Switching among Alternate Synchronization Patterns in an Electrically Coupled Neuronal Model

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CONTENTS

ABSTRACT

We show that the electrically coupled Hindmarsh-Rose neuronal model exhibits various patterns of phase locking at fixed parameter value. Through the analysis of the effective coupling, the system is shown to be stabilized in one of these patterns according to the initial conditions. This corresponds to the parameter-tuning independent mode-switching mechanism that changes the electrical output of neuronal systems. It is also presented how the stable fixed points of the effective coupling which characterize the phase locking patterns depend on the external current.

I. INTRODUCTION

It has been recently shown that diffusive coupling generates the antiphase locking on spiking level at weak coupling [1] and induces dephasing of limit cycle oscillators which results in a new bursting behavior [2]. In this paper we show that various phase locking patterns may coexist in a neuronal system with excitatory electrical coupling at fixed parameter. In other words, the system is ultimately stabilized in one of those phase locking patterns. This stabilized pattern, however, may be reformed to a new phase locking pattern by a slight perturbation at fixed parameter.

The activity changes of theoretical neuronal systems have been generated by two main mechanisms [3]. One is the parameter-dependent mechanism by changing the electrical activities of neurons, by changing the synaptic coupling, etc. The other is parameter-independent one via mode-switching with fixed parameters, which can be manifested by changing the transient inputs. The purpose of this paper is to analyze quantitatively the latter. We show that by changing the initial conditions, which corresponds to changing the transient inputs, the system is switched from a locking mode to another with fixed parameters.

We analyze quantitatively the phase locking patterns through the effective coupling [4] of a limit cycle oscillator model. It is shown that the model with diffusive coupling exhibit dephasing of oscillators on the limit cycle at weak coupling. The dephasing mechanism is explained on the basis of the phase shift analysis and of velocity divergence across the limit cycle. At given values of parameters and coupling strength it is shown that there exist various types of phase locking corresponding to the stable fixed points of the effective coupling. We also present the dependence of the fixed points, therefore, the phase locking patterns, on the external current. For the sake of simplicity, we consider the coupled system of two neurons in this paper. In section II, the one neuron system is explained. The results of this paper are presented in section III. The summary and discussions are contained in section IV.

II. THE MODEL

In this paper we study a system of Hindmarsh-Rose (HR) neurons [5]. Even though this model is not based on physiology, it simulates some features observed in neuronal bursting. The HR model has been originally introduced to give a long interspike interval and burst typical of real neurons. The three variable HR model is given by

$$\begin{aligned} \frac{dX}{dt} &= Y - aX^3 + bX^2 - Z + I \\ \frac{dY}{dt} &= c - dX^2 - Y \\ \frac{dZ}{dt} &= r[s(X-\alpha) - Z] \end{aligned} \tag{1}$$

X is thought of as the membrane voltage of the neuron, Y as the recovery variable, and Z as a slow adaptation current. I is the uniform exter-

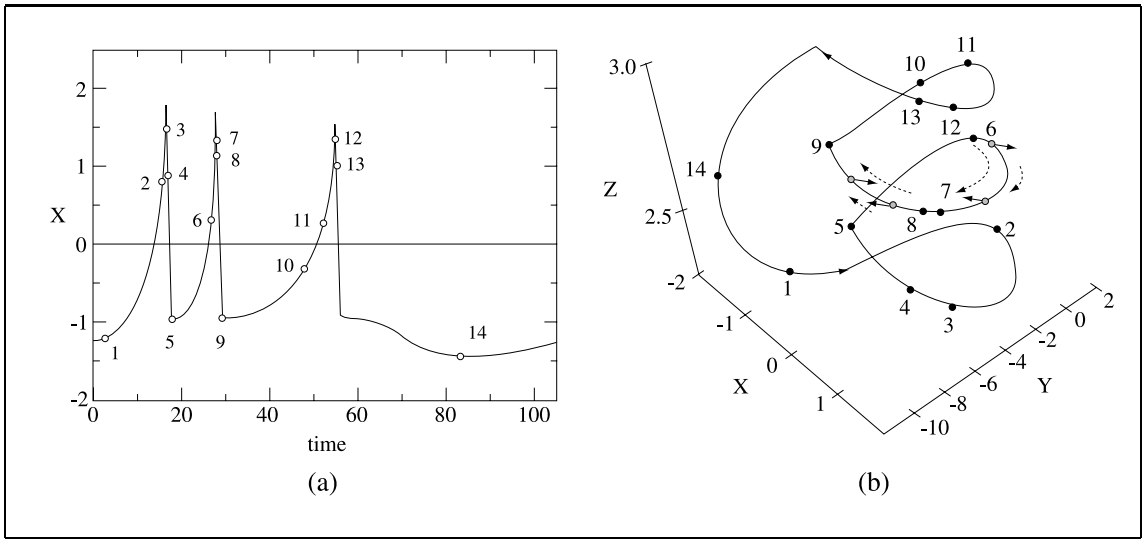

Fig. 1. (a) Membrane voltage, X, (b) 3 (X, Y, Z) dimensional trajectory of Eq. (1). The circles in (a) match the points, 1-14, in (b). The solid arrows show the direction of the force due to the coupling. The difference of the magnitude of the dotted arrows indicates the velocity divergence across the limit cycle, which results in the dephasing.

nal current. α is the membrane voltage when the neuron is in a stable fixed point of the null clines $\frac{dX}{dt}=0$, $\frac{dY}{dt}=0$ for $I=0$. Being a slow variable, Z is considered as a parameter when the fixed points are found. We'll fix the parameters to the values $a=1.0$, $b=3.0$, $c=1.0$, $d=5.0$, $s=4.0$, and $r=0.001$.

A bursting time course for a single neuron is shown in Fig. 1(a) for $I=2.7$ for one period of bursting. The bursting mechanism in the mathematical neuronal systems has been extensively studied in [5], [6]. We refer to [5] for a detailed bursting mechanism of the HR model. A brief explanation on the bursting mechanism of HR model is as follows. As in Fig. 1(a), each burst contains three spikes followed by a quiescent state. The spikes are generated when the neuron is on the limit cycle which surrounds an unstable fixed point. As can be seen in Fig. 1(a), the spike interval becomes longer in a burst as the limit cycle trajectory gets closer to the saddle point separatrix, and eventually the saddle-loop bifurcation occurs. Then the firing ceases and the neuron stays at the stable fixed point until a stable limit cycle appears through a homoclinic connection so that another burst starts. The 3 dimensional (X, Y, and Z) trajectories are shown in Fig. 1(b).

The bursting oscillation is originated from the evolution of the slow variable Z which switches the dynamics of the system between the steady state and the oscillatory state on the limit cycle by changing the geometry of the stable fixed point, saddle point, and the unstable fixed point. According to the detailed role

of the slow and the fast variables, a classification scheme for the bursting oscillation has been proposed by Bertram *et al* [7].

III. RESULTS OF THE TWO NEURON MODEL

The electrically coupled HR model is described by (1) with an additional linear voltage-coupling term, which is given by

$$\begin{aligned}\frac{dX_i}{dt} &= Y_i - aX_i{}^3 + bX_i{}^2 - Z_i + I - K(X_i - X_j)\\ \frac{dY_i}{dt} &= c - dX_i^2 - Y_i\\ \frac{dZ_i}{dt} &= r[s(X_i - \alpha) - Z_i], \end{aligned} \quad (2)$$

where $i, j = 1, 2$ and K denotes the coupling strength.

In order to describe the phase dynamics of the coupling we calculate the effective interactions. Assuming the weak coupling, where we fix $K = 0.01$, the system may be approximated as a phase model [4], where the phase ϕ of a neuron is defined as $\frac{d\phi(X)}{dt} = 1$. The effective coupling $\Gamma(\psi)$ is defined as

$$\frac{d\psi}{dt} = \Gamma(\psi) = \frac{1}{2\pi}\int_0^{2\pi} d\phi Z(\phi)\cdot P(\phi, \psi). \quad (3)$$

where ψ is the difference of the phase of the two neurons, $\phi_1 - \phi_2$, and $Z\cdot P$ is the phase shift defined as $Z(\phi)\cdot P(\phi, \psi) = (grad_X \phi)_{x=X_0(\phi)}\cdot P(\phi, \psi)$, where X_0 is the point on the limit cycle at phase ϕ. Here, we adapted the extended notion of phase using the concept of isochrons which are defined as a subset of domain converging to a point on the limit cycle. $P(\phi, \psi) = P(X_0(\phi), X_0(\phi+\psi))$ describes the rate of change of the state vector X of an oscillator due to the interaction with the other at phase difference ψ. $P(\phi, \psi)$ is the coupling term in (2) expressed as a function of the phases, which is considered as a small perturbation. The sensitivity function $Z(\phi) = (grad_X \phi)_{X=X_0(\phi)}$ gives the change of phase along the limit cycle caused by the change of X: we choose a point X_0 on the limit cycle and X not on the limit cycle but close to X_0, then measure the difference of the two phases corresponding to X_0 and X. The difference of the phase devided by $|X - X_0|$ is the sensitivity function.

To understand how the dephasing comes out, we plot the phase shift, $Z\cdot P$, as a function of $T = \frac{\phi}{2\pi}$ with $\psi = 0.01\pi$ in Fig. 2(a). The points, 1, 2, ..., 14, in Fig. 2(a) corresponding to the ones in Fig. 1(a) are the zeroes of the phase shift. During the period of bursting, the interaction causes the phase difference ψ alternately to increase (a positive value of $Z\cdot P$) or to decrease (a negative value of $Z\cdot P$). One notices the phase shift is almost zero when the oscillator is near the saddle point, i.e., the regions 1-2, 5-6, and 9-10, and at the stable fixed point, 14-1, where the neurons spend their most time in one period of bursting. This implies that the coupling between the neurons influences the phase difference only for a relatively short duration during the period of bursting. Averaging $Z\cdot P$ over one period of butsting as shown in (3), one obtains the positive value of the slope at the origin of the antisymmetric part of the ef-

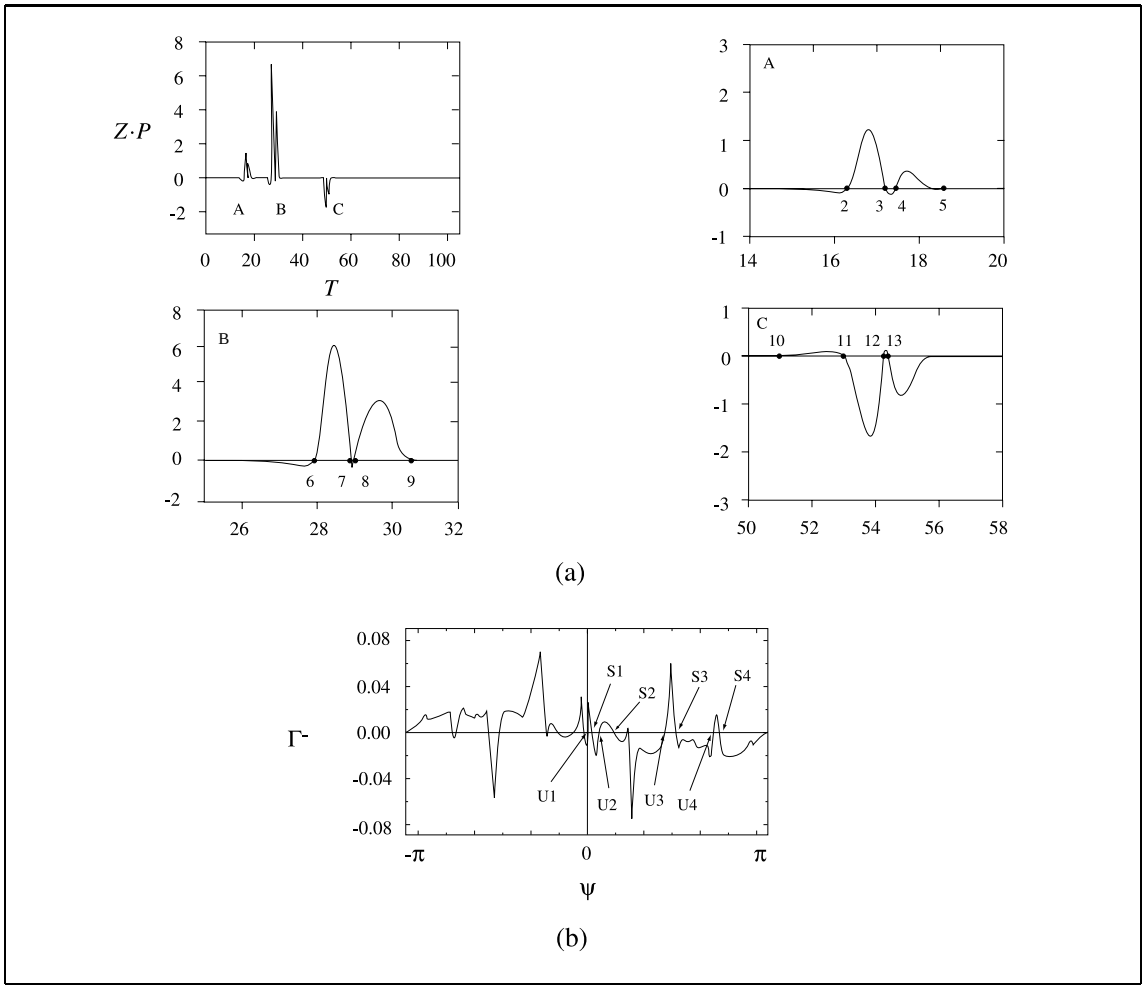

Fig. 2. (a) The phase shift $Z \cdot P$. $T = \frac{\phi}{2\pi}$, $\psi = 0.01\pi$, (b) The antisymmetric part of the effective coupling. U1-4 are unstable fixed points and S1-4 are stable fixed points. There is another pair of zero-crossings just to the right of S2. Since the unstable and stable fixed points are located at almost same point, it is hardly possible to find the phase locking pattern corresponding to the stable fixed point.

fective coupling in Fig. 2(b). This shows that the diffusive coupling of (2) leads to dephasing of the system.

This unexpected dephasing originates from the deformation of the phase flow, i.e., the difference of phase velocity across the limit cycle. As can be seen in Fig. 2(a), dephasing occurs mostly in the regions 6-7 and 8-9. Since Z is a slow variable, we consider the motion of the neuron on two $(X - Y)$ dimensional limit cycle for a clear understanding of the dephasing. The two

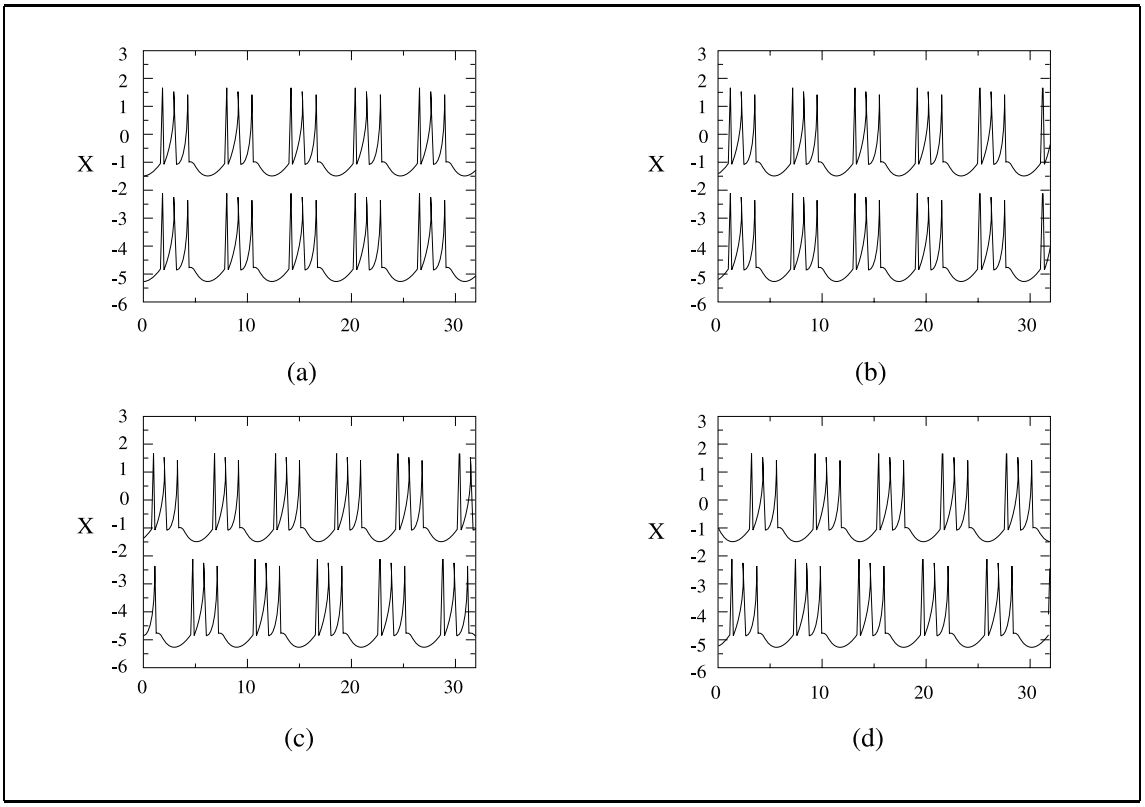

Fig. 3. Phase locking patterns. Each pattern corresponds to one of the stable fixed points in Fig. 2(b).

oscillators between 8 and 9 (or 6 and 7) exert attractive force to each other in the *X* direction due to the diffusive coupling, which can be seen as the solid arrows in Fig. 1(b). The leading oscillator is pushed inside the limit cycle, so that it moves as the dotted arrow inside the limit cycle. On the other hand, the lagging oscillator is pushed toward the leading one as the dotted arrow outside the limit cycle in Fig. 1(b). The dragging effect due to the diffusive coupling, however, is much smaller than the one of the velocity difference across the limit cycle, which can be seen as the difference of the magnitude of the two dotted arrows. This velocity difference across the limit cycle is caused by the geometry of the saddle point and its separatrix. Being closer to the separatrix of the saddle point, the oscillator outside the limit cycle is slower than the one inside the limit cycle. This velocity difference across the limit cycle results in the overall dephasing of the system [2].

In Fig. 2(b) the points S1, S2, S3, and S4 correspond to the stable fixed points. To investigate the phase locking patterns, we con-

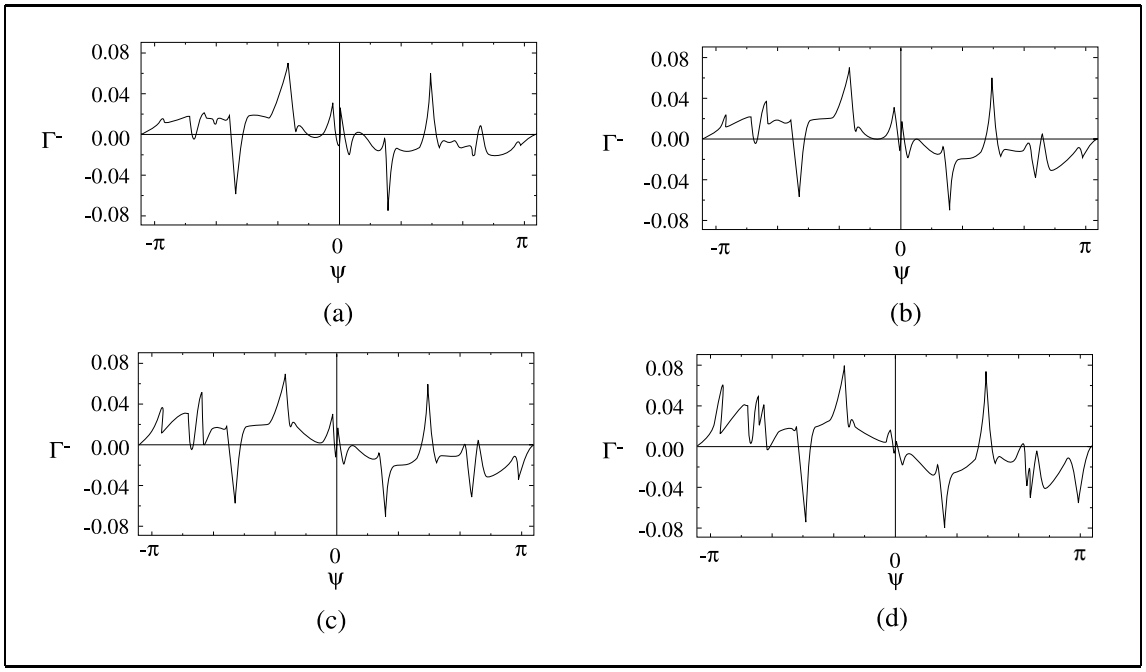

Fig. 4. Antisymmetric part of the effective coupling when (a) $I = 2.74$, (b) $I = 2.8$, (c) $I = 2.84$, and (d) $I = 2.9$. As I gets larger, the number of stabel fixed points is reduced, so that the phase locking is less likely.

sider only the antisymmetric part of the effective coupling, therefore only the positive part of the axis of phase difference in Fig. 2(b). The system is eventually stabilized in one of these points according to the initial conditions. The unstable points U1, U2, U3, and U4 plays a role of separatrix. For example, if the phase difference of the two neurons is initially given by the value between U2 and U3, it is gradually attracted to S2. Thus the system is phase locked with the phase difference given by S2. The reasoning for this is as follows. If the phase difference of the two neurons is initially given by a value between U2 and S2, the effective coupling is positive. This implies that the phase difference gets larger by (3) until it hits S2. By the same argument, the initial phase difference at a value between S2 and U3 is attracted to S2. The initial phase difference in the range of U1 (2, 3, 4) and U2 (3, 4, 1) is gradually moved to S1 (2, 3, 4) where the phase difference of the system is stabilized.

Various synchrony patterns can be seen in Fig. 3, each corresponds to one of the stable fixed points in Fig. 2(b). Obviously, these patterns are not synchronized in the diffusively coupled systems.

In Fig. 4 the dependence of the stable fixed points on the external current I is presented. The locations of fixed points move. As I gets

strong, the number of phase locking patterns is reduced and eventually the system is chaotic.

IV. SUMMARY AND DISCUSSIONS

We have shown that the diffusively coupled neuronal oscillators are not necessarily synchronized but exhibit various rhythmic phase locking patterns. Assuming weak coupling, we have analyzed the effective coupling on the limit cycle of coupled HR model with two neurons. The model has been shown to exhibit stable activity patterns coexisting at specific values of the parameters. The system is eventually stabilized in one of the coexisting patterns which correspond to one of the stable fixed points of the effective coupling according to the initial conditions. The stabilized pattern is reformed to another by a slight transient input at fixed parmeter. This corresponds to the mode-switching mechanism which changes the eletrical properties of the system with fixed parameters.

The appearance of these out-of-phase locking patterns originates from the dephasing of the coupling which is led by the velocity divergence across the limit cycle. It is not, however, always true that the diffusive coupling generates the dephasing. We have studied coupled Hodgkin-Huxley model to see whether diffusive coupling gives rise to dephasing between oscillators or not. Despite of the existence of a saddle point near the limit cycle, we were not able to find the parameter value or the coupling strength where dephasing occurs [8]. It seems that the relation between the geometry of the separatrix and the coupling direction (in our case, X) is crucial to determine the phase dynamics of the system.

As the external current increases for fixed coupling constant, the number of spikes in a burst grows and eventually the system becomes chaotic. As in the case of synaptic coupling [9], the system shows chaotic synchronization, where the fluctuations are correlated. This chaotic synchronization is an intermediate state to the wholly chaotic state as the external current I increases. The form of chaos arising from a model of bursting in excitable membranes have been studied in [10].

The effects of the electrical coupling in a model of the pyloric network of the stomatogastric ganglion in crustacea have been extensively studied to understand how the coupling regulates the firing frequency and burst duration of AB interneuron [11]. We performed a numerical analysis of the electrically coupled HR model with N neurons (unpublished, but with the same numerical technique used in this paper) to study the collective behavior of diffusively coupled systems. In the weak coupling regime, the system is split into clusters. At some range of coupling constant and external current values, the number of clusters are shown to oscillate. These clustering phenomena originate from the dephasing of the coupling, whose quantitative analysis is left for further study.

The rhythemic activity of the oscillatory networks such as the swimming and heartbeat of invertebrates has been widely understood via the post-inhibitory rebound mechanism [12]-[14]. Here, the alternating pattern of activity is produced through the post-inhibitory rebound between the inhibitory coupled neurons or groups of neurons. Adjusting the external current value at some fixed parameter or the coupling strength of (2), we observed various synchrony patterns: the in (anti)-phase locking patterns both on the spiking and on the bursting levels. Our results, therefore, suggest another route to generate the rhythemic patterns, which, however, should be supported by the physiological facts.

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