The Fifth International Conference
on Neural Information Processing
Kitakyushu, Japan October 21-23,1998

NONLINEAR DYNAMICS GENERATING THETA PHASE PRECESSION IN HIPPOCAMPAL CLOSED CIRCUIT AND GENERATION OF EPISODIC MEMORY

Yoko Yamaguchi *,** Bruce L. McNaughton **
* yokoy@j.dendai.ac.jp, ** bruce@nsma.arizona.edu

* Dept. of Information Sciences, College of Science and Engineering, Tokyo Denki University,
Hatoyama, Saitama 350-0394, Japan
**ARL Division of Neural Systems, Memory and Aging, The University of Arizona,
Tucson Arizona 85724-5115, USA

ABSTRACT

We propose a hypothesis for the neural mechanism underlying theta phase precession of place cell firing, based on phase locking by mean field interactions in a coupled-oscillator system, followed by inheritance through the hippocampal circuit. It is demonstrated that the temporal pattern inherited through the network can result in encoding of the temporal sequence of experience as asymmetric connections in the recurrent neural network. The phase distribution predicted by the model is discussed in comparison with experimental observations.

KEYWORDS: hippocampus, theta rhythm, phase locking, phase precession, episodic memory

1. INTRODUCTION

The hippocampal theta rhythm is a regular and robust field potential oscillation observed in rodents during locomotion. Individual pyramidal cells fire robustly when the rat is in specific portions of the environment. These portions are called "place fields", and the population activity has been proposed by O'Keefe and Nadel (1978) to represent a cognitive map. The activities of place units are known to be periodically modulated by the theta rhythm and O'Keefe and Recce (1993) have shown that the spatially specific firing of pyramidal cells in the CA1 region has an interesting temporal correlation with the theta field potential, the phase of the theta cycle at which a pyramidal cell fires advances as the rat passes through the place field. This phase shift effect is called "phase precession". By using large-scale parallel recording, Skaggs et al. (1996) showed that the phase precession is robust and coherent across neural populations, not only in the CA1 field, but also in the fascia dentata, with a phase difference of 90 degrees. They suggested that phase precesion may be generated in an early stage of the hippocampus (perhaps the entorhinal cortex) and inherited by the subsequent modules of the hippocampal circuit.

The neural mechanism of phase precession is an open question and inseparable from its computational function. Tsodyks et al. (1996) proposed that theta phase precession is generated as a result of asymmetric spread of activation through the network (possibly CA3), caused by asymmetry in the synaptic interactions. In this model, the velocity of activity through the place cell sequence is faster than the velocity of the rat through the corresponding places. At the end of each theta cycle, the activity 'jumps' back to the cells corresponding to the current location. A two dimensional variant of this model (Samsonovich and McNaughton, 1997) used head direction cells rather than asymmetric connections to break the symmetry. It has been proposed (Skaggs et al.,1996) that a functional consequence of phase precession is to represent the temporal sequence of place codes in a compressed form, thus facilitating the establishment of asymmetrical connections through the known temporal dynamics of the LTP process.

In the present paper, we propose biologically plausible nonlinear dynamics that can generate phase precession in a neural network with no spatial structure, and study the consequences of these dynamics on the possible encoding of temporal sequences using a hippocampal network model.

2. HYPOTHESIS

It is known that nonlinear oscillators coupled with dissipative mean field interactions show phase locking where the relative phase to the mean field oscillation depends on the individual native frequencies (Fig.1)(see e.g., Yamaguchi and Shimizu,1984). The collective phase locking generates a sequence of oscillations in the order of the native frequencies of the units. Theta phase precession can be phenomenologically reconstructed based on this type of phase locking.

When a neural oscillator has a gradual and slow change in its native frequency of oscillation, and interacts with all other neural oscillators or the local field oscillation, its phase relative to the mean phase changes gradually, as shown in Fig.2. The increase of frequency results in gradual change in the relative phase from behind to advanced. It is plausible that the native frequencies of neural oscillators in the superficial entorhinal cortex may increase steadily during the activation period because of some intrinsic physiological process, thus giving rise to phase precession effects

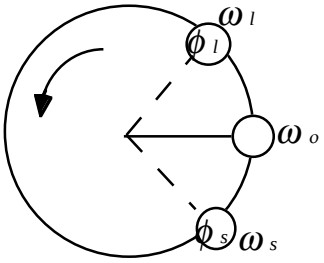

Figure 1 Phase locking in coupled oscillator system. The inequality of native frequencies $\omega_s < \omega_0 < \omega_l$ gives the relative phase relation $\phi_s < \phi_0 < \phi_l$.

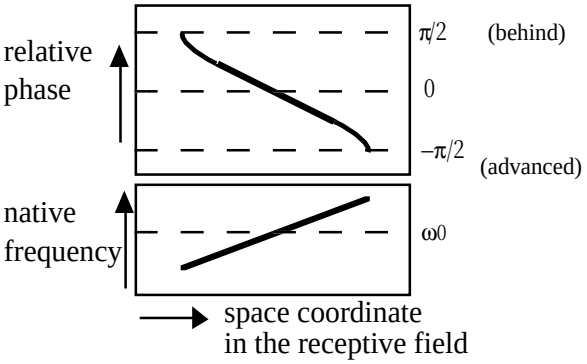

Figure 2 The slow and gradual change of the native frequency (lower) and the relative phase fixed by the mean field interaction(upper).

which are transmitted to other hippocampal subfields. Furthermore, it is possible that the phase difference between dentate gyrus(DG) and CA1 observed by Skaggs et al. (1996) is also present between the superficial and deeper layers (Mitchel and Ranck, 1980; Alonso and Garcia-Austt 1987), which represent, respectively, the input to and output from the hippocampal circuit.

Thus, we propose the hypothesis: 1) phase locking by mean field interactions results in phase precession at the earliest stage of the hippocampal closed loop, in layer II of the entorhinal cortex (ECII for short). 2) The temporally structured pattern of activities in ECII is transmitted into the hippocampal circuit, leading to asymmetric connections based on temporal asymmetry of the LTP mechanism. 3) The final stage of the loop, the deeper layer of the entorhinal cortex (ECIII~VI) is considered to have activities with a phase difference of 90 degrees from that of ECII. That is, the phase precession generated in ECII is roughly duplicated in ECIII with phase delay of 90 degrees.

ECII and ECIII~VI not only transmit signals into circuit but also give the boundary condition of activities in the hippocampal circuit as proposed by Yamaguchi (1996). These boundary conditions give constraints to

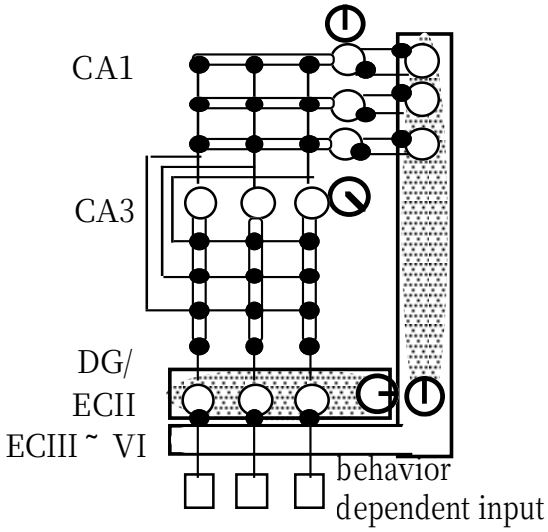

Figure 3 Schematic illustration of the hippocampal circuit model. Open circles denote neural units and small closed circle denote synaptic connections. Open circles with bars represent the local field oscillators and their relative phases.

self-organization of neural networks in the CA3 and CA1 fields representing the temporal structure of the experience.

3. MODEL

The model for nonlinear dynamics of theta rhythm in the hippocampal circuit is illustrated in Fig.3. It consists of four layers termed ECII/DG, CA3, CA1 and ECIII~VI. For simplicity's sake DG is not explicitly described. The connections between these layers are feedforward. The exception is a backward connection from ECIII~VI to CA1. The external input vector $\boldsymbol{I}(t) = \{I_i(t)\}$, which is dependent on the rat's behavior, is fed into ECII/DG. For simplicity, $I_i(t)$ is assumed to correspond to the i-th place field. Every unit in the layer is considered as representative of the population of neural elements.

Temporal evolution of the unit activity shall be described by the phase model, since it is the reduced form of various nonlinear oscillators in general and available for analyses of phase-locking (Kuramoto 1984). The equation of the phase model was developed for neural units with interactions by pulse densities (Yamaguchi 1996). The fundamental equation for the i-th unit in the model is given as follows.

$$\dot{\phi}_i = \omega_i + (\beta_i - J_i(t))\sin\phi_i \qquad (1)$$

where the variable of this motion is the phase ϕ_i (mod 2π) by which the membrane potential of the unit is

given by $\cos\phi_i$. The quantities ω_i and $ß_i$ represent the native angular frequency and the stabilization coefficient, respectively. The term $J_i(t)$ represents the input of external current and coupling of other units that give deporalization or hyperpolarization effects to the membrane potential.

Equation (1) results in a stable limit cycle in the case of $|\omega_i/(ß_i - J_i(t))| > 1$ while otherwise it has a stable equilibrium point with excitability by super-threshold stimuli. The output of the unit is given by a sigmoidal function of the membrane potential. The mean field are described by a local field oscillator unit in each layer. Synaptic plasticity is considered in CA3 recurrent connections and those from CA3 to CA1 as a modified Hebb rule with time delay τ.

4. COMPUTER EXPERIMENTS

We consider the rat is running with a constant velocity to give a sequential increase of $\{I_i(t)\}$. Figure 4 shows an example of results obtained by computer experiments with the model. It is seen that fundamental property of theta phase precession is reconstructed. An asymmetric structure of the connection matrix in CA3 was obtained in these computer experiments. The projection from CA3 to CA1 was obtained so that the diagonal components give maxima. It means that the consistent phase relation between CA3 and CA1 units is selectively potentiated to result in a closed loop from ECII through ECIII~VI so that units in ECII and ECIII~VI have phase difference of 90 degrees. Thus, phase precession generated in ECII, as a temporal pattern in a spatially uniform system, is inherited at its output, resulting in self-organization of a neural network encoding temporal sequences and completion of closed loops in active column of EC.

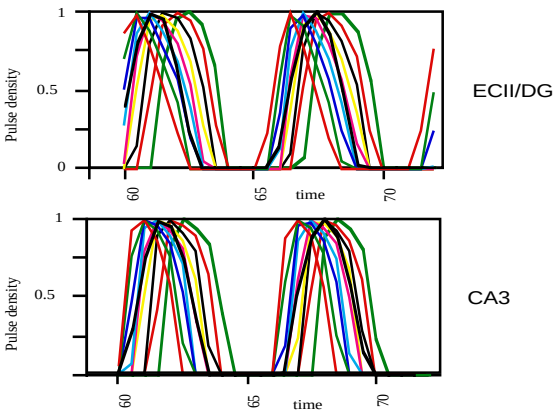

Fig.4a Results of computer experiments. Temporal sequence of units firing generated in ECII/DE (upper) is inherited by units in CA3 (lower). (Each layer includes nine units.)

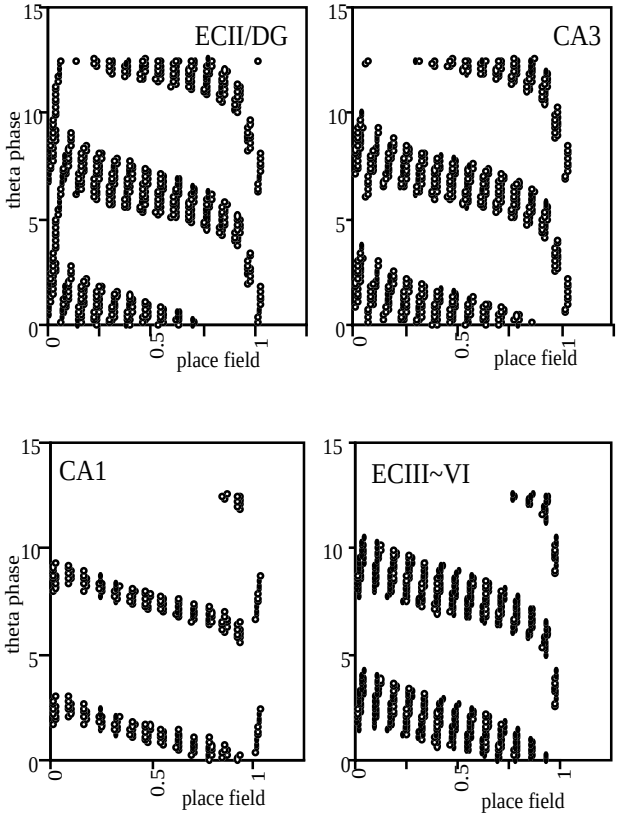

Fig.4b Theta phase distribution of unit firing. Pulse density activities of two units from each layer (units 1 and 7) are shown superimposed. The theta phase is measured with the reference of local field oscillation in ECII/DG in all layers.

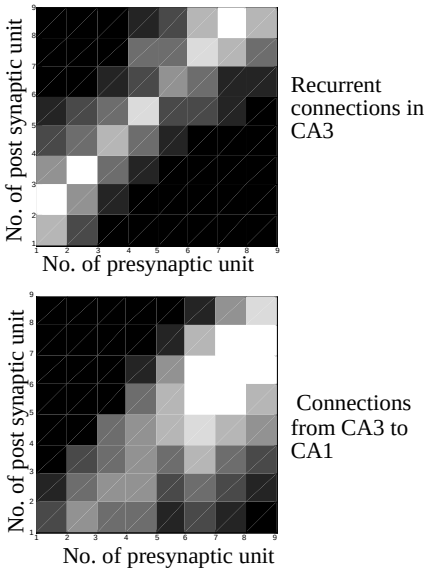

Fig.4c Weights of connection matrix obtained after the one episode of running from place field 1 to 9. White squares indicate the largest value of weights.

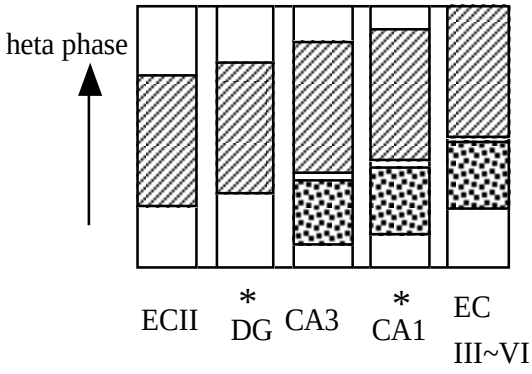

Figure 5 Theta phase precession in individual fields of the hippocampus as a consequence of our present hypothesis. The shaded parts and dotted parts denote two different classes of inheritance. Only the experimental data in fields with * are currently available.

5. DISCUSSION

The theta phase precession reconstructed by the present model is consistent in the range of phase distribution observed at DG. The range in CA1 is larger than the present model. Detailed analysis by Skaggs et al. (1996) suggests that the phase distribution is divided into two components. One component may be the portion inherited from DG while the other seems to emerge additionally advanced to the inheriance component as shown in Fig. 5. This suggests that some sustained activity emerges in CA3 and is inherited by CA1 and the deeper layer of EC. Thus, two components of phase precession observed in CA1 suggest two classes of inheritance. This remains to be proven experimentally. DG is not explicitly evaluated since the present study is restricted to dynamics in a single episode. DG is necessary for efficient input coding e.g., coding of multiple environments or multiple episode, as is mentioned by Marr(1971), and by McNaughton BL, Morris M.(1987).

6. CONCLUSION

Our present hypothesis based on mean field interactions and inheritance within the circuit reconstructs theta phase precessison in general agreement with experimental observations. The temporal compression of successive, overlapping portions of the input sequence that results from phase precession enables self-organization of a neural network encoding the temporal structure of given novel experience. Furthermore, the two temporal sequences of phase precession in the superficial and deeper layers of the entorhinal cortex raises a quite new issue on cortico-hippocampal interactions.

The authors thank Dr. Bill Skaggs, for valuable discussion and encouragement. This research is partially supported by Japanese Grant-in-Aid for Science Research Fund from Ministry of Education, Science and Culture (Nos. 09268236 and 10164243) and by U.S. P.H.S. NS20331.

REFERENCES

[1] O'KeefeJ., Recce M.L. (1993) Phase relationship between hippocampal place units and the EEG theta rhythm. Hippocammpus 3:317-330.

[2] Skaggs WE., McNaughton BL., Willson MA., Barnes CA. (1996) Theta Phase Precession in Hippocampal Neuronal Populations and the Compression of Temporal Sequence. Hippoampus 6:149-172.

[3] O'Keefe J., Nadel L. (1978) The hippocampus as a cognitive map. Oxford: Clarendon Press.

[4] Tsodyks MV., Skaggs WE., Sejnowski TJ., McNaughton BL.(1996) Population Dynamics and Theta Rhythm Phase Precession of Hippocampal Place Cell Firing: A Spiking Neuron Model, Hippocampus 6:271-280.

[5] Samsonovich A., McNaughton BL.(1997) Path Integration and Cognitive Mapping in a Continuous Attractor Neural Network Model. J. Neurosci. 17:5900-5920.

[6]Yamaguchi and Shimizu H (1984) Theory of Self-Synchronization in the Presence of Native Frequency Distribution and external noises. Physica 11D:212-226.

[7] Mitchell SJ, Rank JB, JR. (1980) Generation of theta rhythm in medial entorhinal cortex of freely moving rats. Brain Research, 189:49-66.

[8] Alonso A., Garcia-Austt E.(1987) Neural Sources of theta rhythm in the entorhinal cortex of the rat I Laminar distribution of theta field potentials. Exp Brain Res 67:493-501.

[9] Yamaguchi Y. (1996) Synchronization of Theta Activities as a Supervisory Mechanism of the Memory Formation in a Neural Network Model of the Hippocampus, Kato ed., *Functions and Clinical Relevance of the Hippocampus*, Elsevier, 339-344.

[10] Kuramoto Y (1984) Chemical Oscillations, Waves and Turbulence (Springer, Berlin).

[11] Marr D. (1971) Simple memory: A theory for archicortex. Proc. R. Soc. London Ser.B.262,23-81.

[12] McNaughton BL, Morris RGM.(1987) Hippocampal synaptic enhancement and information storage within a distributed memory system. TINS 10:408-415.