Vol 15 No 10, October 2006
1009-1963/2006/15(10)/2450-08

Chinese Physics

© 2006 Chin. Phys. Soc.
and IOP Publishing Ltd

The spike timing precision of FitzHugh–Nagumo neuron network coupled by gap junctions*

Zhang Su-Hua(张素花)†, Zhan Yong(展　永), Yu Hui(于　慧),
An Hai-Long(安海龙), and Zhao Tong-Jun(赵同军)

School of Sciences, Hebei University of Technology, Tianjin 300130, *China*

(Received 20 December 2005; revised manuscript received 7 June 2006)

It has been proved recently that the spike timing can play an important role in information transmission, so in this paper we develop a network with N-unit FitzHugh–Nagumo neurons coupled by gap junctions and discuss the dependence of the spike timing precision on synaptic coupling strength, the noise intensity and the size of the neuron ensemble. The calculated results show that the spike timing precision decreases as the noise intensity increases; and the ensemble spike timing precision increases with coupling strength increasing. The electric synapse coupling has a more important effect on the spike timing precision than the chemical synapse coupling.

Keywords: spike timing precision, gap junction, FitzHugh–Nagumo model

PACC: 8730C, 8730E

1. Introduction

A fundamental problem in computational neuroscience concerns how information is encoded by the neural architecture of the brain.[1−7] Many debates are focused mainly on two issues. One is whether information is encoded in an average firing rate of neurons (rate code) or in the precise firing time (temporal code). Indeed, in human visual systems, the transmission time between two successive stages of synaptic transmission is taken to be no more than 10 ms on the average.[8] This period is too short to be able to determine the rate accurately. The other issue is whether the information is encoded in the activity of a single (or very few) neuron(s) or a large number of neurons (population or ensemble code). An important part of the answer to the question is that individual element of information is encoded not by single cell, but rather by populations or clusters of cells. This encoding strategy is known as population code, and turns out to be common throughout the nervous system.[9] In the population temporal-code, the specific time of occurrence of spikes may play an important role. In this paper, we consider the population temporal-code model.

It is well known that neurons in brains are subject to various kinds of noises, such as the thermal noise, the ion channels: open and closed, the signal transmission and network effects, etc. These noises can alter the response of neurons in various ways. Although firings of a single neocortical neuron in *vitro* are precise and reliable, those in vivo are quite unreliable.[10] Neural responses are variable: repeating a stimulus yields spike trains that vary in total spikes and spike timing. Therefore, to assess the limits of information transmission from a given functional organization through cortex, neural signals must be analysed with a measure that quantifies spike timing precision. Many researches have been conducted on the properties of spike timing precision. These studies have demonstrated that a suitable noise can enhance the spike timing precision,[11−15] the spike timing precision depends on the stimulus[16] and it underlies the coding efficiency of auditory receptor neurons[17,18] and error correction,[19] etc.

A small patch of cortex may contain hundreds and thousands of neurons, each connecting with thousands of the similar neurons. How to simulate so large a real system is a difficult problem. Computational neuroscientists have so far tried to gain an understanding of the properties of neuron network with two methods: 1) direct numerical simulations, 2) an-

*Project supported by the National Natural Science Foundation of China (Grant Nos 10375016 and 10474018) and the Natural Science Foundation of Hebei Province (Grant Nos C2005000011and A2004000005) and the Key Subject Construction Project of Hebei Provincial University.

†E-mail: zhangsuhua76@163.com

alytical approaches such as mean-field (MF) theories and the Fokker–Planck equation (FPE). For the first method, it is difficult to simulate a realistic neuron network due to the limited computation ability. For the second method, the Fokker–Planck equations are partial differential equations that are difficult to solve. And the MF theories and the FPE adopt the rate-code strategy. In 2003, Hasegawa[20] proposed a dynamical mean-field approximation (DMA) theory to simulate the activities of neuron network. The advantages of this method are that it can reduce the stochastic differential equations to deterministic differential equations, cut down the number of differential equations largely and give qualitative or semiquantitative discussion on the properties of neuron ensembles or clusters. And Hasegawa has proven that the larger the size of network is, the more accurate the computational results will be. Furthermore, the DMA theory based on moments of local and global variables can be applied to more general stochastic systems besides neural networks.[21] But the disadvantage is that it is hard to obtain explicit expressions for the spectrum of the population rate. Furthermore, to our knowledge, no explicit results have been derived for single-neuron measures like the power spectrum (or autocorrelation function) of a single neuron or the cross spectrum (or crosscorrelation function) of two distinct neurons within the network.

The rest of this paper is organized as follows. In Section 2, we develop an N-unit FitzHugh–Nagumo (FN) neuron network and reduce the original $2N$-dimensional stochastic differential equations to eight-dimensional deterministic differential equations by Hasegawa's DMA theory. In Section 3, the spike timing precision is analysed. In Section 4, the effects of noise intensity, coupling strength and size of ensemble on the spike timing precision are investigated. In Section 5, the different effects on the spike timing precision are studied for the electric synapse and the chemical synapse. In Section 6 we give conclusions drawn from the present study.

2. The FitzHugh–Nagumo neuron network

The underlying dynamics of individual neuron are described by many nonlinear equations. The well-known four-dimensional Hodgkin–Huxley differential equations contain various variables of voltage-dependent ionic channels. Although the model parameters have clear physical significations, the complexity of these equations prevents us from understanding the dynamical properties of neuron. Some simple models are attained, such as the three-dimensional Hindmarsh–Rose model, the two-dimensional FN model and the one-dimensional integrate-and-fire model, etc. The FN model can be analysed easily in phase plane, and it shares with the Hodgkin–Huxley model the properties of subthreshold responses and suprathreshold responses, including periodic solutions, representing action potentials or spikes in response to suitable stimuli.

We assume a neuron ensemble to consist of N-unit FN neurons. Dynamics of a single FN neuron i is described by the following nonlinear differential equations:

$$\frac{\mathrm{d}x_i(t)}{\mathrm{d}t} = F[x_i(t)] - cy_i(t) + \frac{w}{N}\sum_{j\neq i} G(x_j(t)) + I^{(e)}(t) + \xi_i(t), \tag{1}$$

$$\frac{\mathrm{d}y_i(t)}{\mathrm{d}t} = bx_i(t) - \mathrm{d}y_i(t) + e, \tag{2}$$

where $F[x(t)] = kx(t)[x(t)-a][1-x(t)]$, $k = 0.5$, $a = 0.1$, $b = 0.015$, $c = 1.0$, $d = 0.003$ and $e = 0$.[22,23] x_i and y_i are the voltage variable and the slow (recovery) variable respectively. $G(x_j(t)) = x_j(t) - x_i(t)$ is the coupling term and denotes the gap coupling (the electric synapse coupling) and w is the coupling strength, and the self-coupling terms are excluded from the equations. N is the normalization factor. The fourth term on the right-hand side in Eq.(1) is the external current

$$I^{(e)}(t) = A\Theta(t - t_{\mathrm{in}})\Theta(t_{\mathrm{in}} + T_{\mathrm{w}} - t) \tag{3}$$

with $\Theta(x) = \begin{cases} 1, & x > 0 \\ 0, & x \leq 0 \end{cases}$, A is the current magnitude, $t_{\rm in}$ is the input time and $T_{\rm w}$ the spike duration. The fifth term on the right-hand side in Eq.(1) is the Gaussian white noise

$$\langle \xi_i(t) \rangle = 0, \langle \xi_i(t) \xi_j(t') \rangle = \beta^2 \delta_{ij} \delta(t - t'), \quad (4)$$

where $\langle \cdots \rangle$ indicates the average over stochastic random variables and β is the noise intensity.

We determine the global variables for the ensemble and their averages by

$$X(t) = \frac{1}{N} \sum_i x_i(t), \quad (5)$$

$$Y(t) = \frac{1}{N} \sum_i y_i(t), \quad (6)$$

$$\mu_1(t) = \langle X(t) \rangle, \quad (7)$$

$$\mu_2(t) = \langle Y(t) \rangle. \quad (8)$$

The variances and covariances between local variables are determined by

$$\gamma_{11} = \frac{1}{N} \sum_i \langle \delta x_i^2 \rangle, \quad \gamma_{22} = \frac{1}{N} \sum_i \langle \delta y_i^2 \rangle, \quad \text{and} \quad \gamma_{12} = \frac{1}{N} \langle \delta x_i \delta y_i \rangle. \quad (9)$$

These variables express the spatial averages of fluctuations in local variables of x_i and y_i.

The variances and covariances between global variables are given by

$$\rho_{11} = \langle \delta X^2 \rangle, \ \rho_{22} = \langle \delta Y^2 \rangle, \ \text{and} \ \rho_{12} = \langle \delta X \delta Y \rangle. \quad (10)$$

These variables express the spatial averages of fluctuations in global variables of X and Y. The following notations are used in Eq.(9) and Eq.(10) respectively:

$$\delta x_i = x(t) - \mu_1(t), \quad \delta y_i = y_i(t) - \mu_2(t); \quad (11)$$

$$\delta X = X(t) - \mu_1(t), \quad \delta Y = Y(t) - \mu_2(t). \quad (12)$$

Numerical calculations have demonstrated that the membrane voltage distribution of a single FN neuron obeys the Gaussian distribution for weak noise intensities.[24,25] In this paper, adopting the dynamical mean-field approximation theory,[20] the averages of fluctuations are expressed in terms of the first and second moments only, and Eqs.(1) and (2) reduce to eight-dimensional deterministic differential equations (the detailed calculations can be found in Appendix A).

$$\frac{\mathrm{d}\mu_1}{\mathrm{d}t} = f_0 + f_2 \gamma_{11} - c\mu_2 + I_i^{(e)}, \quad (13)$$

$$\frac{\mathrm{d}\mu_2}{\mathrm{d}t} = \mathrm{b}\mu_1 - \mathrm{d}\mu_2 + e, \quad (14)$$

$$\frac{\mathrm{d}\gamma_{11}}{\mathrm{d}t} = 2f_1\gamma_{11} + 6f_3\gamma_{11}\gamma_{11} - 2c\gamma_{12} + \beta^2 + 2w(\rho_{11} - \gamma_{11}), \quad (15)$$

$$\frac{\mathrm{d}\gamma_{22}}{\mathrm{d}t} = 2b\gamma_{12} - 2d\gamma_{22}, \quad (16)$$

$$\frac{\mathrm{d}\gamma_{12}}{\mathrm{d}t} = f_1\gamma_{12} + 3f_3\gamma_{11}\gamma_{12} - c\gamma_{22} + b\gamma_{11} - d\gamma_{12} + w(\rho_{12} - \gamma_{12}), \quad (17)$$

$$\frac{\mathrm{d}\rho_{11}}{\mathrm{d}t} = 2f_1\rho_{11} + 6f_3\rho_{11}\gamma_{11} - 2c\rho_{12} + \frac{\beta^2}{N}, \quad (18)$$

$$\frac{\mathrm{d}\rho_{11}}{\mathrm{d}t} = 2b\rho_{12} - 2d\rho_{22}, \quad (19)$$

$$\frac{\mathrm{d}\rho_{12}}{\mathrm{d}t} = f_1\rho_{12} + 3f_3\gamma_{11}\rho_{12} - c\rho_{22} + b\rho_{11} - d\rho_{12}, \quad (20)$$

With $f_l = \left(\frac{1}{l!}\right) F^{(l)}(\mu_1)$. Equation.(13) – (20) describe the properties of the neuron ensemble. It is very easy to see that the dynamical properties are determined by the noise intensity β, the coupling strength w and the size of ensemble N.

3. The spike timing precision

It is very easy to obtain the following expression from Eqs.(15) and (18):

$$\rho_{11} = \begin{cases} \frac{\gamma_{11}}{N}, & \text{for} \quad \frac{w}{\beta^2} \to 0, \\ \gamma_{11}, & \text{for} \quad \frac{\beta^2}{w} \to 0. \end{cases} \tag{21}$$

Equation (21) agrees with the central-limit theorem, and $\frac{\rho_{11}}{\gamma_{11}}$ varies with the parameters of the ensemble. We will show that the variation has an important influence on the firing-time distribution.

When the noise intensity is very small, the membrane voltage of a single neuron obeys the Gaussian distribution, so we obtain a firing-time distribution of a given neuron i (the detailed calculations are seen in Appendix B)

$$Z_{\rm l}(t) \approx \phi\left(\frac{t - t_0^*}{\delta t_{\rm ol}}\right) \frac{\mathrm{d}}{\mathrm{d}t} \frac{\mu_1}{\rho_1} \Theta(\dot{\mu_1}), \tag{22}$$

where $\phi(x) = \frac{1}{\sqrt{2\pi}} \exp\left(-\frac{x^2}{2}\right)$ is the normal distribution function, $\mu_1(t_0^*) = \theta$ with θ being the threshold,

$$\rho_{\rm l} = \sqrt{\gamma_{11}}, \tag{23}$$

$$\delta t_{\rm ol} = \frac{\rho_{\rm l}}{\dot{\mu_1}}. \tag{24}$$

Here μ, $\dot{\mu}$ and $\rho_{\rm l}$ are evaluated at $t = t_0^*$. $Z_{\rm l}(t)$ shows that most of the firing times of neurons are located in the range given by

$$t_{\rm ol} \in [t_0^* - \delta t_{\rm ol}, \quad t_0^* + \delta t_{\rm ol}]. \tag{25}$$

The smaller $\delta t_{\rm ol}$ is, the more precise the spike timing will be.

From the same method, we can obtain that most of the firing times of neuron ensembles are located in the range given by

$$t_{\rm og} \in [t_0^* - \delta t_{\rm og}, \quad t_0^* + \delta t_{\rm og}], \tag{26}$$

where

$$\delta t_{\rm og} = \frac{\sqrt{\rho_{11}}}{\dot{\mu_1}}. \tag{27}$$

The smaller $\delta t_{\rm og}$ is, the more precise the spike timing is.

From Eq.(21), we can see that ρ_{11} is generally smaller than γ_{11}, so $\delta t_{\rm og}$ is smaller than $\delta T_{\rm ol}$. When the ensemble has no coupling and

$$\delta t_{\rm og} = \frac{\delta t_{\rm ol}}{N}, \tag{28}$$

it can be seen that $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ reflect the spike timing precision.

4. Calculated results

From Eqs.(13) – (28), we can calculate the spike timing precision. In this study, we pay our attention to the response of the FN neuron network to a single spike input. A critical magnitude of $A_{\rm c} = 0.0442$, below which firings of neuron determined by Eq.(B1) cannot take place without noises($\beta = 0$), has been found in Ref.[20]. In this paper, we consider the suprathreshold response for $A = 0.1$. We have adopted the parameters of $\theta = 0.5$, $t_{\rm in} = 100$, and $T_{\rm w} = 10$. The fourth-order Runge–Kutta method is adopted to solve Eqs. (13)–(20) with a time step of 0.01, and the initial values of all variables are 0 and all calculated quantities are dimensionless.

Figure 1 indicates that each neuron in the ensemble is independent and stimulated by independent white noises. So when $t < 100$ in Fig.1(a), the average voltage of the neuron ensemble is equal to zero. When an external current is applied to the neurons at $t = 100$, all the neurons emit a spike at a time and the average voltage of the neuron ensemble has a spike. After a refractory period, the global voltage returns to zero again. From Fig.1(b) and Fig.1(c), γ_{11} and ρ_{11} each have two peaks. We may imagine that all the neurons are in the same state without noise, γ_{11} and ρ_{11} and will be equal to zero and have no peaks. So the two peaks of γ_{11} and ρ_{11} are caused by noises.

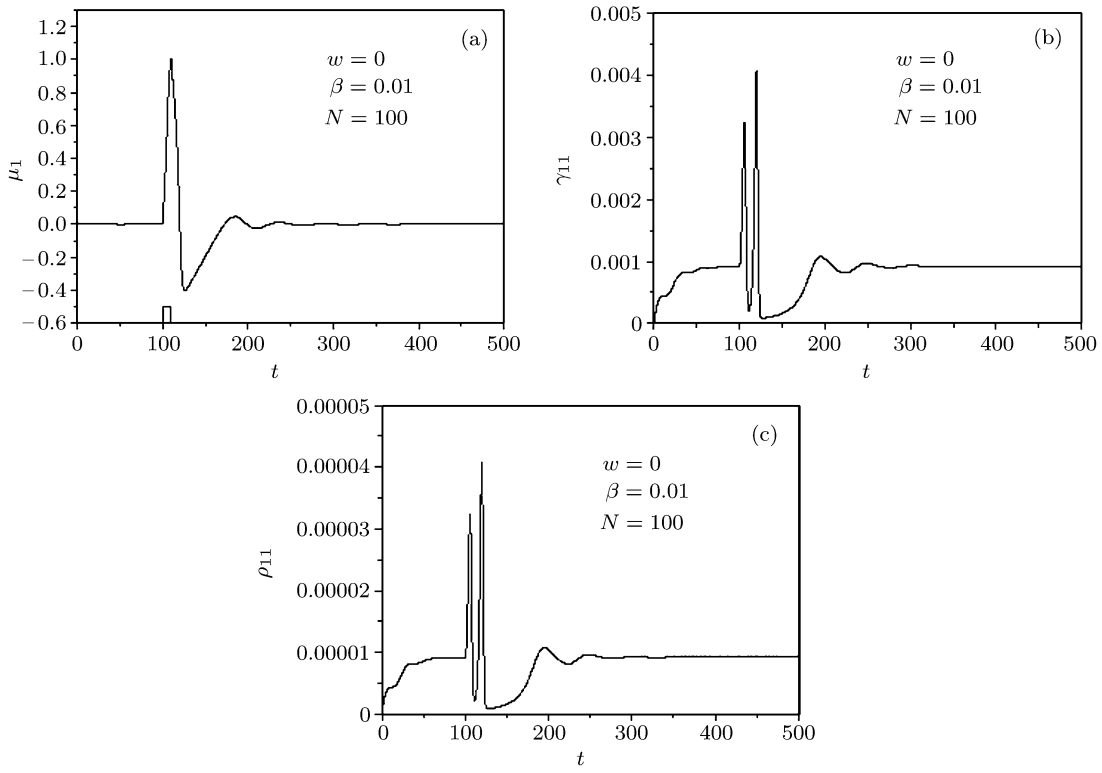


Fig.1. Time courses of mean, variance.

We study the dependence of spike timing precision on coupling strength, noise intensity and size of ensemble numerically.

Noises enhance the neuron random dynamics and the local fluctuations. And it can be expected that as the noise intensity increases, the distribution of membrane potentials becomes wider, fluctuations of firing times increase more but the spike timing precision decreases. From Fig.2(a), we can see when the network has no coupling, the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ satisfy Eq.(28). From Fig.2, the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ increase with the noise intensity increasing. This indicates that noises degrade the spike timing precision. But as coupling strength w increases, the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ decrease. It implies that the coupling suppresses local fluctuations and increases the spike timing precision as expected. The effect of stimulus amplitude on timing precision has also been observed in both recent[26] and classical experiments.[27]

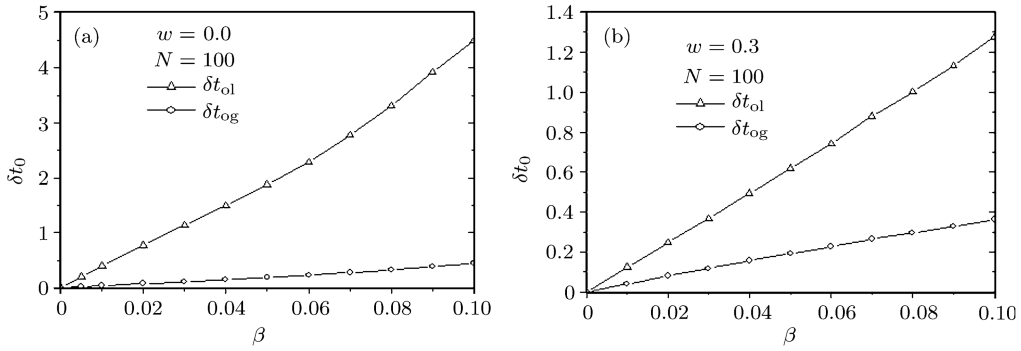


Fig.2. The dependence of spike timing precision on noise intensity.

When no coupling is in the ensemble, each neuron in the ensemble is independent and $\delta t_{\rm ol}$ does not change with the size of ensemble. Equation (28) indicates that $\delta t_{\rm og}$ decreases with the size of the ensemble increasing. Figure 3(b) shows that coupling makes each neuron dynamics in the ensemble change synchronously and suppresses local activities of the ensemble, which reduces the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$. If the size of ensemble is larger and the common and nonrandom currents are applied to the neurons in the

ensemble, the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ decrease. So a large size of the ensemble can increase the spike timing precision.

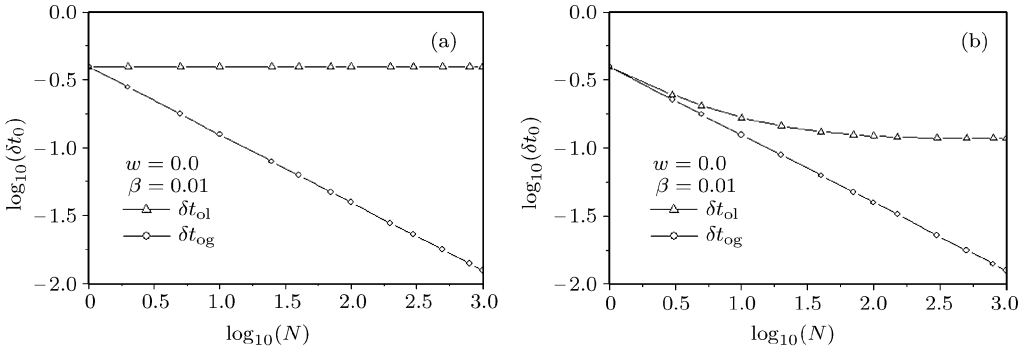


Fig.3. The dependence of spike timing precision on size of ensemble.

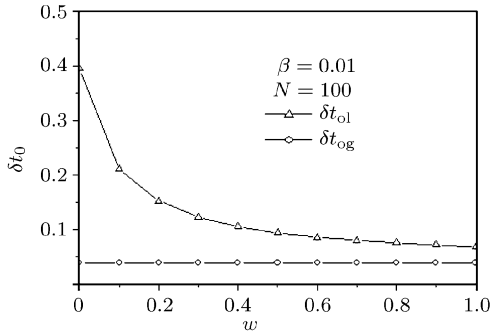


Fig.4. The dependence of spike timing precision on coupling strength.

The function of coupling among neurons is to increase the synchronous dynamics and to suppress local fluctuations. So we can know that $\delta t_{\rm ol}$ decreases with the coupling strength increasing, whereas $\delta t_{\rm og}$ is almost constant with the increase of coupling strength.

From all the calculated results, we can see that the spike timing precision of a given neuron i decreases with the noise strength increasing and increases with the coupling strength and the size of ensemble increasing; The spike timing precision of the ensemble decreases with the noise strength increasing and increases with the size of ensemble increasing. But when the size of ensemble is fixed, the spike timing precision of the network is almost constant with the coupling strength increasing.

4.1. Effects of electric synapse coupling and chemical synapse coupling on spike timing precision

Synapse transmits information among the neurons and can be divided into chemical synapse and electric synapse. Chemical synapse coupling is nonlinear. It functions only when the membrane voltage of the presynaptic neuron reaches a threshold value. And it can emit a spike, stimulates the release of neuron transmitter and exerts an influence on postsynaptic neuron. This coupling is conventionally denoted by the S function. This coupling is adopted in Ref.[20] and expressed as

$$G(x) = \frac{1}{1 + \exp(-(x-\theta)/\alpha)}, \tag{29}$$

where θ is the threshold and α is the width of the S function.

Comparing the effects of electric synapse coupling and chemical synapse coupling on the spike timing precision, the result is as follows:

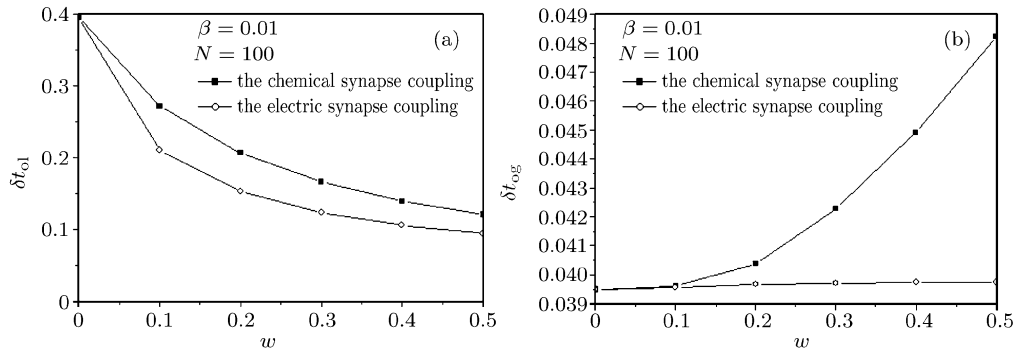


Fig.5. The w dependence of spike timing precision for electric synapse and chemical synapse (Black squares denote the chemical synapse (data cited from Ref.[20]); Open circles represent electric synapse).

It can be seen that when the coupling is the same, the values of $\delta t_{\rm ol}$ and $\delta t_{\rm og}$ for chemical synapse coupling are larger than those for electric synapse coupling, which indicates that the electric coupling has a more significant effect on the spike timing precision. And this effect increases with the coupling strength increasing.

A common feature of the temporal code is the synchronization of spikes among the populations of the neurons. The precision of spike timing is used to characterize the coincidence, so is the synchronization of the spike firings among the neurons. For the ensemble coupled by chemical synapses, the chemical synapse has a characteristic delay time in the transmission of spikes. Owing to this delay, the neurons in the ensemble are difficult to synchronize. For the electric synapse, the spikes also show slight transmission delays, but much smaller than those in the chemical synapse. If we increase the coupling strength, a large-sized system would remain synchronized. So the distribution of membrane potential is narrower and fluctuations in firing time decrease more. This would not happen in the chemical synapse, because the delay is intrinsic to the synapse and does not depend on the coupling strength. This is a fundamental, qualitative difference between chemical coupling and electrical coupling, which is worth pointing out.

5. Conclusions

A number of experimental data supporting the temporal code have been reported recently, and the temporal code assumes that the spike timing plays an important role in information transmission. Neural responses are variable in *vivo*, and the spike timing precision can be used to analyse neural signals. In this paper, we construct an N-unit FN neuron ensemble with electric synapse coupling, adopt the dynamical mean-field approximation theory and study the dependence of the spike timing precision on coupling strength, size of ensemble and noise intensity. The calculated results indicate that as the noise intensity increases, the spike timing precision decreases. As the coupling strength increases, the ensemble spike timing precision increases. For electric synapse coupling and chemical synapse coupling, the electric synapse coupling has a more important effect on spike timing precision.

Appendix A

We may expand Eqs.(1) and (2) around the average until the third-order terms. Equation (1) and (2) are expressed as

$$\frac{{\rm d}x_i}{{\rm d}t} = F(\mu_1) + F'(\mu_1)\delta x_i + \frac{1}{2}F^{(2)}(\mu_1)\delta x_i^2 + \frac{1}{6}F^{(3)}(\mu_1)\delta x_i^3 - c\mu_2 - c\delta y_i + I_i^{(c)} + I_i^{(e)} + \xi_i, \tag{A1}$$

$$\frac{{\rm d}y_i}{{\rm d}t} = b\mu_1 - {\rm d}\mu_2 + b\delta x_i - d\delta y_i + e, \tag{A2}$$

where

$$I_i^{(c)} = \frac{w}{N}\sum_{j(\neq i)}\delta x_j(t) - w\left(1 - \frac{1}{N}\right)\delta x_i(t). \tag{A3}$$

With the formulas (11), (A1), (A2) and (A3), we can have

$$\frac{{\rm d}\delta x_i}{{\rm d}t} = +f_1\delta x_i + f_2(\delta x_i^2 - \gamma_{11}) + f_2\delta x_i^3 - c\delta y_i + \delta I_i^{(c)} + \xi_i, \tag{A4}$$

$$\frac{{\rm d}\delta y_i}{{\rm d}t} = b\delta x_i - {\rm d}\delta y_i, \tag{A5}$$

where

$$\delta I_i^{(\rm c)} = \frac{w}{N}\sum_{j(\neq i)}\delta x_j(t) - w\left(1 - \frac{1}{N}\right)\delta x_i(t). \tag{A6}$$

By using Eqs.(A1) – (A4), we can obtain Eq.(13), and from Eqs.(A2) – (A5), we can have Eq.(14).

$$\frac{\mathrm{d}\gamma_{11}}{\mathrm{d}t} = \frac{1}{N}\sum_i \left\langle 2\delta x_i \frac{\mathrm{d}\delta x_i}{\mathrm{d}t} \right\rangle, \tag{A7}$$

$$\frac{\mathrm{d}\gamma_{22}}{\mathrm{d}t} = \frac{1}{N}\sum_i \left\langle 2\delta y_i \frac{\mathrm{d}\delta y_i}{\mathrm{d}t} \right\rangle, \tag{A8}$$

$$\frac{\mathrm{d}\gamma_{12}}{\mathrm{d}t} = \frac{1}{N}\sum_i \left\langle \delta y_i \frac{\mathrm{d}\delta x_i}{\mathrm{d}t} + \delta x_i \frac{\mathrm{d}\delta y_i}{\mathrm{d}t} \right\rangle, \tag{A9}$$

$$\frac{\mathrm{d}\rho_{11}}{\mathrm{d}t} = \frac{1}{N^2}\sum_j\sum_i \left\langle 2\delta x_i \frac{\mathrm{d}\delta x_i}{\mathrm{d}t} \right\rangle, \tag{A10}$$

$$\frac{\mathrm{d}\rho_{22}}{\mathrm{d}t} = \frac{1}{N^2}\sum_j\sum_i \left\langle 2\delta y_i \frac{\mathrm{d}\delta y_i}{\mathrm{d}t} \right\rangle, \tag{A11}$$

$$\frac{\mathrm{d}\rho_{12}}{\mathrm{d}t} = \frac{1}{N^2}\sum_j\sum_i \left\langle \delta y_i \frac{\mathrm{d}\delta x_i}{\mathrm{d}t} + \delta x_i \frac{\mathrm{d}\delta y_i}{\mathrm{d}t} \right\rangle, \tag{A12}$$

We have adopted the following Gaussian decoupling and mean-field approximation.[18]

1) The fourth-order variances are assumed to be

$$\frac{1}{N}\sum_i \langle \delta x_i^4 \rangle \cong 3\gamma_{11}\gamma_{11}, \tag{A13}$$

$$\frac{1}{N}\sum_i \langle \delta x_i^3 \delta y_i \rangle \cong 3\gamma_{11}\gamma_{12}, \tag{A14}$$

$$\frac{1}{N^2}\sum_j\sum_i \langle \delta x_i \delta x_j^3 \rangle \cong 3\gamma_{11}\rho_{11}, \tag{A15}$$

$$\frac{1}{N^2}\sum_j\sum_i \langle \delta y_i \delta x_j^3 \rangle \cong 3\gamma_{11}\rho_{12}, \tag{A16}$$

With the other fourth-order terms set to be zero.

2) The third-order variances and terms higher than the fourth order are neglected. From Eqs.(A1) – (A12), we can obtain Eqs.(15) – (20).

Appendix B

The nth firing time of a given neuron i in the network t_{oin} is determined by the time when $x_i(t)$ crosses the threshold θ, namely

$$t_{\mathrm{oin}} = t|_{x_i(t)=\theta} \text{ with } \dot{x}_i > 0 \text{ and } t \geq t_{\mathrm{oin}-1} + \tau_{\mathrm{r}}, \tag{B1}$$

where τ_{r} is the refractory period.

When the noise intensity is very small, the membrane voltage of neuron obeys the normal distribution. So we can have the following distribution for the membrane voltage $x_i(t)$:

$$P(x_i) = \simeq \tfrac{1}{\rho_{\mathrm{l}}}\phi\left(\frac{x_i - \mu_1}{\rho_{\mathrm{l}}}\right), \tag{B2}$$

where $\phi(x)$ denotes the normal distribution function

$$\phi(x) \simeq \frac{1}{\sqrt{2\pi}}\exp\left(-\frac{x^2}{2}\right). \tag{B3}$$

The probability $W_{\mathrm{oi}}(t)$ of $x_i(t)$ at t being above the threshold θ is given by

$$W_{\mathrm{oi}}(t) = 1 - \int_{-\infty}^{\theta} \phi\left(\frac{x_i - \mu_1}{\rho_{\mathrm{l}}}\right). \tag{B4}$$

From Eq.(B4), we can obtain Eq.(22).

References

[1] deCharms R C 1998 *Proc. Natl. Acad. Sci.* U.S.A. **95** 15166

[2] Eggermont J J 1998 *Neurosci. Biobehav. Rev.* **22** 355

[3] Ursey W M and Reid R C 1999 *Annu. Rev. Physiol.* **61** 435

[4] deCharms R C and Zador A 2000 *Annu. Rev. Neurosci.* **23** 613

[5] Pouget A, Dayan P and Zemel R 2000 *Nat. Neurosci.* **1** 125

[6] Wang Q Y, Lu Q S and Wang H X 2005 *Chin. Phys.* **14** 2189

[7] Jin W Y, Xu J X, Wu Y and Hong L 2004 *Chin. Phys.* **13** 63

[8] Thorpe S, Fize D and Marlot C 1996 *Nature* (London) **381** 520

[9] Pouget A, Dayan P and Zemel R 2000 *Nature* **1** 125

[10] Gammaitoni L, H?nggi P, Jung P and Marchesoni F 1998 *Rev. Mod. Phys.* **70** 223

[11] Collins J J, Chow C C and Imhoff T T 1995 *Nature* (London) **376** 236

[12] Yang Zh Q and Lu Q S 2006 *Chin. Phys.* **15** 518

[13] Wu Y, Xu J X, He D H and Jin W Y 2005 *Acta Phys. Sin.* **54** 3457 (in Chinese)

[14] Gluckman B J, Netoff T I, Neel E J, Ditto W L, Spano M L and Schiff S J 1996 *Phys. Rev. Lett.* **77** 4098

[15] Pei X, Wilkens L and Moss F 1996 *Phys. Rev. Lett.* **77** 4679

[16] Yu Y G, Liu F, Wang J and Wang W 2001 *Phys. Lett.* A **282** 23

[17] Rokem A, Watzl S, Gollisch T, Stemmler M, Andreas V M Herz and Inés Samengo 2006 *J. Neurophysiol.* **95** 2541

[18] van Rossum M C W, O'Brien B J and Smith R G 2003 *J. Neurophysiol.* **89** 2406

[19] Stiber M 2005 *Neural Computation* **17** 1577

[20] Hasegawa H 2003 *Phys. Rev.* E **67** 041903

[21] Hasegawa H 2004 *Phys. Rev.* E **70** 021911

[22] Rodriguez R and Tuckwell H C 1996 *Phys. Rev.* E **54** 5585

[23] Tuckwell H C and Rodriguez R 1998 *J. Comput. Neurosci.* **5** 91

[24] Tanabe S and Pakdaman K 2001 *Phys. Rev.* E **63** 031911

[25] Stevens C F and Zador A M 1998 *Nat. Neurosci.* **1** 210

[26] Mainen Z F and Sejnowski T J 1995 *Science* **268** 1503

[27] Rose J E, Brugge J F, Anderson D J and Hind J E 1967 *J. Neurophysiol.* **30** 769