Chapter 16
Dynamics of Intermittent Synchronization of Neural Activity

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16.1 Synchronized Neural Activity

Electrical activity of neurons and neural populations in the brain frequently exhibits some degree of synchrony (defined in some specific ways). Multiple experimental studies indicate that neural synchronization is important for various functions of the brain. For example, neural synchrony has been observed in relation to perception [11], memory [12], other cognitive functions [13], and motor functions [16, 31]. Different mechanisms underlying the involvement of oscillatory neural activity in neural function have been considered [8]. Abnormally high (and sometimes abnormally low) strength of neural synchrony has been implied to be critical for the symptoms of several neurological and psychiatric disorders [32, 34], including Parkinson's disease (e.g., [29]) and schizophrenia (e.g., [35]).

Synchronization is a widely observed phenomenon and has been traditionally studied with the methods of physics and nonlinear dynamics [23]. One of the scholars, who early recognized the importance and relevance of physical studies of synchronization to neuroscience was Misha (Mikhail Izrailevich, as one would

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politely call him in his native Russian) Rabinovich. He inspired his students and engaged his collaborators [2], and authored many important and elegant studies of the mechanisms and functions of neural synchrony (for example, [1, 7, 10, 17, 33]). Misha pointed out that neural synchronization may have some peculiar properties, which are not frequently observed in other synchronized systems in nature, because of the very specific functions of neural systems [18].

Interestingly, Misha Rabinovich discussed the potential generality of transient dynamics in neuroscience [24–26]. The transient (intermittent) synchrony appears to be the norm in the synchronized dynamics of neural circuits of the brain. Even at the rest state, without any transient stimuli, perfect synchrony in the brain has not been reported. This is probably not very surprising. At the rest state these circuits should be ready to respond to different stimuli. If they were in a completely synchronized stable state, it would probably require more time and/or efforts to respond to stimuli.

16.2   Fine Temporal Structure of Intermittent Synchronization

Neuroscience traditionally operates with observables. Usually, in the context of neural synchrony, these are intra- or extracellularly recorded electric potentials as a function of time. However, a consideration of the dynamics of synchronization and desynchronization in terms of the phase space helps to understand the nature of these phenomena.

16.2.1   *Phase Space-Based View and Time-Series-Based View of Synchronized and Desynchronized Episodes*

Let's think of coupled neurons or neural populations where each individual unit exhibits oscillatory dynamics. Coupling is strong enough and dynamic is synchronous so that there is a stable synchronization manifold in the phase space. As we discussed above, this is a straightforward, but not an experimentally realistic scenario. Experimentally relevant, but relatively weak intermittent synchrony may correspond to the following dynamics: the system moves into vicinity of an unstable synchronization manifold, but eventually leaves it because this manifold is unstable. Since the synchrony is relatively weak, the system will spend substantial amount of time away from the synchronization manifold. In this case a study of the properties of synchronization manifold (e.g., its loss of stability and associated types of intermittency) informs of what happens in the system of interest only for a small fraction of time.

Unlike the stability of the synchronization manifold, however, the properties of the periphery of the phase space are not universal in general. But it does not

mean they are irrelevant to the dynamics of the neuronal networks. To connect these ideas to experimental data we will look at these issues from the time-series analysis perspective.

16.2.2 Dynamics of Desynchronization Episodes: Time-Series Analysis

The phase can be extracted from a "good" oscillatory data (the data with relatively narrow and prominent peak in the spectrum) in different ways, we will use Hilbert phase [23]. Using Hilbert transform one obtains an analytic signal $\xi(t)$ from a real time series $x(t)$ as follows:

$$\xi(t) = x(t) + i\bar{x}(t) \tag{16.1}$$

$$\bar{x}(t) = \frac{1}{\pi} \, \mathrm{PV} \int_{-\infty}^{\infty} \frac{x(\tau)}{t - \tau} d\tau. \tag{16.2}$$

Let the phase of the analytic signal $\xi(t)$, say $\varphi(t)$, be the Hilbert phase of the time series. Then it is given by

$$z(t) = \frac{\xi(t)}{\|\xi(t)\|} = e^{i\varphi(t)}. \tag{16.3}$$

If the phase difference between two oscillators tends to be close (in some specific sense) to some constant value, then we can consider this as a synchronized dynamics.

One can compute a fairly standard phase locking index for two phases $\varphi_1(t)$ and $\varphi_2(t)$:

$$\gamma = \|\frac{1}{N} \sum_{j=1}^{N} e^{i\Phi_j}\|, \tag{16.4}$$

where $\Phi_j = \varphi_1(t_j) - \varphi_2(t_j)$ and N is the number of data points (for the case of discrete time-series). This index varies between zero (no phase locking) and one (perfect phase locking) [23].

For further analysis (originally developed in [3, 21]), we consider a first-return map for the phase difference (see also [30]). In other words, we are considering whether the phase difference is close to its preferred (locked) state or not once per cycle of oscillations (we assume this preferred state exists and can be extracted from the data, otherwise the described procedure does not make sense). How close it should be depends on a particular problem under consideration. We will consider

the case where we require the phase difference to be within $\pi/2$ of the preferred phase difference.

Whenever the phase of one signal crosses a check point (which may be assumed to be zero) from negative to positive values, the phase of the other signal is recorded, resulting in a set of consecutive phase values $\{\phi_i\}_{i=1}^{N}$. Since the phase of one signal is zero, this is actually the phase difference between two oscillators measured once per cycle of oscillations. Now consider (ϕ_i, ϕ_{i+1}) space. The predominantly synchronous dynamics will appear as a cluster of points on the diagonal $\phi_{i+1} = \phi_i$ (note that this phase space is actually a torus). For the uniformity of analysis, all values of the phases may be shifted in such a way that the center of the cluster lies at the center of the first quadrant. The phase space is then partitioned into four equally spaced regions. Figure 16.1 shows a diagram for this first-return map. The first region is considered to be a synchronous state while other regions (II, III, and IV) are considered to be desynchronized states. If the phase difference is required to be not within $\pi/2$ of the preferred phase difference, but within different tolerance limit, different partition will be required.

One can define the transition rates $r_{1,2,3,4}$ for transitions between four regions of the phase space as a ratio of the number of points leaving a region to the total number of points in that region (see [3, 21, 30]). For example, r_1 is the number of points leaving the region I for the region II divided by the total number of points in the region I. One can also define the duration of desynchronization events to explore how long do the desynchronization events last. Here, the duration
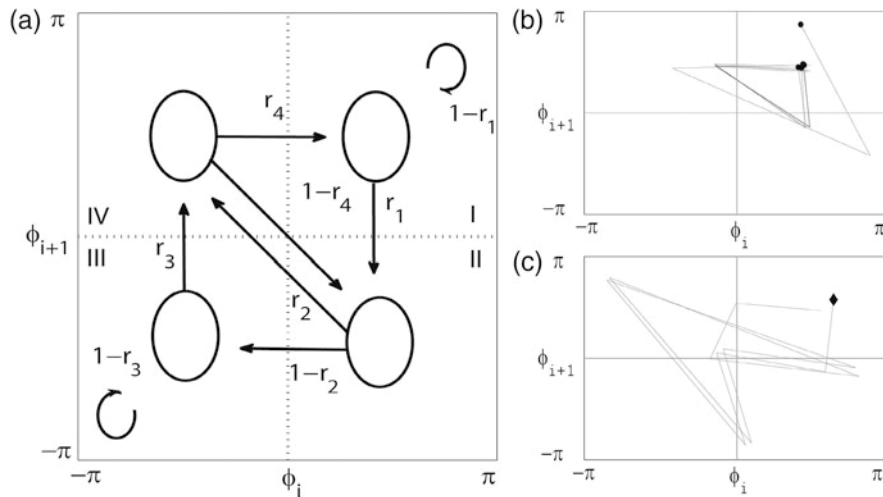
Fig. 16.1 (**a**) Diagram of the phase space of (ϕ_i, ϕ_{i+1}) first-return map. The *arrows* indicate all possible transitions from one region to another and $r_{1,2,3,4}$ indicate the corresponding transition rates. The synchronized state is placed at the center of the region I and three other regions are desynchronized states. (**b**) presents an example of dynamics with numerous short desynchronizations. (**c**) presents an example of dynamics with a very long desynchronization event

of desynchronization events is defined as the number of steps that the system spends away from the region I minus one. For example, the shortest duration of a desynchronization event corresponds to the shortest path II → IV → I (Fig. 16.1b). This corresponds to the length of one cycle of desynchronization events. Length of two cycles corresponds to the path II → III → IV → I and longer lengths of desynchronization events will have many different paths.

16.2.3 An Analysis of a Simple Model System

To illustrate some of the ideas discussed above, following [3] we will consider an example of a very simple coupled system: two coupled skewed tent maps. While this example may be ill-suited to study phase synchronization [28], it helps to illustrate the major ideas in a very simple setting. Consider a skew tent map

$$
f(a, x) = \begin{cases} \frac{x}{a}, & \text{if } 0 \le x \le a, \\ \frac{1-x}{1-a}, & \text{if } a < x \le 1, \end{cases} \tag{16.5}
$$

where $0 < a < 1$. Two such maps, described by variables x and y, are coupled in the following way:

$$
\begin{aligned}
x(t+1) &= (1-\varepsilon)f(a, x(t)) + \varepsilon f(a, y(t)), \\
y(t+1) &= \varepsilon f(a, x(t)) + (1-\varepsilon)f(a, y(t)),
\end{aligned} \tag{16.6}
$$

where ε is the coupling strength. The difference of the variables of two maps $\phi(t) = y(t) - x(t)$ may serve as a proxy for the phase difference. The synchronous state is $x = y$. It becomes stable for ε larger than a critical value ε_c. Two Lyapunov exponents ($\lambda(a)$ and $\lambda_\perp(a, \varepsilon)$) can be computed analytically [23] and are not changed if a is changed into $(1 - a)$, i.e. they are symmetrical about $a = 1/2$.

Therefore, two different pairs of maps with symmetrical values of a have the same values of Lyapunov exponents (in particular, the same value of $\lambda_\perp(a, \varepsilon)$, which characterizes the stability of the synchronous state). Thus they have the same expansive/contractive properties on the average. But the two systems are different. In one case, the map is strongly expansive in a small area of the phase space, while in the other case the map is less expansive, but the corresponding area is larger. The properties of the desynchronized dynamics are different between the two systems in the intermittently synchronous dynamics (the coupling value ε is less than ε_c) [3]. The transition rates r_i and the distributions of desynchronization events durations are markedly different between the dynamics of $a = 0.1$ and $a = 0.9$ (Fig. 16.2). This example shows that there may be different temporal patterns of synchronized and desynchronized dynamics in the coupled systems, which have the same stability properties of synchronized state.
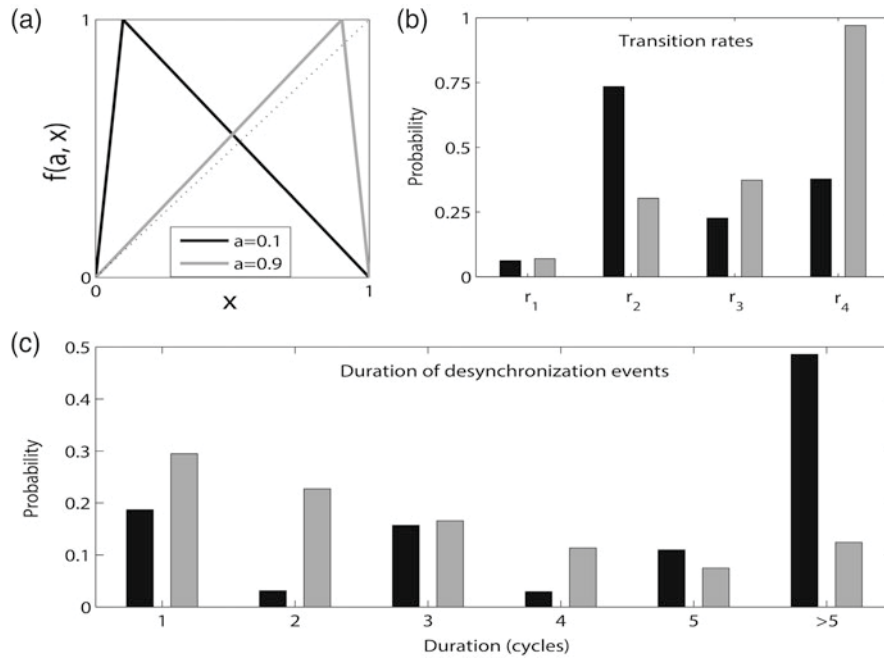
Fig. 16.2 (**a**) Two kinds of skew tent maps, with $a = 0.1$ (*black*) and $a = 0.9$ (*gray*). The coupled black maps and gray maps have identical Lyapunov exponents but different expansive/contractive properties in different areas of the phase space. (**b**) transition rates and (**c**) distribution of durations of desynchronization events for both types of coupled maps [30]. Here, ">5" is a sum of the relative frequencies of all desynchronizations longer than five

16.3 Applications to Experimental Neuroscience Data

In this section we will discuss the analysis of the temporal dynamics of synchronization in several different neuroscience experiments. There are different species involved (rodents and humans), different types of recorded data (spikes, local field potentials (LFP), and electroencephalogram (EEG)), different brain areas, and different brain states. What is general here is that in all these cases we are dealing with the synchronized oscillations in the neural activity of mammalian brains.

16.3.1 EEG Recordings in Healthy Human Subjects

In a recent study [4], EEGs recorded in a group of a hundred of healthy subjects were subjected to the analysis described above. EEGs were recorded from scalp electrodes and beta-band oscillations were extracted from the recordings. The data were acquired while subjects were at rest or executed a simple motor task (open
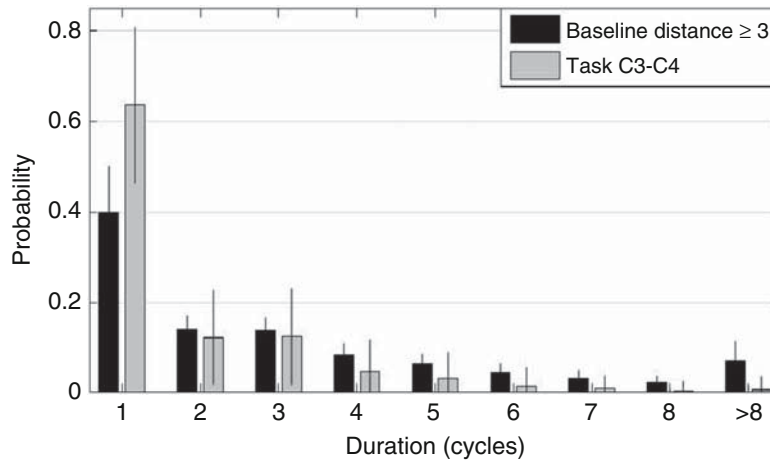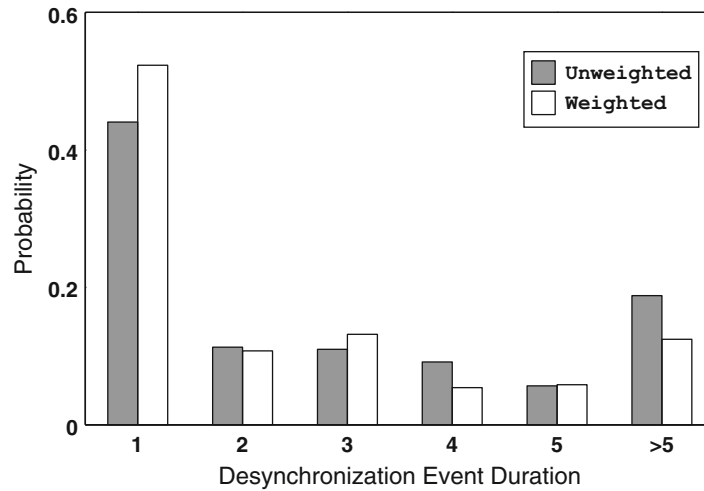
Fig. 16.3 Distribution of desynchronization durations for "Baseline distance $\geq$ 3" (*black*) and "Task C3-C4" (*gray*). Here, ">8" is a sum of the relative frequencies of all desynchronizations longer than eight. Mean $\pm$ SD is presented

and close fists). The resulting distribution of the durations of desynchronizations is presented in Fig. 16.3. In the rest state the pairs of distant electrodes, "Baseline distance $\geq$ 3", were considered to minimize a cross-talk between nearby electrodes. During a motor task, synchronization between a pair of electrodes over motor cortices was analyzed,"Task C3-C4."

The results do depend quantitatively on many factors, but qualitatively the shortest possible desynchronization is always the most frequent: for different brain rhythms, at rest and during a motor task execution, and for different arrangements of considered electrodes.

16.3.2   Spikes and Local Field Potentials in the Basal Ganglia of the Parkinson's Disease Patients

A study by Park et al. [21] analyzed the fine temporal structure of the phase-locking of neural oscillations in Parkinson's disease in the subthalamic nucleus (STN, a part of the basal ganglia, subcortical brain nuclei related to Parkinson's disease). Spiking units and LFPs were recorded with microelectrodes in a group of patients during a neurosurgery to implant deep brain stimulator electrode. LFPs are usually believed to be formed by synaptic currents and STN apparently lacks intranuclear connections. Thus, unlike cortex, spikes and LFPs in STN are formed by different processes. Episodes of beta-band activity were extracted from the data because this activity is associated with hypokinetic motor symptoms of the disease. The segments of data recordings with statistically significant synchrony strength were further selected.

Fig. 16.4 The distribution of the durations of desynchronization events in the data from patients with Parkinson's disease. The histograms of desynchronization event durations were computed in two ways, unweighted (*gray bars*) and weighted (*white bars*) proportionally to the length of the analyzed data segments. All durations that are greater than or equal to six cycles of oscillations are pooled together in ">5" group

The distributions of the durations of desynchronization events in the resulting data are presented in Fig. 16.4. The most frequent duration of desynchronization events is the shortest one and the probability to observe a desynchronization event of a duration decreases as the event duration increases. Two different ways of computing the frequencies, unweighted and weighted proportionally to the length of the analyzed data segments, yield qualitatively the same results.

Similar results were obtained after the analysis of the recordings from a different brain nuclei, internal Globus Pallidus [27]. Modeling studies of the beta-band oscillations in parkinsonian basal ganglia reproduce short desynchronization dynamics even in very small networks [19, 20] and in more realistic, larger networks [22] reach quantitative agreement with experiments.

16.3.3 Local Field Potentials in the Cortex and Hippocampus in the Rodents Undergoing Behavioral Sensitization

Another application of the analysis of desynchronization durations to neurophysiology was done at [5]. The data were recorded from hippocampus and prefrontal cortex of rats undergoing the protocol of behavioral sensitization. This protocol is used to study drug addiction in experimental animals and aforementioned brain areas are known to be directly involved in the addiction phenomena. The animals

received an injection of amphetamine every other days for several days. The dynamics of theta-band oscillation were analyzed. In all animals (including the ones from the control group) and at every stage of the protocol short desynchronizations were observed most frequently. The distribution of the durations of desynchronization events was different for different stages of the protocol, but short desynchronizations always prevailed.

Interestingly, at the very beginning of the protocol, initial drug injection does not alter average synchrony strength of theta-band oscillation. But the distribution of desynchronization durations is changed in response to the action of the drug [5]. Eventually, after more injections, average synchrony started to exhibit differences as well. There are two (not necessarily mutually exclusive) explanations for this. The first one is that the distribution of desynchronization durations is altered independently of average synchrony strength in an experiment. The second one is that as desynchronization durations are reorganized, so the average synchrony is, but it changes so weakly that the change of average synchrony has not been detected statistically. Either way the distribution of desynchronization durations turns to be more sensitive to the changes in a neuronal system, rather than average synchrony.

16.3.4   Cardiac and Respiratory Rhythms in Healthy Subjects and Subjects with Coronary Artery Disease

Final experimental example considered here is the analysis of phase-locking between respiration and heartbeat rhythms [6]. These rhythms are not usually thought of as brain rhythms. However, respiratory rhythm is generated in the brain and cardiac rhythm is generated by the electrically active cells in the heart's sinoatrial node, which bear some similarity to neurons (both cardiac cells and neurons have active membrane channels and produce relaxational oscillations). It is interesting to mention that cardiorespiratory phase-locking is almost never 1 : 1, unlike the neurophysiological phenomena described above. In general, this is $n : m$ phase-locking, n and m are small integers, and respiration is a few times slower than heartbeat. In the study [6] the methods described in the Sect. 16.2.2 were generalized to 1 : m case. This generalization is relatively straightforward: the value of the phase of the fast rhythm is recorded when the phase of the slow rhythm is going through the checkpoint.

The results are qualitatively similar to the experiments with the neural signals described above. The distributions of durations of desynchronization events do depend on many factors such as age, coronary artery disease status, ratio of frequencies of the oscillations. However, short desynchronizations are the most prevalent (although their relative frequencies in cardiorespiratory synchronization generally tend to be a bit lower than that in the neural systems described above).

16.4 Discussion

Since neural synchronization is usually intermittent even in the rest states, the analysis of distributions of synchronization and desynchronization intervals provides some interesting information about dynamics of neural systems. Properties of the synchronization intervals in neural systems have been studied earlier in the context of study of different types of intermittencies (e.g., [14, 15, 36]). But if neural synchrony is weak (which is usually the case), substantial fraction of time is spent in the desynchronized events. The studies reviewed in this paper indicate that quite different neural systems share similar properties of desynchronization events, which implies certain universality among these systems. We will discuss some potential ramifications of these observations.

16.4.1 *Measuring Fine Temporal Structure of Synchronized Dynamics in Neuroscience*

Measuring properties of desynchronization durations opens a way for two interesting possibilities in neuroscience. The first one is the use of the desynchronization durations as a diagnostic tool. The experiments with behavioral sensitization discussed above indicate that the distribution of desynchronization durations may be altered more easily than the synchrony strength and may serve as an early predictor of less subtle changes in a neuronal circuit and its dynamics in response to the drug injection. Perhaps properties of the distribution of desynchronization durations may serve as a clinical tool to diagnose otherwise undetectable changes in the neurophysiology.

The second one is the use of the desynchronization properties to match dynamical models to the real systems. If a system spends a substantial fraction of time away from synchronous state, then properties of the desynchronized states are important in order to have an adequate model of this system. One possibility is to use the transition rates, that describe the transitions between different parts of the phase space, and to develop a model in such a way that these transition rates in the model are matched to the ones derived from experimental data (like it was done in [9, 22]). This kind of matching may be important in the modeling studies of the modulation of synchrony in neuronal networks. If only the synchronized states in the model and real phase space are similar, but peripheries of the phase spaces are organized in different ways, then a modulation or stimulation during desynchronized episodes may lead to different consequences in the model and real systems (see [9]).

16.4.2   Potential Functional Significance of Observed Short Desynchronizations Dynamics in Neural Data

An interesting similarity among several experimental results described in the previous section is that in all these cases short desynchronizations dominate in the time-series (the mode of distribution of desynchronization durations equals to 1). For generic coupled oscillators, the mode of this distribution may have a higher value. This distribution is defined by the properties of a phase space periphery away from the synchronization state. Thus, for generic coupled oscillators, one may not necessarily expect much of universality here. Unlike the loss of stability of synchronization manifold, the reinjection mechanism is not necessarily universal. However, neural desynchronizations in the brain under very much different conditions and in different species appear to be universal: short desynchronizations prevail. There may be something in the very common properties of neurons (perhaps properties of kinetics of membrane channels), that universally facilitates short desynchronization dynamics.

The observed prevalence of short desynchronizations naturally brings a question of whether this property may have any significant functional advantage. We suppose that short desynchronization dynamics may make neuronal circuits more amenable to control by other brain parts or sensory inputs. Neural circuits need to be able to transiently create some synchronized states in response to external inputs. Short desynchronization dynamics means that although average synchrony is low, the system moves into vicinity of a synchronized state fairly frequently. This also implies that (for average synchrony been equal) synchronized state without inputs should be relatively strongly unstable. This arrangement may be more conducive to quick and efficient formation and break-up of transiently synchronized states in response to the external inputs to the network.

References

1. Abarbanel, H.D.I., Huerta, R., Rabinovich, M.I., Rulkov N.F., Rowat P.F., Selverston A.I.: Synchronized action of synaptically coupled chaotic model neurons. Neural Comput. **8**, 1567–1602 (1996)
2. Abarbanel, H.D.I., Rabinovich, M.I., Selverston, A., Bazhenov, M.V., Huerta, R., Sushchik, M.M., Rubchinskii, L.L.: Synchronization in neural networks. Phys. Usp. **39**, 337–362 (1996)
3. Ahn, S., Park, C., Rubchinsky, L.L.: Detecting the temporal structure of intermittent phase locking. Phys. Rev. E **84**, 016201 (2011)
4. Ahn, S., Rubchinsky, L.L.: Short desynchronization episodes prevail in synchronous dynamics of human brain rhythms. Chaos **23**, 013138 (2013)
5. Ahn, S., Rubchinsky, L.L., Lapish, C.C.: Dynamical reorganization of synchronous activity patterns in prefrontal cortex-hippocampus networks during behavioral sensitization. Cereb. Cortex **24**(10), 2556–2561 (2014)
6. Ahn, S., Solfest, J., Rubchinsky, L.L.: Fine temporal structure of cardiorespiratory synchronization. Am. J. Physiol. Heart Circ. Physiol. **306**, 755–763 (2014)

7. Bazhenov, M., Stopfer, M., Rabinovich, M., Huerta, R., Abarbanel, H.D.I., Sejnowski, T.J., Laurent, G.: Model of transient oscillatory synchronization in the locust antennal lobe. Neuron **30**, 553–567 (2001)
8. Buzsáki, G., Draguhn, A.: Neuronal oscillations in cortical networks. Science **304**, 1926–1929 (2004)
9. Dovzhenok, A., Park, C., Worth R.M., Rubchinsky L.L.: Failure of delayed feedback deep brain stimulation for intermittent pathological synchronization in Parkinson's disease. PLoS One **8**, e58264 (2013)
10. Elson, R.C., Selverston, A.I., Huerta, R., Rulkov, N.F., Rabinovich, M.I., Abarbanel, H.D.I.: Synchronous behavior of two coupled biological neurons. Phys. Rev. Lett. **81**, 5692–5695 (1998)
11. Engel, A.K., Fries, P., Singer, W.: Dynamic predictions: oscillations and synchrony in top-down processing. Nat. Rev. Neurosci. **2**, 704–716 (2001)
12. Fell, J., Axmacher, N.: The role of phase synchronization in memory processes. Nat. Rev. Neurosci. **12**, 105–118 (2011)
13. Fries, P.: Rhythms for cognition: communication through coherence. Neuron **88**(1), 220–235 (2015)
14. Gong, P., Nikolaev, A.R., Leeuwen, C.V.: Intermittent dynamics underlying the intrinsic fluctuations of the collective synchronization patterns in electrocortical activity. Phys. Rev. E **76**, 011904 (2007)
15. Hramov, A.E., Koronovskii, A.A., Midzyanovskaya, I.S., Sitnikova, E., Rijn, C.M.V.: On-off intermittency in time series of spontaneous paroxysmal activity in rats with genetic absence epilepsy. Chaos **16**, 043111 (2006)
16. Murthy, V.N., Fetz, E.E.: Oscillatory activity in sensorimotor cortex of awake monkeys: synchronization of local field potentials and relation to behavior. J. Neurophysiol. **76**(6), 3949–3967 (1996)
17. Nowotny, T., Zhigulin, V.P., Selverston, A.I., Abarbanel, H.D.I., Rabinovich, M.I.: Enhancement of synchronization in a hybrid neural circuit by spike-timing dependent plasticity. J. Neurosci. **23**(30), 9776–9785 (2003)
18. Nowotny, T., Huerta, R., Rabinovich, M.I.: Neuronal synchrony: peculiarity and generality. Chaos **18**, 037119 (2008)
19. Park, C., Rubchinsky, L.L.: Intermittent synchronization in a network of bursting neurons. Chaos **21**, 033125 (2011)
20. Park, C., Rubchinsky, L.L.: Potential mechanisms for imperfect synchronization in parkinsonian basal ganglia. PLoS One **7**(12), e51530 (2012)
21. Park, C., Worth, R.M., Rubchinsky, L.L.: Fine temporal structure of beta oscillations synchronization in subthalamic nucleus in Parkinson's disease. J. Neurophysiol. **103**, 2707–2716 (2010)
22. Park, C., Worth, R.M., Rubchinsky, L.L.: Neural dynamics in Parkinsonian brain: the boundary between synchronized and nonsynchronized dynamics. Phys. Rev. E **83**, 042901 (2011)
23. Pikovsky, A., Rosenblum, M., Kurths, J.: Synchronization: A Universal Concept in Nonlinear Sciences. Cambridge University Press, Cambridge (2001)
24. Rabinovich, M.I., Huerta R., Laurent G: Transient dynamics for neural processing. Science **321**, 48–50 (2008)
25. Rabinovich, M.I., Varona, P., Selverston, A.I., Abarbanel, H.D.I.: Dynamical principles in neuroscience. Rev. Mod. Phys. **78**, 1213–1265 (2006)
26. Rabinovich, M.I., Simmons, A.N., Varona, P.: Dynamical bridge between brain and mind. Trends Cogn. Sci. **19**, 453–461 (2015)
27. Ratnadurai-Giridharan, S., Zauber, S.E., Worth, R.M., Witt, T., Ahn, S., Rubchinsky, L.L.: Temporal patterning of neural synchrony in the basal ganglia in Parkinson's disease. Clin. Neurophysiol. **127**, 1743–1745 (2016)
28. Rosenblum, M.G., Pikovsky, A.S., Kurth, J.: Comment on phase synchronization in discrete chaotic systems. Phys. Rev. E **63**, 058201 (2001)

29. Rubchinsky, L.L., Park, C., Worth R.M.: Intermittent neural synchronization in Parkinson's disease. Nonlinear Dyn. **68**, 329–346 (2012)
30. Rubchinsky, L.L., Ahn, S., Park, C.: Dynamics of desynchronized episodes in intermittent synchronization. Front. Phys. **2**, 38 (2014)
31. Sanes, J.N., Donoghue, J.P.: Oscillations in local field potentials of the primate motor cortex during voluntary movement. Proc. Natl. Acad. Sci. U. S. A. **90**, 4470–4474 (1993)
32. Schnitzler, A., Gross, J.: Normal and pathological oscillatory communication in the brain. Nat. Rev. Neurosci. **6**, 285–296 (2005)
33. Szücs, A., Huerta, R., Rabinovich, M.I., Selverston, A.I.: Robust microcircuit synchronization by inhibitory connections. Neuron **61**, 439–453 (2009)
34. Uhlhaas, P.J., Singer, W.: Neural synchrony in brain disorders: relevance for cognitive dysfunctions and pathophysiology. Neuron **52**, 155–168 (2006)
35. Uhlhaas, P.J., Singer, W.: Abnormal neural oscillations and synchrony in schizophrenia. Nat. Rev. Neurosci. **11**, 100–113 (2010)
36. Velazquez, J.L., Khosravani, H., Lozano, A., Bardakjian, B.L., Carlen, P.L., Wennberg, R.: Type III intermittency in human partial epilepsy. Eur. J. Neurosci. **11**(7), 2571–2576 (1999)