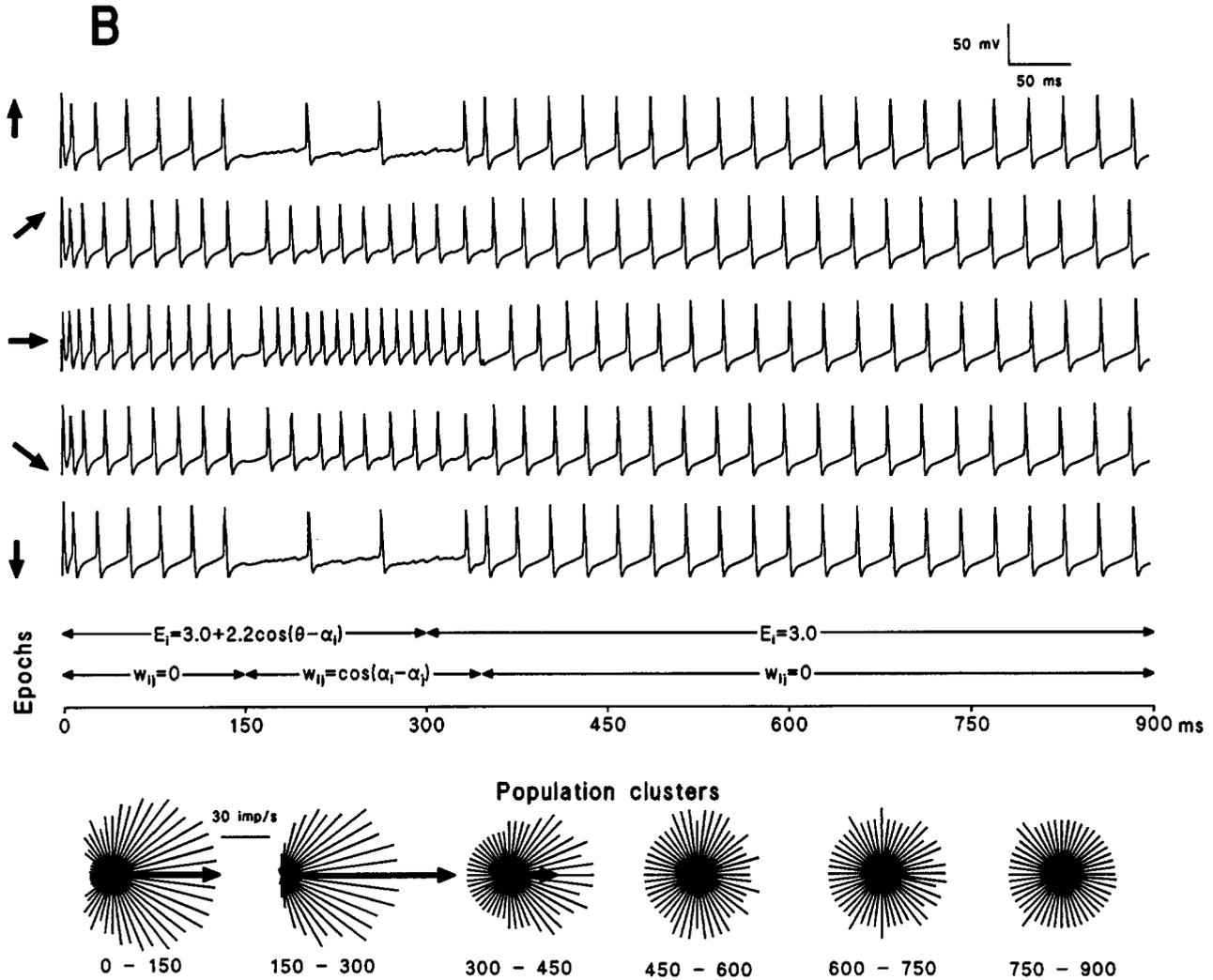


Fig. 1B. (Continued on p. 83)

period begins at 300 ms, when the part of the external currents that produced the directional diversification of the neurons [second term on the right-hand side of (2)] is switched off. Starting from this moment, there is no directional influence coming from outside the network, since all neurons receive the equal extra current $E_i = 3.0$ nA. In spite of this, the directional tendency of the network (the neuronal population vector) is still present (see the spike trains and population clusters at the interval 300–900 ms) due to the correlated interactions between cells. The data presented in Fig. 1B demonstrate that this stability of the directional tendency is not due to a residual effect of the previous external directional influence. Indeed, all parameters in this trial are the same as in Fig. 1A up to the instant of time 350 ms, when all the synaptic connection strengths were set to zero. It can be seen that the directional tendency disappears within an interval of less than 100 ms, and the population clusters become symmetric. These simulations show that the experimentally observed stability of the directional

tendency of the neuronal population vector lasting several hundred milliseconds (Georgopoulos et al. 1986, 1989a, 1993) is most probably due to correlated synaptic connections between cells even without any directional influence from areas outside the motor cortex.

Next, the ability of the network to reproduce a basic type of transformation of the neuronal population vector, namely its rotation, was investigated. This is demonstrated by Fig. 1C, D. During these trials, the external currents E_i as well as the symmetric components of the connection strengths w_{ij} were not changed, and the externally given direction was the same as in previous trials ($\theta = 0$ deg). In the trial shown in Fig. 1C, during the first 400 ms, the sign of the antisymmetric component of the connection strengths [that is, $\sin(\alpha_i - \alpha_j)$; see (3)] was negative. This resulted in a stable population vector pointing at -45 deg (see clusters for the intervals 0–150 and 150–300 ms). Then, at the point in time 400 ms, the sign of antisymmetric component of the connection strengths was changed from negative to positive. This

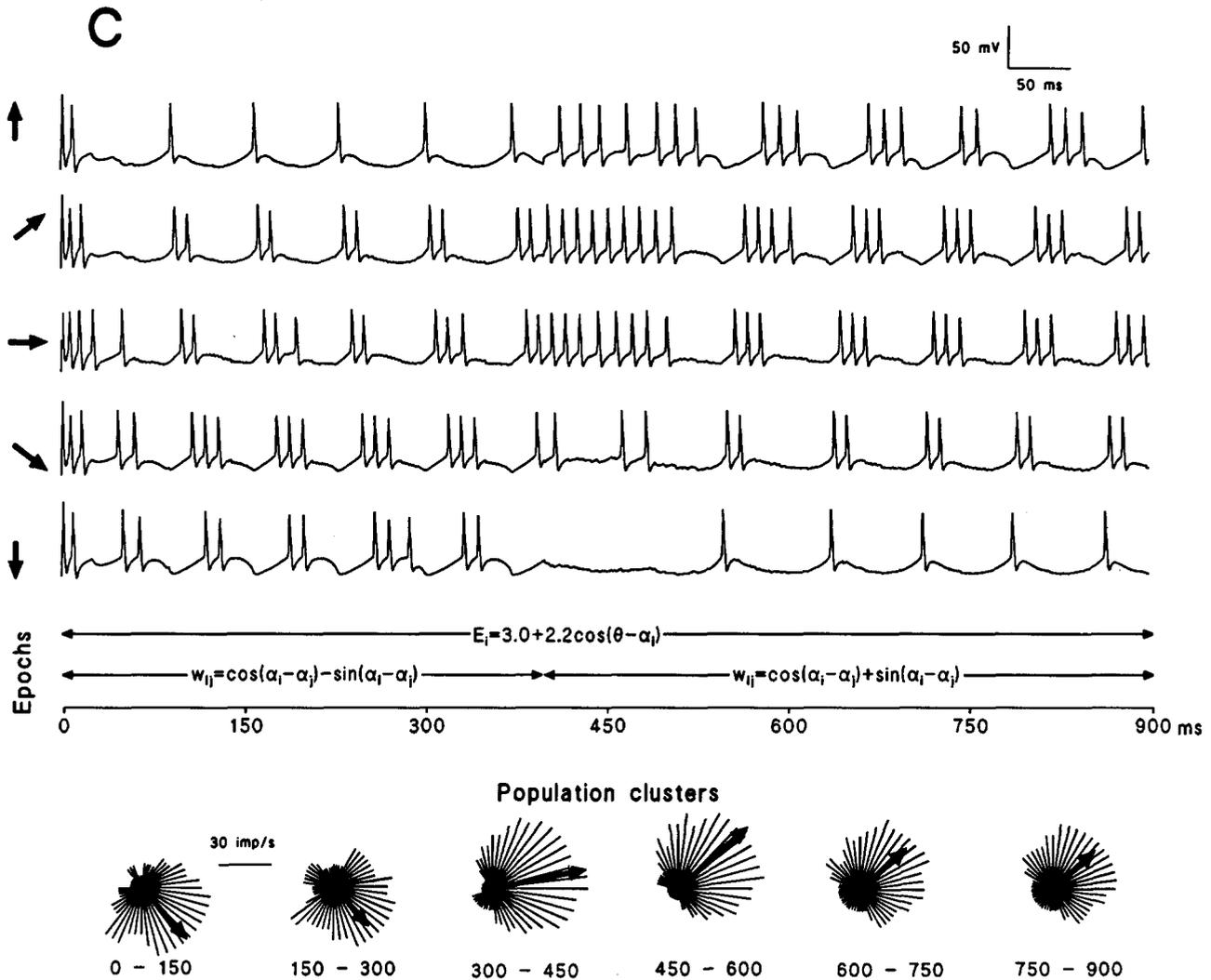


Fig. 1C. (Continued on p. 84)

resulted in a 90 deg counterclockwise rotation of the neuronal population vector, from -45 deg to $+45$ deg (see clusters at intervals 300–450 and 450–600 ms). The population vector stabilized and pointed in this last direction for the remaining period of 500 ms (see clusters at intervals 600–750 and 750–900 ms), during which the sign of the antisymmetric component of the connection strengths was kept positive (see the epochs' description rows in Fig. 1). In Fig. 1D, the clockwise rotation seen is due to the opposite assignment of the signs.

The particular value of the angle that characterizes the equilibrium direction of the neuronal population vector depends on the relation between the weights of the symmetric and antisymmetric components of the connection strengths [parameters d and c in (3), respectively]. The specific choice of the expression for the w_{ij} values (3) makes it possible to vary the angle between $\theta - 90$ and $\theta + 90$ deg. The velocity of the rotation can be easily evaluated from the data presented by the time needed for the network to change the intensity and frequency of the

bursts (see spike trains in Fig. 1C, D at the interval between 300 and 600 ms). The rotation at 90 deg takes approximately 100–150 ms (between 400 and 550 ms), which gives a velocity value of about 600–900 deg/s. This value is close to the experimentally measured angular velocity of the rotation of the neuronal population vector, which is approximately 400–700 deg/s (Georgopoulos et al. 1989b; Lurito et al. 1991).

4 Conclusion

These results demonstrate that the network of spiking neurons used can reproduce the main features of experimental findings concerning the directional operations of the neuronal population vector, including its rotation observed in a directional transformation task. The following remarks are noteworthy. Firstly, the present network, although closer to reality than networks of nonspiking neurons, is still far removed from the actual

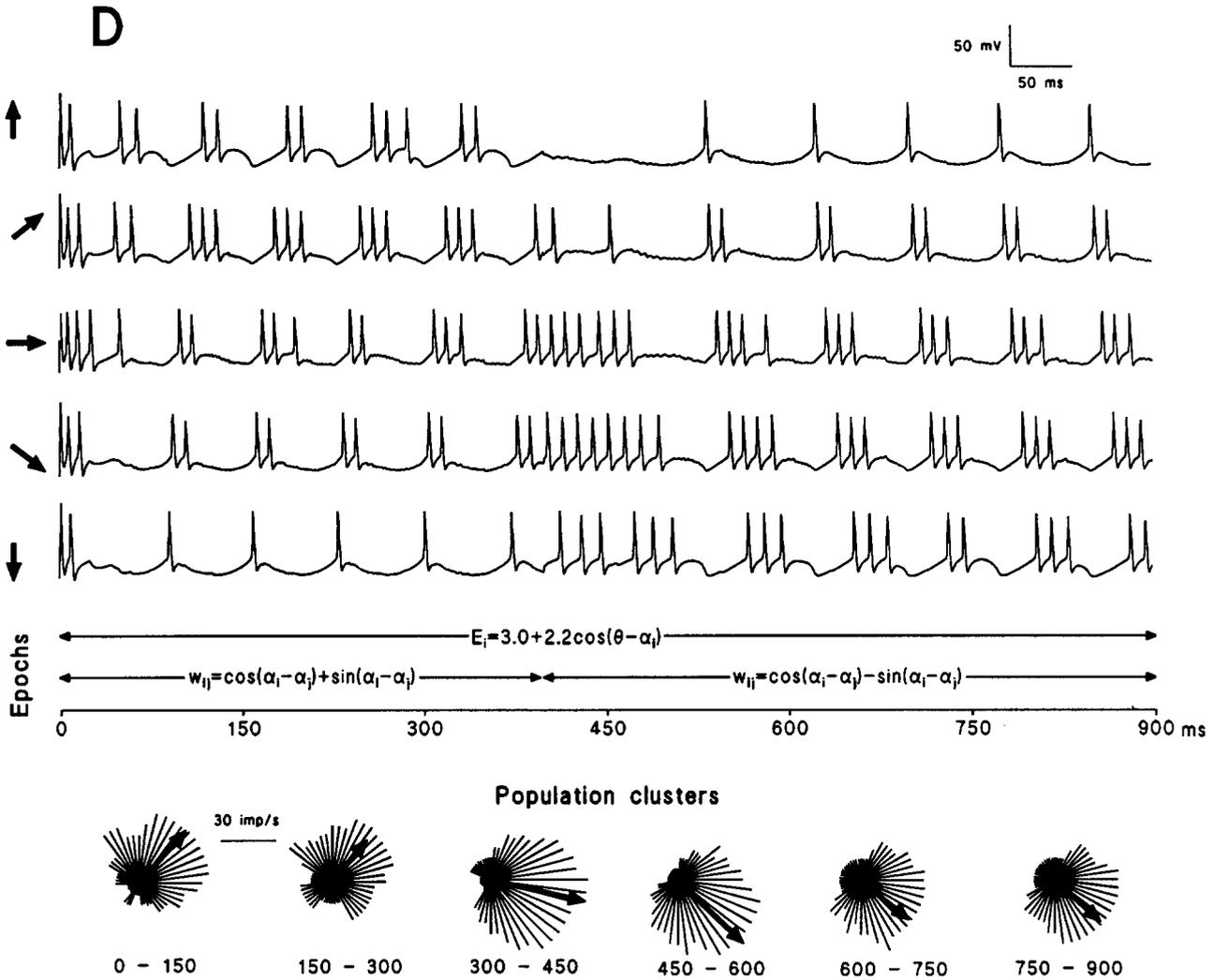


Fig. 1A–D. Simulations activity of the network that consists of 48 heavily interconnected, directionally tuned neurons. *Five rows at the top* represent the time dependence of the membrane potentials (the spike trains) for five selected neurons whose preferred directions are shown by *arrows* to the left of each row (90, 45, 0, –45, –90 deg in order from top to bottom). The *sixth row* specifies expressions that are used for the extra currents [see (2); angle θ is set to zero] and for the connection strengths [see (3)] at different time intervals of the simulations (values of the currents are given in nA units; the connection strengths are

dimensionless). The *lowest row* shows the activities of all 48 neurons arranged in the population clusters. The *direction of each thin line* corresponds to the preferred direction of the neuron. The *length of each thin line* represents the firing frequency (in units impulses/s) produced by the neuron at the time interval shown under each cluster. The *heavy arrows* are the neuronal population vectors calculated for the corresponding clusters in accordance with the definition (1). Lengths of the population vectors are reduced tenfold

configuration of the motor cortex in its morphological, physiological, and neurochemical details. Secondly, the results are not specific to the set of parameters chosen for modeling single neurons as evidenced by the fact that the results were robust with respect to changes of parameters within a reasonable range of 15–20% of their original values. Moreover, we obtained qualitatively similar results using a model with simplified channel kinetics close to the one suggested by Bush and Sejnowski (1991). Although NMDA channels were included in our model, they were not manipulated in our simulations. Thirdly, concerning the assignment of the preferred directions of the neurons, we ensured that injecting external current is not the only way to achieve this assignment. Indeed, we obtained the same results when we modeled external directional influences

by adding extra neurons with correspondingly assigned strengths of the synaptic connections. Fourthly, in this study only one type of correlation between the weight of the synaptic connection and the difference between the preferred directions of neurons in a pair involved in the connection (3) has been tested. Previous results of a study of the stability problem performed in Georgopoulos et al. (1993) and Lukashin and Georgopoulos (1994) within a more simple model showed that other types of this correlation are possible. Finally, the global change of the synaptic connection strengths that induced the rotation of the population vector might have its biological analogue in the so-called saliency systems which can affect many synapses simultaneously by the release of neuromodulators (Harris-Warrick and Marder 1991; Tononi et al. 1992).

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