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Self-calming of a random network of dendritic neurons

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ABSTRACT

A network of neurons with dendritic dynamics is analyzed in this paper. Two stable regimes of the complete network can coexist under continuous weak stimulation: the oscillatory synchronized regime and the quiet regime, where all neurons stop firing completely. It is shown that a single control pulse can calm a single neuron as well as the whole network, and the network stays in the quiet regime as long as the weak stimulation is turned on. It is also demonstrated that the same control technique can be effectively used to calm a random Erdös–Renyi network of dendritic neurons. Moreover, it appears that the random network of dendritic neurons can evolve into the quiet regime without applying any external pulse-based control techniques.

1. Introduction

Synchronization processes of the brain have attracted the attention of many researchers during the last decades [1–5]. In several neurological diseases such as Parkinson's disease (PS) or essential tremor (ET), brain function is impaired by pathological synchronization processes [6]. Electrical deep brain stimulation is one of the standard treatments of PS and ET [7,8] when a permanent high amplitude periodic pulse train suppresses neuronal firing [9,10]. However, the mechanism of electrical deep brain stimulation is not yet fully understood [9]; the therapeutic effect of the stimulation often decreases over time [11].

Novel pulse-based stimulation techniques are being developed which enable the selective desynchronization of pathological synchronized processes in networks of phase oscillators [4,12–17]. Though generic phase oscillators can be used to approximate the dynamics of periodically active neurons [2,18–20], the pulse-based stimulation requires to take the dendritic dynamics of neurons into account [21–23]. Individual neurons are capable of exhibiting transient dynamics when exposed to electrical stimulation [24,25]. Incorporation of the dendritic dynamics into a generic phase oscillator model helps to describe the effective inertia of a neuron when the response of the neuron's state to perturbations is not instantaneous but exhibits smooth transient dynamics [21,26]. The dendritic dynamics significantly changes the response to the stimulation of a single neuron and a network of synaptically interacting neurons [26]. Two stable regimes for a single neuron with dendritic dynamics can coexist: the oscillatory regime, where the stimulation alters only the firing rate of the neuron, and the quiet regime, where the neuron stops firing completely [26].

The opposite phenomenon and techniques for synchronization of a desynchronized network is also an important research area. Transient synchronization with a control pulse is analyzed in [27]; control of synchronization with the delayed feedback is proposed in [28]; control of synchronization in the brain by the projections is investigated in [29].

The main objective of this paper is to show that a network of dendritic neurons can be controlled not only by means of external impulses and/or external stimulation. We will demonstrate that a gradual degradation of a complete network of dendritic neurons towards a random Erdös–Renyi network can cause the effect of self-calming of the whole network. We speculate that such a computational phenomenon may represent the well-known medical technique known as "the gamma knife" used to eliminate synchronized tangles of dendritic neurons causing epileptic seizures based on the precise exposure of those tangles by a high dose of radiation therapy [30]. Such an exposure could damage some random links between neurons in the epileptic focus (well before damaging neurons). Our computational results show that the intensity of the exposure must be carefully preselected for an individual tangle of neurons because an over-exposure may damage too many interconnections between dendritic neurons. Our experiments show that such an over-exposure could then result into a synchronized firing of the random network again.

The application of artificial neural networks in modeling nonlinear biological processes has a central drawback: the lack of a precise method to choose the most appropriate network topology [31].

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There are many studies on biological neural networks, but they mostly focus on the identification of regular biological networks with clear structures, such as the neocortex [32,33], and seldom account for the seemingly irregular structure of the vast majority of biological neural networks. Our approach is also based on a homogenous network and thus it can be considered as a simple model of a basic circuit of the neocortex only.

This paper is organized as follows. The model of a single neuron with dendritic dynamics is presented in Section 2; dynamics of a single neuron under continuous excitation is analyzed in Section 3; dynamics of a complete network of neurons is discussed in Section 4. Erdös–Renyi random networks of dendritic neurons are analyzed in Section 5; desynchronization and control of random incomplete networks is investigated in Section 6, and concluding remarks are given in Section 7.

2. The model of a single neuron

We will use a model of a neuron with dendritic dynamics which is an extension of Haken second order phase oscillator model [26,34,35]. This model of a neuron consists of a single dendrite and an axon. The equation of the dynamics of the dendrite is given by

$$\dot{\psi}_i(t) = \alpha f(\phi_j(t)) - \gamma\psi(t) + \beta\xi(t), \tag{1}$$

where $\dot{\psi}$ is the dendritic current of the ith neuron; $f(\phi_j)$ is the synaptic input from the jth neuron; α is the synaptic strength; γ is the damping inherent in the dendrite; $\xi(i)$ is Gaussian white noise, statistically independent for each neuron, with zero mean and a standard deviation equal to one; and β is the noise intensity. The phase dynamics of the axon is given by [6,26,35]

$$\dot{\phi}_i(t) = c\psi_i(t) + \omega, \tag{2}$$

where ω is the driving force and currents from the dendrite ψ_i are scaled by a constant c. The described model of a dendritic neuron is equivalent to a single second order differential equation

$$\ddot{\phi}_i(t) = Af(\phi_j(t)) + \gamma(\omega - \dot{\phi}_j(t)) + c\beta\xi(t), \tag{3}$$

where $A = c\cdot\alpha$ and $\ddot{\phi}_i$ represents the dendritic currents. Note that the driving force ω is the same for all neurons in the network. Such a condition stands for an important assumption of the homogeneity of the network (all neurons are similar) [6,26]. After introducing coupling and stimulation, the governing differential equation reads

$$m\ddot{\phi}_j = \omega - \dot{\phi}_j + W_j + S_j(t,\phi_j) + \sqrt{2D}\xi(t), \tag{4}$$

where $m = 1/\gamma$ is the inertia of the oscillator; $\sqrt{2D} = c\beta/\gamma$; W_j is the dynamic coupling between the jth neuron and other neurons of the network and $S_j(t,\phi_j)$ is the stimulation of the jth neuron. The electrical stimulation of a single neuron shows a phase-dependent response [36,37], and can be modeled as a periodic function of ϕ_j:

$$S_j(t,\phi_j) = A(t)\cos(\phi_j), \tag{5}$$

where $A(t)$ is a time dependent stimulation intensity function (stimulation is off when $A(t)=0$) [13,15,26].

In general, phases of neurons are allowed to run free. The phase of an individual neuron is normalized to the interval between 0 and 1 just for the purpose of determination of its firing state. A single neuron fires whenever its phase vanishes (modulo one) [26]. The state of a neuron is defined by its firing function $\sigma(\phi_j)$:

$$\sigma(\phi_j(t)) = \begin{cases} 1 : \cos(2\pi\phi_j(t)) > c \\ 0\text{: otherwise,} \end{cases} \tag{6}$$

where c is the firing threshold; note that $\cos(2\pi\mathrm{mod}(\phi_j(t),1)) = \cos(2\pi\phi_j(t))$ due to the periodicity of the cosine function. As mentioned previously, $\sigma(\phi_j)$ plays no direct role in the calculation other than plotting results in a "spiky" manner. We follow the model introduced in [26] and assume that $c=0.975$ and $m=1$.

Networks of such nonlinear second order phase oscillators have been studied numerically in the context of phase synchronization [18], phase-frequency synchronization [39,40], emergence of spontaneous oscillations due to time delay and inertia [41,42] and optimal desynchronization [26].

3. Dynamics of a single neuron under continuous excitation

Dynamics of the phase of a single neuron in absence of coupling and noise is described by the following differential equation [26]:

$$\ddot{\phi} = \omega - \dot{\phi} + A(t)\cos(\phi), \tag{7}$$

Eq. (7) becomes a linear differential equation at $A(t)=0$ (when stimulation is off). The solution of Eq. (7) converges to

$$\phi = \omega t, \tag{8}$$

as t increases. It can be seen that the frequency of spikes becomes equal to ω when transient processes cease down.

Next, we will assume that $A(t)=a$, where a is a positive constant. Then, Eq. (7) represents a nonlinear pendulum [43] with a constant drag moment ω. The variable change $\phi = \varphi + \pi/2$ transforms Eq. (7) into the following differential equation:

$$\ddot{\varphi} + \dot{\varphi} + a\sin\varphi = \omega. \tag{9}$$

For $a < \omega$ only a stable limit cycle exists; the system has no fixed points. A saddle node bifurcation occurs at $a=\omega$ (marked as solid dots in Fig. 1A). For $a \geq \omega$ Eq. (7) has two types of fixed points: ($\phi = \cos^{-1}(-\omega/a)$; $\dot{\phi} = 0$)) which is a stable focus point and ($\phi = -\cos^{-1}(-\omega/a)$; $\dot{\phi} = 0$) which is an unstable saddle point. A homoclinic bifurcation of the saddle point occurs as the stimulation intensity reaches a critical value a_h (marked as star symbols in Fig. 1A). Two attractors (the limit cycle and the stable fixed point) coexist in the region $\omega \leq a < a_h$. Basin boundaries of these attractors are illustrated in Fig. 1B at $\omega = 2\pi$ and $a = 1.2\omega$. Black solid circles denote saddle points; empty circles—stable focus points; the thick solid line—the limit cycle; gray shaded areas denote the basin of attraction of stable focus points.

The bistability of the system (the limit cycle and the focus point) corresponds to different neuron behaviors. The focus point corresponds to a quiet regime when the neuron does not fire (the neuron's phase velocity is equal to zero then). The limit cycle corresponds to an oscillatory regime when the neuron fires with slightly oscillating frequency modulated by the stimulation.

The bistability of the limit cycle and the stable fixed point enables the development of effective attractor control strategies based on a short external impulse. Appropriately tuned parameters of the impulse can effectively quench the firing of the neuron. Initially the system oscillates in the limit cycle before the impulse (Fig. 2A); the neuron fires at a slightly varying rate (Fig. 2B). The system is kicked into the basin of attraction of the stable fixed point after the impulse is applied (the magnitude of the impulse is -40π; the duration of the impulse is 0.02). The neuron fires several times before the system settles on the fixed point and stays calm as long as the continuous stimulation is not switched off.

Fig. 2 reveals some limits of the model of the dendritic neuron. At the end before the neuron falls silent it makes a spike with not so much a peak but a plateau in Fig. 2B. This is due to the slow variation of the phase of the neuron as the solution of Eq. (9)

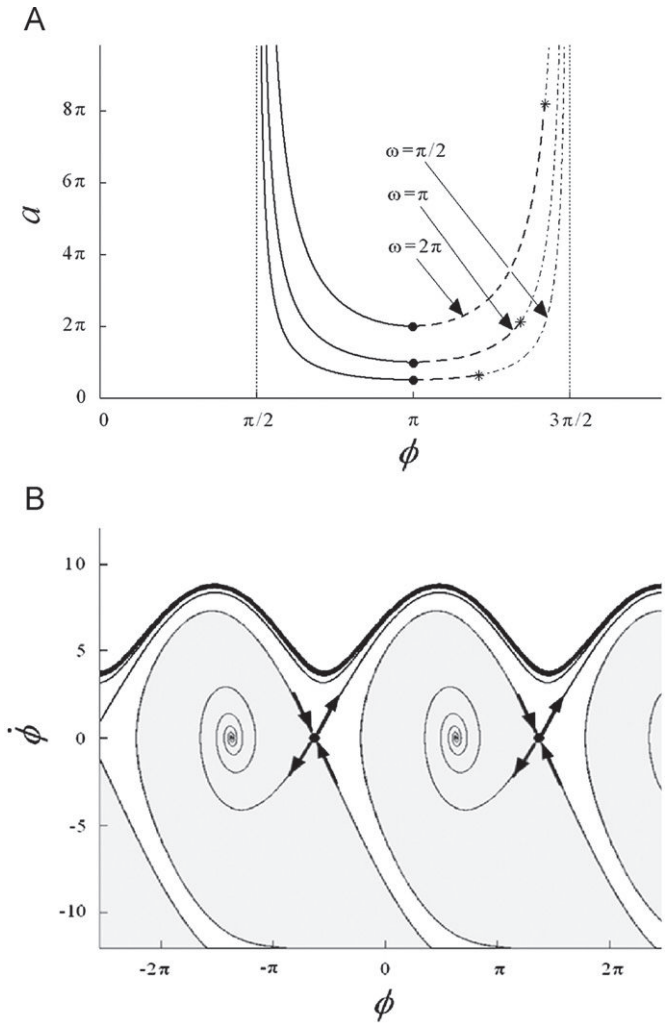

Fig. 1. (A) A bifurcation diagram of a single neuron under continuous stimulation $\dot{\phi}=\omega-\phi+a\cos(\phi)$. No fixed points exist at $a<\omega$. Saddle node bifurcations occur at $a=\omega$ (marked as solid dots; solid lines represent stable fixed points; dashed lines stand for saddle points). Homoclinic bifurcations of saddle points are marked by star symbols (a stable limit cycle and a stable fixed point coexist on thick dashed lines). (B) Basin boundaries for a single neuron under continuous stimulation $\dot{\phi}=2\pi-\phi+2.4\pi\cos(\phi)$; saddle points are marked as solid dots; the stable limit cycle is marked by a thick solid line. Basin of attraction of stable fixed points is light gray shaded.

approaches the stable fixed point. In this respect the model of the neuron with dendritic dynamics described in [26] differs from the integrate-and-fire neuron [38] because there is no phase reset after a spike. Moreover, it could occur that the phase of the neuron at the stable fixed point (modulus 1) could be higher than the value of the constant c in Eq. (6) and then the quiet state of the neuron would represent the state of continuous depolarization (that does not happen in Fig. 2). Such a "plateau" spike would be clearly implausible biologically but it would not affect the bistability of the limit cycle and the stable fixed point. As mentioned previously, Eq. (6) is used only to transform the continuous solution of Eq. (4) into spikes representing the firing of the neuron. In any case the neuron does not generate spikes if the solution of Eq. (4) remains in the vicinity of the stable fixed point. Moreover, the state of a neuron in the network is determined not by the fact that other neurons connected to it are switched "on" or "off", but by values of their continuous phases (a detailed discussion of the model of a network of neurons in given in the next section).

Note that the value of the amplitude of the continuous stimulation at which the homoclinic bifurcation occurs at $\omega=2\pi$ is $a_h=25.74$ (Fig. 1A). In other words, one has to use the amplitude of continuous stimulation $a>25.74$ in order to secure the quenching of the neuron for all possible initial conditions of the system. In fact, all stimulation amplitudes used in [26] are higher than a_h. Our computation setup exploits much lower amplitudes of stimulation. The dendritic neuron can be successfully controlled in the region of bistability by a single control pulse (one must select an appropriate phase of the impulse of course). Such impulse based control strategy for a complete network of dendritic neurons is investigated in the next section.

4. Dynamics of a complete network of dendritic neurons

It is well known that networks of coupled one-dimensional oscillators can be used to model networks of synaptically interacting neurons [2,4,20]. Introduction of the dendritic dynamics into the model of neurons significantly alters the response of the neuron to the stimulation. A bistability of the response of a single neuron can be observed under medium stimulation amplitudes [26]. Moreover, it is shown in [26] that desynchronization techniques based on multiple pulses can be adapted to networks of coupled neurons with dendritic dynamics.

The dynamics of a network of globally coupled neurons with dendritic dynamics is described by Eq. (4); the global coupling is implemented as a 2π periodic function:

$$W_j=\frac{K}{N}\sum_{k=1}^{n}\sin(\phi_k-\phi_j), \tag{10}$$

where coefficient K represents the mean-field coupling strength.

Without stimulation, synchronization of the neurons will occur if the coupling strength exceeds a critical value [39]. Then, the neurons form a synchronized cluster and the network behaves as a single oscillator. We follow the model developed in [26] and assume $K=8\pi$. We investigate a network of $N=100$ globally coupled neurons with dendritic dynamics. In order to visualize the dynamics of the system we use the mean phase velocity $\dot{\Phi}(t)$ of the network defined as

$$\dot{\Phi}(t)=\frac{1}{N}\sum_{k=1}^{N}\dot{\phi}_k(t). \tag{11}$$

The mean phase velocity allows convenient visualization of the state of the network. Time intervals where $\dot{\Phi}(t)=0$ correspond to the quiet regime: all neurons do not fire then.

It is clear that different initial conditions may result into different states of the network after transient processes cease down. $2N$ initial conditions $\phi_k(0)$; $\dot{\phi}_k(0)$; $k=1,2,\ldots,N$ determine the dynamics of the network (in case of a continuous stimulation with a constant amplitude). It is assumed that all neurons are in the firing regime at the beginning of the experiment: $\dot{\phi}_k(0)=2\pi$; $k=1,2,\ldots,N$. Moreover, we assume that all phases of the neurons are also identical at the beginning of the experiment: $\phi_1(0)=\phi_2(0)=\ldots=\phi_N(0)=\phi_0$ (in that case the coupling energy for the whole network is equal to zero and the network is completely synchronized). Since the stimulation is a harmonic function of phase (Eq. (5)), it is enough to study the interval $0\le\phi_0\le2\pi$.

The network is synchronized in the oscillatory regime under a weak continuous stimulation ($a=4\pi<K$); the evolution of the mean phase velocity at different initial phases is plotted in Fig. 3A. Note that the network is also synchronized in the oscillatory regime when the amplitude of stimulation is zero.

The evolution of the network depends on the initial phase ϕ_0 under medium strength stimulation (Fig. 3B; $a=6\pi$). The network

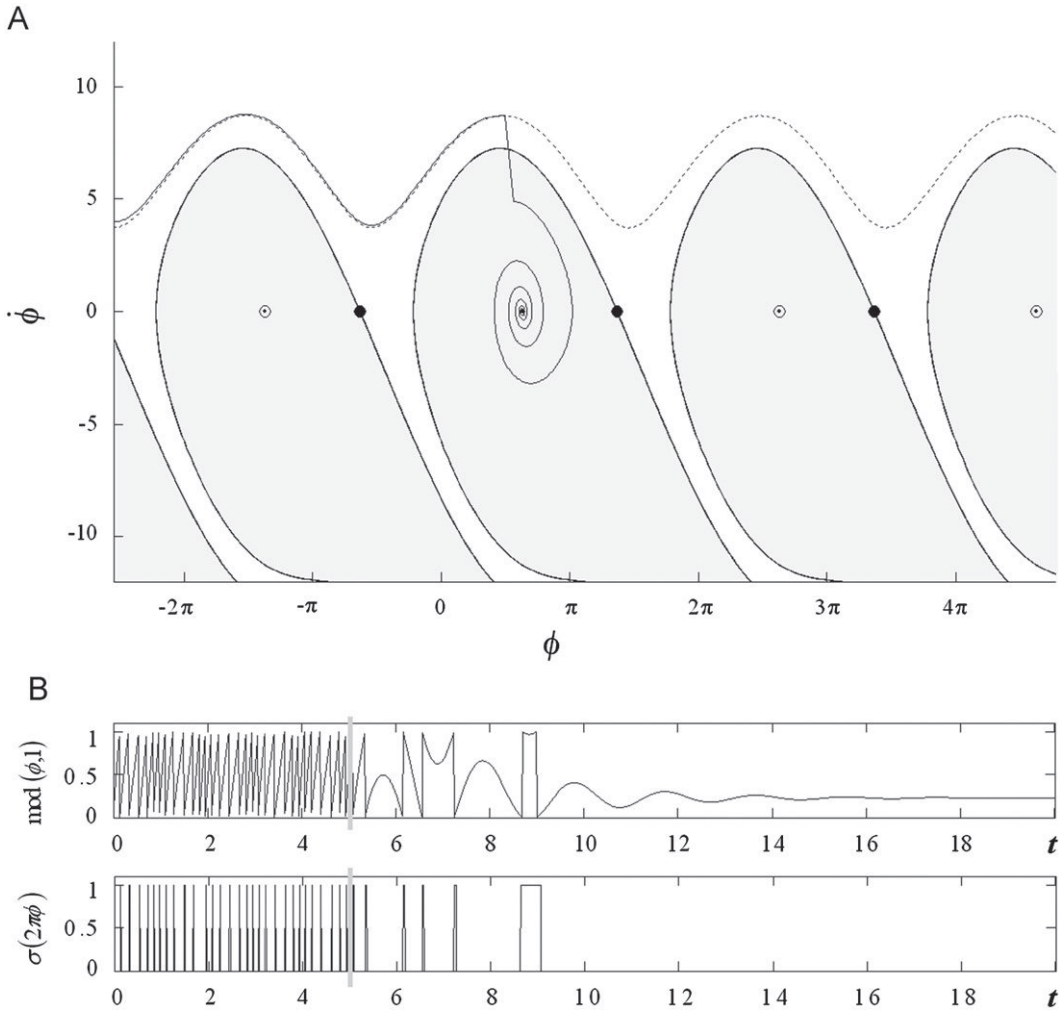

Fig. 2. A short impulse kicks the system from the stable limit cycle to the basin boundary of the stable fixed point; dynamics of the neuron is described by $\ddot{\phi} = 2\pi - \dot{\phi} + 2.4\pi\cos(\phi)$. (A) The trajectory on the phase plane $(\phi; \dot{\phi})$; the limit cycle is depicted by a dashed line; stable fixed points and saddle points are shown as empty circles and solid dots, respectively. (B) The variation of the state of the neuron in time. The phase of the neuron normalized to the interval between 0 and 1 is illustrated in the top plot; the state of the neuron is illustrated in the bottom plot. Thick gray vertical lines show the moment of the impulse in both plots.

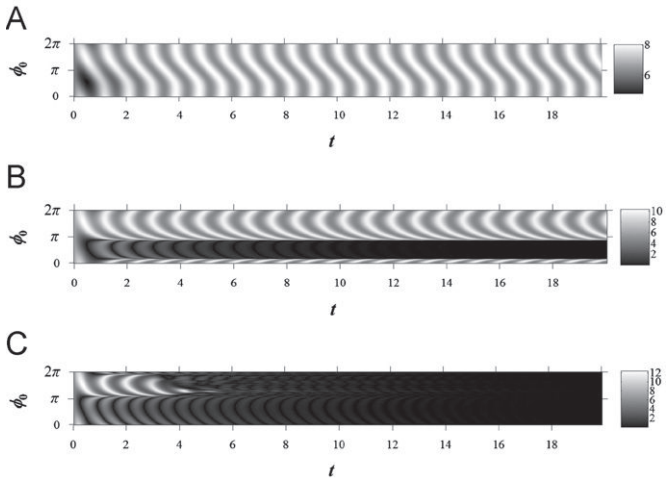

Fig. 3. The evolution of the mean phase velocity of the network in time from different initial phases ϕ_0: (A) the synchronization of the network in the oscillatory regime under weak continuous stimulation ($a=4\pi$). (B) The evolution of the network depends of the initial phase under medium continuous stimulation ($a=6\pi$). (C) The network calms down for all initial phases under strong stimulation ($a=10\pi$); the noise intensity D is 0.07; $\omega=2\pi$; $K=8\pi$.

oscillates for some initial phases but there exists an interval of initial phases where the network calms down eventually (note that $6\pi > a_h$ at $\omega=2\pi$). Strong stimulation ($a=10\pi > K$) ensures that the network evolves into the quiet regime for all initial phases (Fig. 3C).

It can be noticed that the output of continuous stimulation (even a weak one) is phase dependent [4]. Moreover, the single neuron model showed that a short strong impulse can perpetually transfer the system from the oscillatory mode to the quiet state (as long as the weak stimulation is on). The same control technique can be applied for a network of 100 globally coupled neurons. Note that such a control technique is completely different from the desynchronization technique used in [26] (where the network is exposed to a series of relatively long stimulation pulses).

We use weak stimulation ($a=5\pi$) and apply the short impulse at the moment when the phase mean velocity of the network $\dot{\Phi}(t)$ reaches its maximum in the oscillatory regime (the limit cycle is closest to the separatrix then (Fig. 2)). The magnitude of the impulse is the same as used for the control of a single neuron; the impulse is applied at the same moment to all neurons of the network. First of all, we let the network to settle into the oscillatory regime (Fig. 4). After few periods of oscillation, we

select the time when the phase mean velocity of the network is highest and apply the impulse. This time is clearly seen in Fig. 4 at the boundary between the oscillatory motion and the darker region corresponding to the convergence of the network to the stable fixed point. This boundary line (separating processes before and after the impulse) is not straight because there are phase differences between oscillatory regimes of the network which had started from different initial phases of the network ϕ_0.

The question of how robust is the proposed method to noise becomes natural. All experiments with networks of dendritic neurons are performed at the noise intensity $D=0.07$ (following the original model developed in [26]). So in general, the method is robust to noise. Nevertheless, we increase the noise intensity and test how much noise can be tolerated by the proposed control technique. A naked eye cannot see any differences between Fig. 4A (no noise) and Fig. 4B ($D=0.07$). Note that we plot the absolute mean phase velocity of the network in Fig. 4A–C in order to visualize the calm state of the network by the same color (the calm state corresponds to the black color in Fig. 4). A further increase in noise intensity can damage the concept of the method based on a single control pulse (Fig. 6C at $D=2.0$). The mean phase velocity of the network stays high and the network does not calm down after the control impulse is applied at some initial phases (Fig. 4C). Such a result is not unexpected since the control method is based on the bistability in nodes of the network what induces the bistability in the whole network. Other control techniques should be used when the stochasticity exceeds the deterministic dynamics in the model describing the network of neurons with dendritic dynamics.

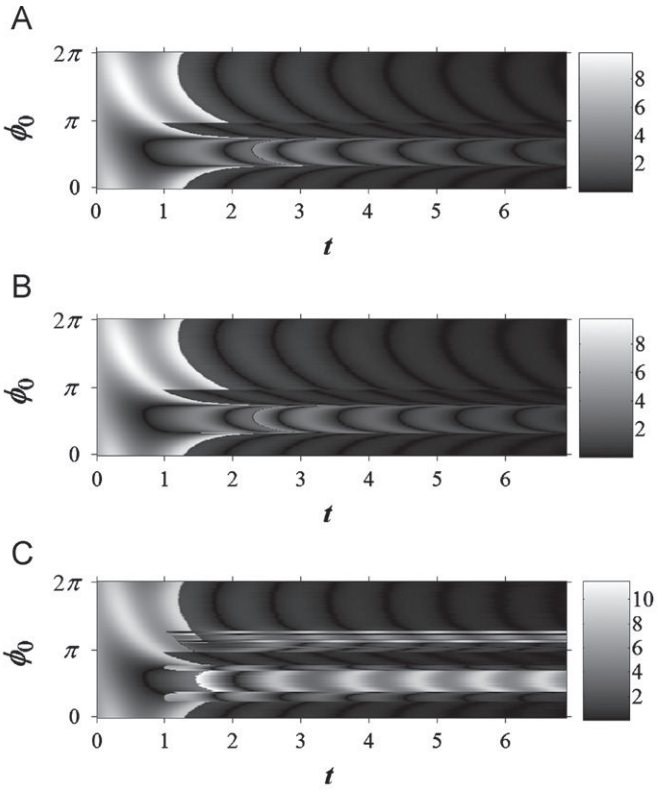

Fig. 4. Control of the network of 100 globally coupled dendritic neurons under weak stimulation ($a=5\pi$); the evolution of the absolute mean phase velocity from different initial phases ϕ_0 is shown (the black color stands for the quite regime). Initially, the network settles into the oscillatory regime. A short impulse is applied after few periods of oscillations and forces the system to converge to a quiet regime; the time of the impulse is phase dependent; $\omega=2\pi$; $K=8\pi$; the noise intensity is 0 (part A); 0.07 (part B) and 2.0 (part C).

It is important to note that such control strategy is independent from the initial phase of the network and works well at weak stimulations (which alone are incapable to calm the network for all initial phases). Moreover, a single impulse is required only and the network stays calm as long as the weak stimulation is on (which in itself can be considered as a definite advantage over techniques used in [26]).

5. Dynamics of a random network of dendritic neurons

Starting with a fully connected set on N nodes, one well-known way to generate a random network is to select two nodes at random and delete an edge between them. The networks in the resulting sequence are known as Erdös–Renyi random graphs [44]. With the continued discarding of random edges, the structural features of the network evolve. At some point, after m edges have been discarded, a structural feature can change abruptly [45,46] (the structural feature examined in [45] is the size of the largest connected component of the graph).

We construct a random Erdös–Renyi network of 100 dendritic neurons and define the parameter representing a fraction of discarded connections between neurons

$$\gamma=\frac{2m}{N(N-1)}, \tag{12}$$

where m is the number of discarded edges ($\gamma=0$ corresponds to a complete graph; $\gamma=1$ to an empty edgeless graph). We will use continuous weak stimulation at $a=5\pi$ and the coupling strength $K=8\pi$ ($\omega=2\pi$ as before); the fraction of discarded connections between neurons is set to $\gamma=0.1$.

A single impulse control method used to calm down the Erdös–Renyi network of dendritic neurons is illustrated in Fig. 5. The moment when the control impulse is applied corresponds to a local maximum of phase velocities of all neurons in the synchronized random network. A single impulse calms all neurons of the network (Fig. 5)—though the transient process after the impulse looks quite complex, all neurons settle into a stable fixed point and the network keeps completely silent (though the weak stimulation is on). The control strategy based on a single control pulse works well not only on complete but also on random networks of dendritic neurons.

6. Self-calming of a random network of dendritic neurons

The fully connected set of 100 neurons ($\gamma=0$) oscillates at the continuous weak stimulation and no neurons calm down in the process (Fig. 3A). All isolated neurons fire continuously under weak stimulation in an empty edgeless network at $\gamma=1$ (as predicted by Fig. 1A). Naturally the question arises what would happen to a random network of dendritic neurons at different fractions of discarded connections. Some neurons could possibly evolve into a silent state after transient processes in the network cease down. We introduce the parameter β which describes the ratio of quiet neurons in the network in the steady state after the transients. It is clear that $\beta=0$ at $\gamma=0$ and at $\gamma=1$.

We repeat computational experiments for 30 times for every value of γ in order to produce intervals of confidence for the ratio of quiet neurons which emerges after transient processes cease down (different configurations of random Erdös–Renyi networks may correspond to the same value of γ). The relationship among β and γ is illustrated in Fig. 6.

An interesting situation occurs when the parameter γ is in the range between 0.2 and 0.5 (Fig. 6): the whole Erdös–Renyi

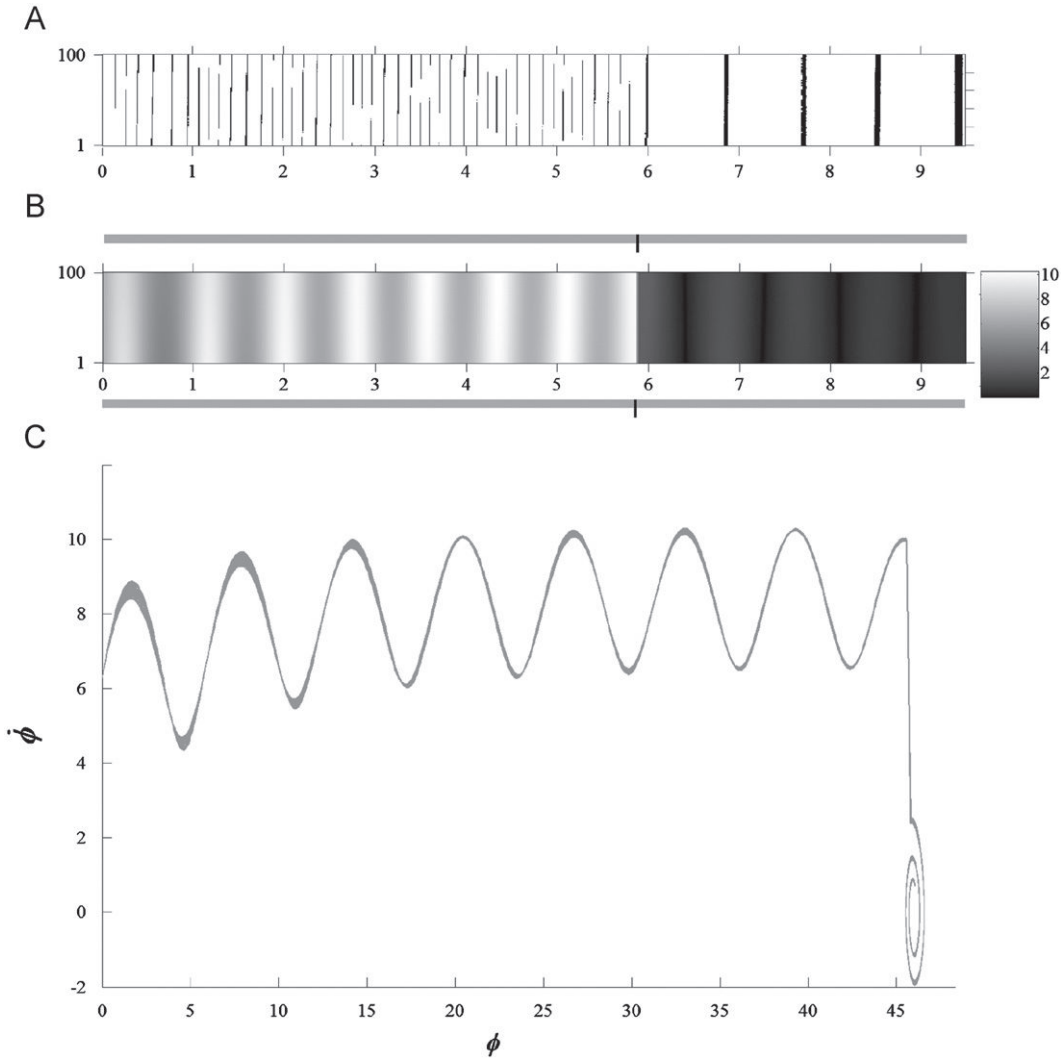

Fig. 5. Calming of the Erdös–Renyi network of 100 neurons by a single control pulse; $\gamma=0.1$; $\omega=2\pi$; $K=8\pi$; $\phi_0=0$, $\dot{\phi}_0=2\pi$; $D=0.07$; the magnitude of the impulse is -40π. The impulse is applied at $t=5.98$; the magnitude of the continuous weak stimulation is $a=5\pi$. (A) Shows the transient dynamics of 100 individual neurons (black dots correspond to time moments when neurons are active). (B) Illustrates individual phase velocities of 100 neurons. (C) Shows individual trajectories of neurons in the phase plane $(\phi;\dot{\phi})$.

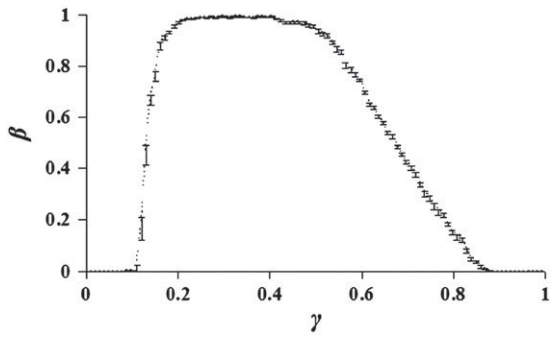

Fig. 6. A diagram illustrating the ratio of quiet neurons β in a random network of 100 neurons (the dotted line); γ is the parameter representing a fraction of discarded connections between neurons. Vertical intervals signify intervals of 95% confidence computed for 30 different realizations of random neuron networks. The dotted line stands for the arithmetic average. The amplitude of the continuous weak stimulation a is 5π; $\omega=2\pi$; $K=8\pi$; $\phi_0=0$, $\dot{\phi}_0=0$; $D=0.07$.

network calms down at the continuous weak stimulation. This is an unexpected effect.

The analysis of spatially structured neuronal networks is a rich field for experimental and theoretical studies [47]. A large variety of spatio-temporal patterns has been uncovered in such networks: localized oscillations in which two bumps oscillate in antiphase, chaotic states occurring through a cascade of period-doubling bifurcations, traveling waves, lurching waves, etc. [48]. Such networks may exhibit bistability between various patterns in numerous regions of parameter space. But the beauty of the effect of spontaneous self-calming of the random network of dendritic neurons lies in the fact that no external control techniques are required to push the network from one state to another state. Moreover, as mentioned in the Introduction, this phenomenon might be the dynamical ground for a surgery technique known as ''gamma knife''.

We plot phase velocities of individual neurons in Fig. 7 in order to visualize transient processes in Erdös–Renyi networks at different values of the parameter γ. The network oscillates at $\gamma=0.1$ and at $\gamma=0.9$ (note different grayscale levels for Fig. 7A–C), but internal self-desynchronization of neurons occurs at $\gamma=0.4$ what leads to the complete calming of the network (Fig. 7B).

So far, initial phases of all neurons in an Erdös–Renyi network have been set to $\phi_0=0$. Naturally, it would be interesting to explore the dynamics of the network for different values of initial phases (similarly to computational experiments performed with a complete network in Fig. 3A). The dependence of the ratio of quiet

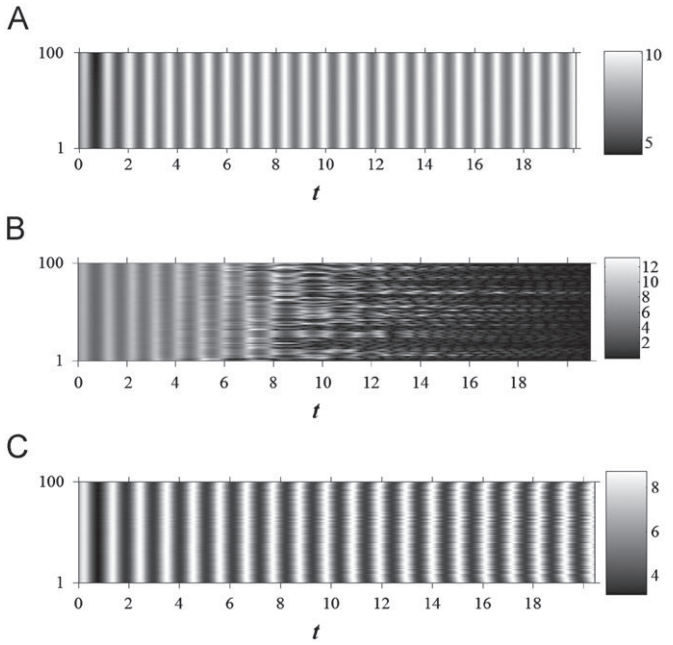

Fig. 7. Transient dynamics of Erdös–Renyi networks at $\gamma=0.1$ (A); $\gamma=0.4$ (B) and $\gamma=0.9$ (C). Phase velocities of each individual neuron (vertical axes stand for the number of a neuron) are plotted at $a=5\pi$; $\omega=2\pi$; $K=8\pi$; $\phi_0=0$, $\dot{\phi}_0=2\pi$; $D=0.07$.

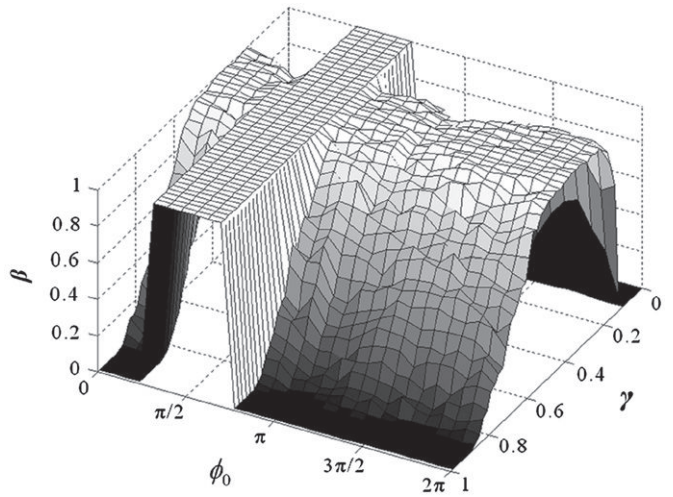

Fig. 8. The dependence of the ratio of quiet neurons β in a random network of 100 neurons (the mean of 4 different realizations of Erdös–Renyi networks) on the fraction of discarded connections between neurons γ and the initial phase ϕ_0 at $a=5\pi$; $\omega=2\pi$; $K=8\pi$; $\dot{\phi}_0=2\pi$; $D=0.07$.

neurons β in a random network of 100 neurons on the fraction of discarded connections between neurons γ and the initial phase ϕ_0 is illustrated in Fig. 8. The network evolves to a calm state when initial phases are around $\pi/2$ (Fig. 8). That corresponds well to the results illustrated in Fig. 3B (at $\gamma=0$). It must be noted that we plot the state of the network after transient processes have died down in Fig. 8, while transient evolutions are illustrated in Fig. 3A–C. This explains the apparent independence of the state of the random network from the initial condition ϕ_0 (outside the band of initial phases resulting into the quiet state). The effect of bistability observed in the complete network is present in the random network too.

The situation is completely different for such initial phases which do not result into the evolution of a complete network into a calm state. Then, an appropriate selection of γ results into a calm state of the Erdös–Renyi network, regardless of the initial phase (Fig. 8). This is an unexpected result because it appears that a network of dendritic neurons can be controlled not only by means of external impulses and/or external stimulation but by the dilution of the connectivity of the network.

So far we have used same initial conditions for all neurons of the random network. In other words, the random network was in a perfectly synchronized state at the beginning of the computational experiment. Now we investigate the evolution of a random Erdös–Renyi network when initial phases of all neurons are random. We use a random number generator to generate $\phi_j(0)$; $j=1,2,\ldots,N$ as a set of random numbers distributed evenly in the interval $[0;\ 2\pi/\omega]$. Similarly, we generate a set of random numbers with Gaussian distribution (the mean equal to 2π and the variance equal to 0.5π) and assign these random numbers as initial phase velocities of neurons.

We fix these initial conditions and perform computational experiments with different fractions of discarded connections between neurons (same initial conditions are used for different γ). Moreover, connections between neurons in the random network are discarded sequentially. In other words, we do not generate a new random Erdös–Renyi network each time we need a concrete value of γ. We start the computational experiment from a complete network and then discard random connections (every time the evolution of the random network is started from previously fixed initial conditions). Such computational setup helps to mimic the effect of the "gamma knife".

Fig. 9 shows transient evolution of the random network from random initial conditions. Fig. 9A represents the amplitude of the synchronization order parameter computed using the following equation [26]:

$$r\exp(i\psi)=\frac{1}{N}\sum_{j=1}^{N}\exp(i\phi_j), \tag{13}$$

where i is the imaginary unit; r is the order parameter and ψ is the average phase (the modulus of r is the measure of the amount of the collective behavior in the network).

Fig. 9B shows the ratio of quiet neurons β (the total number of neurons is 100 as previously). Note that random initial conditions do not desynchronize the random network—it almost immediately evolves into a synchronous firing mode for any value of γ (Fig. 9). It is interesting to note that small values of γ (almost complete

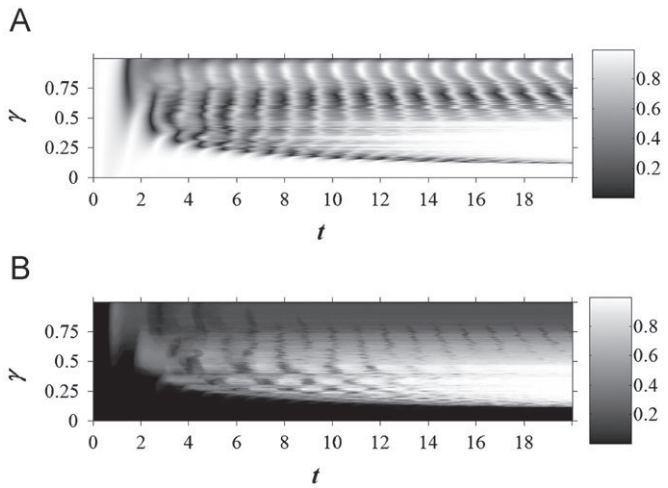

Fig. 9. The evolution of random Erdös–Renyi networks $a=5\pi$; $\omega=2\pi$; $K=8\pi$; $D=0.07$; initial phases and initial phase velocities are random. Part A shows the amplitude of the synchronization order parameter; B shows the ratio of quiet neurons.

network) ensure that the random network still oscillates in the synchronous firing mode (the amplitude of the synchronization order parameter is almost equal to 1; the ratio of quiet neurons is almost equal to 0). An almost empty network (values of γ approach 1) results into the firing of individual neurons (or isolated groups of neurons)—the amplitude of the synchronization order parameter is not constant in time; the ratio of quiet neurons is far from 1. But an intriguing situation occurs around $0.15<\gamma<0.48$ (Fig. 9). The random network quiets down; the amplitude of the synchronization order parameter becomes equal to 1 and the ratio of quiet neurons also becomes equal to 1. It must be noted that all computational experiments are performed at the noise intensity D equal to 0.07

7. Conclusions

Generic phase oscillators are widely used to model the dynamics of biological neural networks. Incorporation of the dendritic dynamics into a model of a generic phase oscillator enables the description of the effective inertia of a neuron. The dendritic dynamics significantly changes the dynamics of a network of neurons under weak continuous stimulation. Two stable regimes can coexist: the quiet regime where all neurons stop firing, and the oscillatory synchronized regime, where the stimulation only alters the firing rates of neurons.

From the physical point of view, the model of a dendritic neuron under continuous stimulation represents a nonlinear pendulum under a constant drag. Coexisting attractors of a nonlinear pendulum under a drag can be controlled by small impulses of an external force. We have shown that such control strategy can be effectively applied also for the whole network of synaptically interacting neurons and can be used to calm random networks of dendritic neurons.

So far we have been using a basic model of a dendritic neuron to simulate dynamical processes occurring in networks of neurons. The model of the neuron could be more sophisticated and a constant phase shift or higher order terms could be assumed (as implemented in [13]). A study of the stability of the results against modifications of the model of the neuron could be performed. Nevertheless, the main objective of this manuscript is to illustrate some dynamical features of the network of dendritic neurons (especially the effect of self-calming of the random network). The investigation of networks of neurons described by more sophisticated models is a definite topic of future research.

The computational effect observed for the random network of dendritic neurons can be described as a self-calming of the network when it evolves from the firing state to the quiet state. It is important to note that no other control strategies are used except the ability to control the ratio of discarded connections between neurons to the number of all possible connections in a complete network. Such an effect can be observed only when a specific fraction of all connections is discarded; the random network evolves into a synchronized firing state again if too many connections are discarded. Such a computational effect may be useful to explain the dynamical ground for specific phenomena occurring in biological networks of dendritic neurons.

It is shown that a degradation of a complete network of dendritic neurons towards a random Erdös–Renyi network can cause the effect of self-calming of the whole network. As mentioned in Section 1, we speculate that such a computational phenomenon may represent the well-known medical technique known as "the gamma knife" used to eliminate synchronized tangles of dendritic neurons causing epileptic seizures. Though our computational results do not have any clinical validation, the effect of self-calming of the whole network seems very interesting from the point of view of the selection of the exposure. Our results show that the annihilation of too many synaptic links between neurons (caused by the overexposure of the network by a high dose of radiation therapy) leads to a synchronized state of the random network again.

As mentioned previously, such a conclusion is based only on computational experiments without biological or clinical validation. Nevertheless, we use a standard model of the neuron used to simulate the behavior of biological neural networks in human brain. Thus, even if our results would have no biological meaning, the effect of self-calming of a network is a new feature and could be interesting and applicable in different areas of network science and engineering.

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