Modeling Brain States and Functions with a Chaotic Neural Network

Vladimir E. Bondarenko
Department of Physiology and Biophysics, School of Medicine and Biomedical Sciences, State University of New York at Buffalo, 124 Sherman Hall, Buffalo, NY 14214-3078, USA. E-mail: bond@sai.msu.su.

Abstract An analog neural network model with time delays was studied which demonstrated chaos similar to human and animal EEGs. Measures of chaos and correlation were used to investigate chaotic neural network behavior under the different external conditions and internal structural changes. External sinusoidal and pulsed periodic forces were used for chaos control. They produced transitions, "chaos-order" and "chaos-chaos", which were similar to those observed experimentally in the brain. A chaotic neural network state was introduced which is similar to that proposed for the brain. It is characterized by two types of memory and correlation structure of neuronal activities. Pathological non-chaotic neural network activity and activity with decreased chaos is induced by weakening interneuronal connections. Possible application to Parkinson's disease and dementia is discussed.
Keywords: Chaotic neural network, chaos control, EEG, Shannon entropy, epilepsy.

1 Introduction. Supporting Reductionism

Recent developments in neuroscience allowed consideration of consciousness as a scientific problem [28]. However, many obstacles hinder the resolution of this problem. The two main paradigms within which most scientists and philosophers now work are the holistic and reductionist approaches to the problem of consciousness [3].

The holistic approach considers consciousness a consequence of brain function. Consciousness cannot be reduced simply to the activity of neural ensembles because it is subjective and exists in the brains of humans or higher mammals. The typical holistic definition of consciousness is given by J. R. Searle (1998): "'consciousness' refers to those states of sentience and awareness that typically begin when we wake from a dreamless sleep and continue through the day until we fall asleep again, die, go into a coma or otherwise become 'unconscious'." The holistic approach has been successfully used in the nineteenth century as demonstrated by the discovery that lesions in different areas of the brain are responsible for defects in different cognitive functions.

The reductionist approach supposes that consciousness is an objective phenomenon and can be described in terms of neural activities. At present, such reduction of conscious brain states to the activities of neural circuits and ensembles is still far

from resolution. However, it successfully challenges the holistic approach. One direction in reductionism is artificial neural networks that allow simulation of brain functions such as object recognition and memory [18]. More comprehensive neural network models explain neuronal activities of particular brain areas [15]. It was also proposed that the activity of the brain could be described in terms of computation and information processing [21].

At present time the holistic approach has become obsolete. A modification of this approach has been made by J. R. Searle [28, 29, 30], but even the modified approach has unclear interpretations of the subjectivity of consciousness. We overcome this, probably most important, issue to show advantage of reductionism.

Thesis about subjectivity of consciousness. The holistic approach considers that the consciousness is subjective, and we can only study its objective features. For example, scientifically, we cannot describe pain as a subjective sense, but we can describe indirect objective evidence of pain. We consider such an approach as incorrect. To explain our point of view, consider the childhood of any conscious adult person. Everybody who has a child knows how it is difficult to understand whether or not a child suffers pain. We can only use indirect objective evidence of pain or disease. However, talking to a child, we usually call his state as a state of pain. Finally, when child grows, he learns that his state, as described above, is the state of pain. Thus, when a child learns word "pain" he begins first from objective indirect evidence of this sense through learning about the meaning of this word with the help of adults. From a reductionist point of view, nothing is lost scientifically if we consider the state of pain as an objective state with definite indirect and objective evidence for it. The problem with the holistic approach in this case is that it does not consider how an individual learns about his own senses and that the senses are only generalized terms for individual states or states of his brain.

One more interesting recent discovery is a chaotic activity of neural circuits and ensembles in the brain on the macroscopic level as recorded by EEG [4]. This discovery is especially interesting when taken into account with the discovery of unconsciousness by Sigmund Freud at the beginning of 20th century. The paradigm of unconsciousness considers that wild chaotic forces exist in human will along with rational forces. On the other hand, the brain also can produce specific chaos which is called low-dimensional chaos. This chaos differs from random noise because it can be described by a finite, relatively small number of equations, and can produce relatively large fluctuations. So, we can suppose that the chaotic activity of the brain can produce unconscious chaotic will. Thus, the study of brain chaos can be an important key for understanding the problem of unconsciousness and consciousness.

Taking into account this situation and the rapid growth of non-linear science in the 1980s, several studies have been done concerning the origin of human and animal EEGs [4, 12, 11, 33, 13, 24, 25]. These studies showed that human and animal EEGs have relatively small numbers of degrees of freedom (finite fractal or correlation dimension) and therefore can be described by a finite number of

equations. The number of degrees of freedom in an EEG can change depending on brain state: 6–9 in the waking state, 4–5 during sleep, and 2–4 during epileptic seizures [11, 33, 24, 25]. However, there are investigations that have shown that human and animal EEGs can be considered as filtered noise [36, 1, 37].

Chaotic outputs were obtained in single neuron and neural network models [22, 14]. Quantitative characteristics of chaos in neurons and neural networks with iterative dynamics (Lyapunov exponent, fractal and information dimensions) were investigated in [2, 23].

A neural network model that produced chaotic activity similar to human and animal EEGs was studied in [5, 6]. The characteristics of chaos, such as correlation dimension ν and the largest Lyapunov exponent λ, were in agreement with the experimental values. The control of chaos in a neural network with time delay by external sinusoidal force was investigated in [7, 8]. It was found that transitions in neural network activities occur when varying the amplitude and frequency of external sinusoidal force. The chaotic neural network activity was also controlled by external pulsed periodic force [10]. The results of simulations in [5, 6, 7, 8, 10] are in qualitative agreement with the experiments on controlling chaos in the brain [17, 19, 26].

Finally, the concept of coherent neuronal ensemble, which is created by synchronous activity of individual neurons, was developed by [31, 32]. This concept establishes a link between the synchronous activity of neural ensemble, brain states and neural correlates of consciousness. The ensemble consists of a temporary association of neurons into functionally coherent assemblies that as a whole represent a particular perceptual object. In this neuronal representation strategy, at different times, each neuron participates in different ensembles, because a particular feature can be part of many different perceptual objects. Additionally, these representations are very flexible and allow rapid changes of neural ensemble structure depending on external input and changes in environment. So, the content of input signals is represented not only by individual neurons, but also by dynamically associated ensembles. The experiments show a correlation between the occurence of response synchronization and brain states [32].

In this paper we investigate the chaotic neural network which produces chaos similar to human or animal EEGs [5, 6] at different network parameters. For investigation of changes in neural network activities, we used measures of chaos and interneuronal correlation, such as correlation dimension ν, largest Lyapunov exponent λ, Shannon entropy S_{Sh}, renormalized Shannon entropy S_r, and Pearson's correlation coefficient r. Simultaneous control of several characteristics of both neural inputs and outputs allows reliable detection of changes in neural network activities. The role of time delay is studied for the production of different types of activities and for control purposes. Irregular transitions, "chaos–order" and "chaos–chaos", with different characteristics of neuronal soma potential time series, are found under the external action of sinusoidal and pulsed periodic forces. We also introduced a

chaotic neural network state which is analogus to the brain state. It is characterized by two types of memory (memory I and memory II) and by correlation structure of neuronal activities. Memory I is formed by the pattern of the constant parts of soma potentials. Memory II is created by the pattern of oscillation amplitudes. Stimulus-evoked activity of the chaotic neural network is investigated when external pulsed periodic force with different amplitudes is applied. Synchronization and epilepsy-like phenomena are observed in this case. Epilepsy-like activity can be achieved by increased chaotic neural network excitability. Substantial changes in chaos and both types of memory are found under weakening interneuronal connections. Possible application of our model for Parkinson's disease and dementia is discussed.

2 Neural Network Model and Method of Analysis

The neural network model is described by the set of differential equations:

$$\dot{u}_i(t) = -u_i(t) - \sum_{j=1}^{M} a_{ij} f(u_j(t-\tau_j)) + e_i(t), \qquad i,j = 1,2,...,M, \tag{1}$$

where $u_i(t)$ is the input signal of the ith neuron, M is the number of neurons, a_{ij} are the coupling coefficients between the neurons, τ_j is the time delay of the jth neuron output, $f(x) = c \cdot \tanh(x-p)$, $\mathbf{e}(t)$ is the external force, p is the threshold of the nonlinear function $f(x)$. According to [18], u_i is the mean soma potential of a neuron from the total effect of its excitatory and inhibitory inputs. They form the EEG-like electrical activity of the neural network. Output signal of the neuron $f(u_i)$ is considered as the short-term average of its firing rate. The coupling coefficients are produced by random numerical generator in the interval from -2.0 to $+2.0$. All $M = 10$ neurons are organized in a single layer, with the connections "all-with-all".

Eqs. 1 are solved by the fourth-order Runge–Kutta method with the time step $h = 0.01$. The frequency spectra are calculated using the digital fast Fourier transform from the time series of $N = 16384$ points. The correlation dimension ν [16] is calculated, using $N = 16384$ points as well. Control calculations of ν made for several time series with $N = 131072$ points showed the values of the correlation dimension close to those obtained from time series with $N = 16384$ points.

The largest Lyapunov exponent is calculated as described in [27]. It is defined as

$$\lambda = \lim_{t\to\infty} \lim_{D(0)\to 0} t^{-1} \ln[D(t)/D(0)], \tag{2}$$

where $D(t)$ and $D(0)$ are the distances between the perturbed and unperturbed trajectories at the current and the initial moments, respectively. The largest Lyapunov exponent λ is calculated from time series of $N = 131072$ points.

The Shannon entropy was calculated from equation

$$S_{Sh} = -\sum_{k=1}^{K} p_k \ln p_k, \tag{3}$$

where p_k are the probabilities of finding the trajectory in kth subinterval of the interval of the amplitude variation. Here, $K = 128$, $\Delta_{Sh} = 8.0$.

Along with S_{Sh}, the renormalized Shannon entropy S_r is used [7]. S_r does not depend on the averaged effective energy E, because for the calculation of S_r we renormalize Δ_{Sh} which is used in eq. 3:

$$\Delta_r = 4\sqrt{E}\Delta_{Sh}, \tag{4}$$

where

$$E = \frac{1}{N}\sum_{i=1}^{N}\left(\frac{u_i - \overline{u}}{A}\right)^2. \tag{5}$$

Here $\overline{u}$ is the constant part of u, $A = 512$ is the normalization constant. Then renormalized Shannon entropy S_r is calculated from eq. 3, but with the new subinterval length Δ_r defined by eq. 4.

Pearson's correlation coefficient r is defined by equation

$$r = \frac{S_{ij}}{(S_{ii}S_{jj})^{1/2}}, \tag{6}$$

where $S_{ii} = \sum_{k=1}^{N}(u_{ki} - \overline{u}_i)^2$, $S_{jj} = \sum_{k=1}^{N}(u_{kj} - \overline{u}_j)^2$, and $S_{ij} = \sum_{k=1}^{N}(u_{ki} - \overline{u}_i)(u_{kj} - \overline{u}_j)$ for ith and jth neuronal inputs. Coefficient r is also calculated for neuronal outputs. In this case the input signal u in eq. 6 is replaced by output one $f(u)$. Pearson's correlation coefficient is used for evaluation of the degree of synchronization between two neuronal inputs or outputs.

For the estimation of neural network excitability, the average value of coupling coefficients is used

$$\overline{a} = \frac{1}{M^2}\sum_{i,j} a_{ij}. \tag{7}$$

In the case when the sum of excitatory connection strengths is equal to the sum of inhibitory connection strengths, $\overline{a} = 0$. By increase of $\overline{a}$ to positive values, we increase neural network excitability.

3 Simulation Results and Discussion

3.1 Role of Time Delay

Time delay plays important role in the production of different types of neural network activities. Depending on τ, we obtain both non-chaotic and chaotic input and output time series.

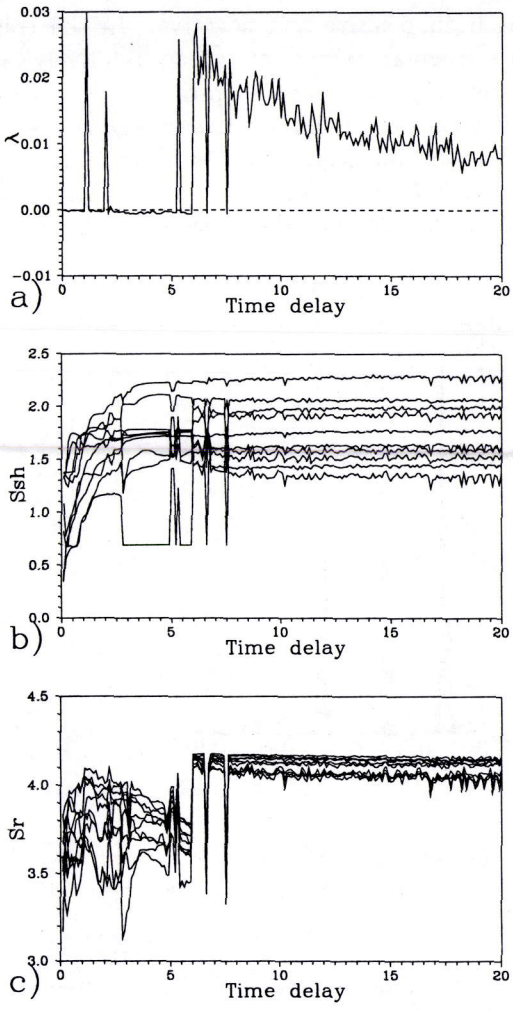

Fig. 1: Largest Lyapunov exponent λ (a), Shannon entropy S_{Sh} (b), and renormalized Shannon entropy S_r (c) as functions of time delay τ: M=10, c=3.0, $e_i(t)$=0.0, $\overline{a}$ = 0.0. Figures 1b and c show the dependences for all 10 neurons. [Reprinted from [9], Copyright (2001), with permission from Elsevier Science.]

Figure 1a shows the dependence of the largest Lyapunov exponent on τ. The calculations are made with the step $\Delta\tau = 0.1$. At small delays ($\tau < 5.0$) we have only two relatively large positive values of the largest Lyapunov exponent: $\lambda(\tau = 1.1) = 0.030$ and $\lambda(\tau = 2.0) = 0.018$. These values indicate chaotic solutions of eqs. 1. The correlation dimensions are $\nu(\tau = 1.1) = 1.7$ and $\nu(\tau = 2.0) = 1.06$, respectively. In these cases we have relatively low-dimensional chaos ($\nu < 3$). For other values of $\tau < 5.0$ we find only small magnitudes of the largest Lyapunov

exponent $|\lambda| < 0.001$, both positive and negative. In the transition range of τ ($5.0 < \tau \leq 7.5$), we see irregular changes of λ, from relatively large positive values to $\lambda \approx 0$. Further increase of the time delay τ produces only chaotic neural network activities, with positive values of the largest Lyapunov exponent (Fig. 1a). In this case, the value of correlation dimension ν varies from 5.8 to 7.5, which is close to those observed in experiments with human or animal EEGs in the waking state (from 6 to 9) [4, 12, 11, 33, 13, 24, 25]. We consider this kind of activity developed chaos.

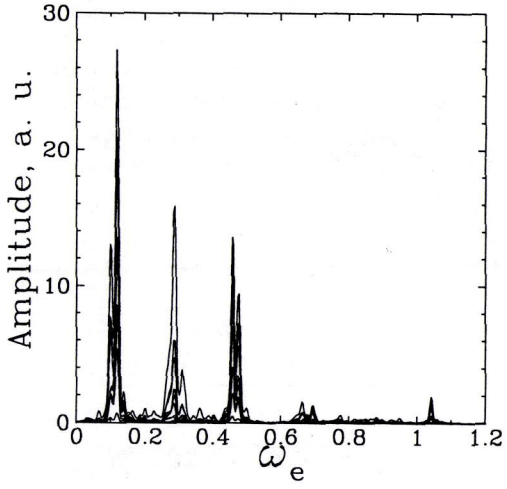

Fig. 2: Spectra of the soma potentials for all 10 neurons. $M = 10$, $c = 3.0$, $\tau = 10.0$, $\overline{a} = 0.0$, $e_i(t) = 0.0$. [Reprinted from [8], Copyright (1997), with permission from Elsevier Science.]

Figures 1b and c show the dependences of the Shannon entropy S_{Sh} and the renormalized Shannon entropy S_r on the time delay τ for the activities (soma potentials) of all 10 neurons. All quantities behave irregularly with τ up to $\tau \approx 7.5$, showing abrupt changes in their values. They produce different "patterns of entropy". The dependence $S_{Sh}(\tau)$ does not indicate abrupt changes of the degree of chaos in the neuronal activities at $\tau = 1.1$ and $\tau = 2.0$ (Fig. 1b) which are clearly seen from Fig. 1a. Only the dependence $S_r(\tau)$ increases at these time delays. In the transition range of τ ($5.0 < \tau \leq 7.5$) we see irregular abrupt changes of all entropies and their patterns (see Figs. 1b and c) when the degree of chaos changes, according to changes of the largest Lyapunov exponent (Fig. 1a). Note that only changes of S_r correlate with changes of λ: S_r increases every time as λ increases, and S_r decreases every time as λ becomes close to zero (see Figs. 1a and 1c). When $\tau > 7.5$ we observe a stable picture for $S_{Sh}(\tau)$ and $S_r(\tau)$. In this range of time delays the order of curves $S_{Sh}(\tau)$ for different neuron numbers (6, 5, 3, 7, 8, 1, 10, 9, 4, 2,

from top to bottom) in Fig. 1b does not change. However, the dependence $S_\tau(\tau)$ shows the distribution of neurons over two clusters. First (upper) cluster includes neuron numbers 2, 4, 7, 8, 9, 10. These neurons produce more chaotic activities. The second (bottom) cluster consists of neuron numbers 1, 3, 5, 6. They have less chaotic activities.

For the case of developed chaos typical spectra of all 10 neuronal soma potentials are shown in Fig. 2 ($\tau = 10.0$). The peak frequencies are in the ratios of 0.12:0.28:0.46:1.04. Similar ratios of the main rhythms of the human EEG (delta-, theta-, alpha, and beta-rhythms) are also observed in the experiments: 2.3:5.5:10.5:21.5 [20]. However, depending on the matrix of coupling coefficients a_{ij}, the spectra of neural activities can be different.

Thus we show that time delay can be used for controlling chaos in neural networks and for the production of both chaotic and non-chaotic activities. At time delays $\tau > 7.5$, we have stable neural network operation with developed chaos similar to human or animal EEGs in the waking state.

3.2 Chaos Control by External Sinusoidal and Pulsed Forces

Activity of chaotic neural network with developed chaos ($\nu > 3$) can be controlled by external action of sinusoidal force. In this case, $e_i = e \cdot \sin \omega_e t$.

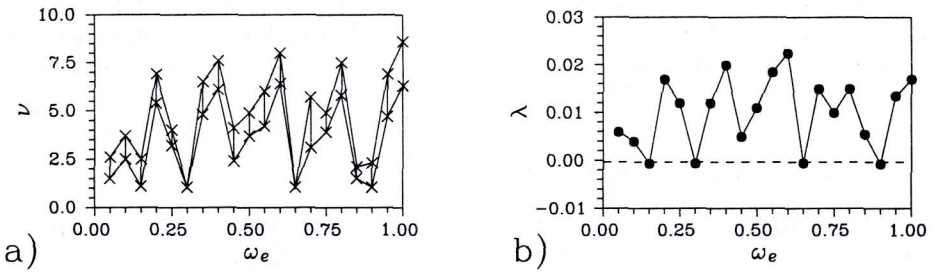

Fig. 3: Correlation dimension ν (a) and largest Lyapunov exponent λ (b) as functions of external periodic force frequency ω_e: $M = 10$, $c = 3.0$, $\tau = 10.0$, $\overline{a} = 0.0$, $e = 7.0$. For the correlation dimension the intervals in which ν varies are shown. [Reprinted from [8], Copyright (1997), with permission from Elsevier Science.]

Correlation dimension ν and the largest Lyapunov exponent λ as functions of the sinusoidal frequency ω_e are shown in Fig. 3. We observe transitions between relatively low-dimensional ($\nu \leq 3$) and high-dimensional ($\nu \sim 5-8$) chaotic regimes, with the largest Lyapunov exponent having larger values in high-dimensional cases than in low-dimensional ones (Fig. 3b). As a rule, low-dimensional chaos is observed when the frequency of external force is close to the eigenfrequency of self-excited oscillations in the neural network without external action.

Similar irregular behavior of the chaotic neural network activity is observed at the external action of pulsed periodic force by varying frequency ω_e (Fig. 4). The external force represents very short pulses, as in experiments [17, 19], with relatively large interpulse intervals. This situation is close to experimental conditions in [17, 19]. As in the case of sinusoidal force, we obtain transition "chaos–order" and "chaos–chaos" with different characteristics.

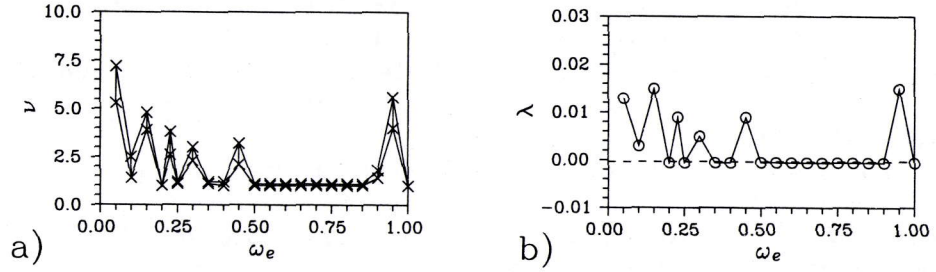

Fig. 4: Correlation dimension ν (a) and largest Lyapunov exponent λ (b) as functions of external pulsed force frequency ω_e: $M = 10$, $c = 3.0$, $\tau = 10.0$, $\overline{a} = 0.0$, $e = 2000.0$.

Figures 3 and 4 are in a qualitative agreement with Fig. 8 from [19] where chaotic regimes alternate with phase-locked regimes. Such reaction of both our model and animal EEG to the external force is typical for non-linear dynamical systems, but not noisy ones. This supports a point of view that the human or animal EEGs have non-linear origin.

3.3 Chaotic Neural Network State

As mentioned in the introduction, chaotic neural network state can be determined by two types of memories and correlation structure of inputs and outputs. Figure 5 shows dependences of constant and variable parts of input and output neuronal activities and Pearson's correlation coefficients between the 1st neuron and the other neurons for input and output signals on the neuron number. It means that the neural network under consideration possesses two types of memory which create the neural network state: memory I formed by pattern of constant parts of neuronal activities and memory II formed by oscillation amplitudes. In addition, patterns of correlation coefficients allow detection of the degree of synchronization between neurons at the different types of the neural network activities (chaotic, quasiperiodic, or periodic). Finally, we assume that our chaotic neural network creates its state which is characterized by the pattern of constant parts of neuronal activities (memory I), by the oscillation amplitudes (memory II), and by the pattern of Pearson's correlation coefficients (correlation structure).

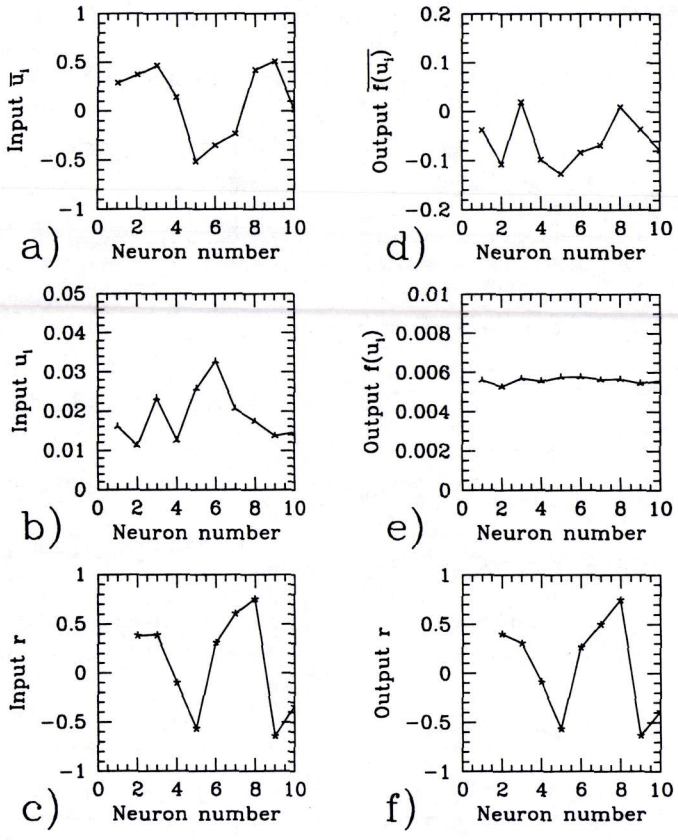

Fig. 5: Constant part of input (a) and output (d) time series, oscillation amplitudes of input (b) and output (e) signals, and Pearson's correlation coefficients between the 1st neuron and other neurons at input (c) and output (f) of the neural natwork versus neuron number. M=10, c=3.0, $\tau = 10.0$, $e_i(t)$=0.0, $\overline{a} = 0.0$.

Note that the amplitude of neural network outputs only slightly dependent on the neuron number. It means that the output information is carried by averaged firing rate of each neuron in our model. However, input information is carried by both constant and variable parts of the signal. So, each neuron in the chaotic neural network processes information by its transfer from one characteristic (soma potential) to another (averaged firing rate).

3.4 External Stimulation and Epilepsy-Like Phenomena

To simulate chaotic neural network response to external stimulation, we applied external periodic pulsed force with a frequency of $\omega_e = 0.25$ and a pulse duration of 0.1 in dimensionless units. We investigated the network response for different stimulus amplitudes $e = 300$ and $e = 3000$, which effect the behavior of the chaotic neural network in different ways. The results are presented in Fig. 6.

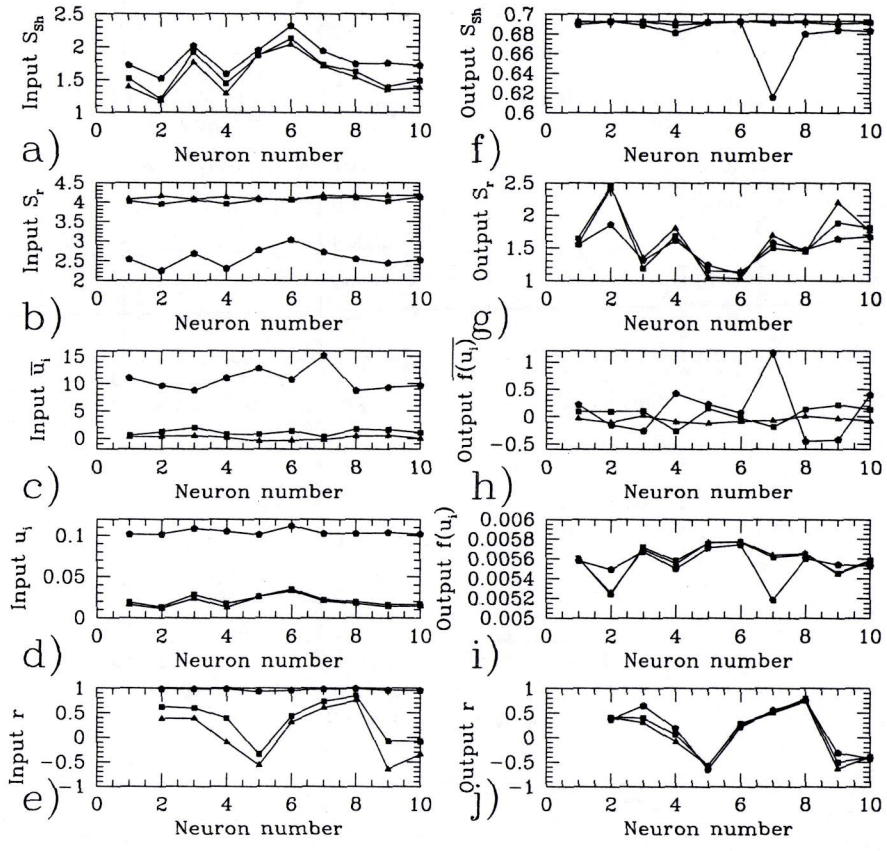

Fig. 6: Shannon entropy S_{Sh} (a, f), renormalized Shannon entropy S_r (b, g), constant part (c, h), oscillation amplitude (d, i), and Pearson's correlation coefficient between the 1st neuron and other neurons (e, j) for neuronal input (a–e) and output (f–j) time series as functions of neuron number in the neural network with pulse external periodic force. Pulse force frequency is $\omega_e = 0.25$, amplitudes e_i are 0 (triangles), 300 (squares), and 3000 (pentagons) in dimensionless units. M=10, c=3.0, $\tau = 10.0$, $\overline{a} = 0.0$.

The neural network does not show considerable changes in activity when external periodic pulsed force increases from $e = 0$ to $e = 300$. It has the same values of $\nu = 6.0$–7.4 and $\lambda = +0.013$. Only small changes in the patterns of $\overline{u_i}$, $\overline{f(u_i)}$, and small increases of synchronization detected by r for input time series are available. Increase in synchronization upon external stimulation was found in the experiments with the brain [31, 32, 35, 38].

Futher increase of e to 3000 produces larger changes in neural network activity (Fig. 6). The largest Lyapunov exponent becomes zero $\lambda = 0.0$. Renormalized Shannon entropy (pentagons in Fig. 6b) demonstrates a considerable decrease of its value for input activity of all 10 neurons in the network, showing decrease in chaos. Dramatic changes are also observed for Pearson's correlation coefficient (Fig. 6e) and oscillation amplitude (Fig. 6d). It means that the input activity of all neurons becomes fully correlated and their oscillation amplitude increases. Thus, with a relatively strong external stimulus, our neural network shows a decrease in chaos and large-amplitude synchronous oscillations. Such behavior is similar to stimulus-evoked epilepsy, for example, due to a steady flashing light or loud sound, that is observed in the experiments and in clinical practice.

Epilepsy-like phenomena can be produced in chaotic neural network at the increase in neural network excitability. When the amplitude of external forces is zero and $\overline{a} = 0.0$, the neural network produces chaotic outputs with the correlation dimensions $\nu = 6.0$–7.4 (depending on the ordinal number of the neuron) and the dimensionless largest Lyapunov exponent $\lambda = +0.013$. The characteristic time series of input of the 3rd neuron for this case are shown in Fig. 7a. When increasing the fraction of excitatory coupling coefficients by variation of the average value $\overline{a}$ from 0 to 0.8, we replace relatively high-dimensional chaotic activity with $\nu = 6.0$–7.4 (soma potential) by low-dimensional one with $\nu = 2.8$–3.1. The time series for the 3rd neuronal input in this case are shown in Fig. 7b. The largest Lyapunov exponent decreases to the value $\lambda = +0.010$. It is also accompanied by a growth of Pearson's correlation coefficient r. Further increase in $\overline{a}$ to 1.4 leads to further increase in oscillation amplitude, decrease in correlation dimension to 1, decrease of λ to 0, and increase in r to 1.0.

Such behavior of our neural network model is also similar to epilepsy due to increased brain excitability. The obtained correlation dimensions in the neural network model are close to those observed in experimental epilepsy studies 2–4 [4, 33, 13]. The value of the largest Lyapunov exponent is still positive, as was found experimentally. Epilepsy causes brain states which are different from those in normal conditions (convulsions, loss of memory, etc.). Our chaotic neural network model also shows changes of memory I, memory II, and correlation coefficients which characterize neural network state in the regime of developed chaos to another ones for the regime with decreased chaos.

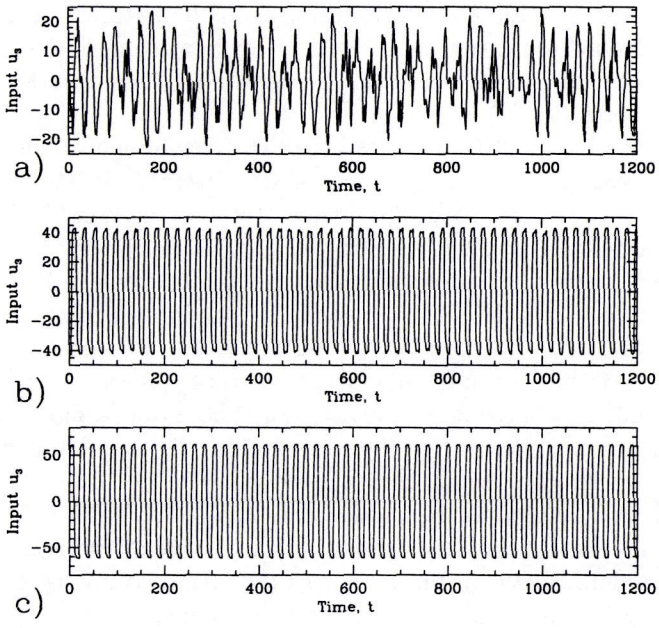

Fig. 7: Time series of the neural network inputs for the 3rd neuron, depending on the excitability $\overline{a}$ (M=10, c=3.0, τ = 10.0, $e_i(t)$=0.0): $\overline{a}$ = 0.0 (a), $\overline{a}$ = 0.8 (b), and $\overline{a}$ = 1.4 (c).

3.5 Modeling Dementia and Parkinson's Disease by Weakening Interneuron Connections

Changes of connection strengths substantially influence the chaotic neural network activity. Figure 8 shows the dependences of largest Lyapunov exponent, renormalized Shannon entropy, constant component of neuron inputs, and oscillation amplitudes as functions of the connection strength c.

At the decrease of c from 3.0 to 2.5, we do not observe substantial changes in neural network activity. However, when c decreases to 2.0, the degree of chaos changes too. The changes are detected by a small decrease of renormalized Shannon entropy (Fig. 8b). Further, even a smaller change in c from 2.0 to 1.85 leads to a stronger change in neural network activity. So, the largest Lyapunov exponent becomes zero, indicating disappearance of chaos (Fig. 8a) at c = 1.85. It is followed by considerable decrease in renormalized Shannon entropy S_r (Fig. 8b). The pattern of constant components of neuronal inputs also shows substantial changes (Fig. 8c). When c goes to 1.0, the neural network activity becomes close to periodic with

a correlation dimension $\nu \approx 1$ at all neuronal inputs. The changes of Pearson's correlation coefficients are not substantial when c decreases from 3.0 to 1.0.

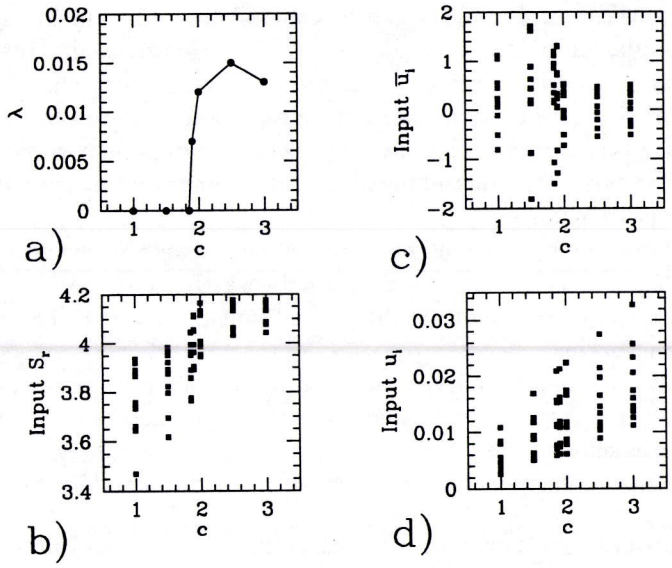

Fig. 8: Largest Lyapunov exponent λ (a), renormalized Shannon entropy S_r (b), constant component of neuron inputs $\overline{u}$ (c), and oscillation amplitudes (d) versus connection strength coefficient c: M=10, τ = 10.0, $e_i(t)$=0.0, $\overline{a}$=0.0.

Changes of neural network dynamics at the decrease of coupling strength between the neurons show similarities to changes of EEG characteristics for patients with dementia and Parkinson's disease. These patients have EEGs with decreased values of the correlation dimension (4.31 for dementia and 4.89 for Parkinson's disease versus 5.81 for control [34]). It is known that the cause of dementia and Parkinson's disease is impaired neuronal function, particularly, decreased connection strength between neurons through impaired transmitter release. Our neural network model demonstrates a decrease of correlation dimension from normal values 6.0–7.4 to 2.5–4.2 upon decreasing the coupling between neurons. The last values are close to those observed in patients with dementia ($\nu = 4.31 \pm 1.40$) and Parkinson's disease ($\nu = 4.89 \pm 0.70$) [34].

4 Conclusions

Thus, supporting the reductionist point of view on the problem of consciousness and memory in the brain, we investigated a neural network model which produces chaos similar to the human or animal EEGs. Such a neural network can be considered a

simple model for the brain, because it describes several types of the brain's properties (similar characteristics of chaos, similar response to external stimulation, similar information processing, etc.).

The chaos in the neural network can be controlled by the time delay or external action of sinusoidal or pulsed periodic forces. The chaotic neural network shows behavior similar to that observed in experiments on controlling chaos in the brain.

A concept of chaotic neural network state can be formulated by analogy to the brain state which is characterized by two types of memories and correlation structure of the network activity. The neural network state changes if any type of memory or correlation structure changes.

Relatively large external stimuli or increased neural network excitability produce epilepsy-like phenomena in a chaotic neural network. It characterized by a decrease of chaos, an increase in oscillation amplitude and synchronization. These are features of epilepsy in the brain.

Pathological non-chaotic activity appears in the chaotic neural network upon decreasing connection strengths between neurons. The decrease in correlation dimension upon weakening connection strengths from the normal values of 6.0–7.4 to 2.5–4.2 is similar to that observed in patients with dementia and Parkinson's disease.

Acknowledgements Author would like to thank Ms. Sangita Patel, Dr. Michael Morales and Dr. Randall Rasmusson (SUNY at Buffalo) for careful reading this paper and discussion. This research was supported by the USA National Science Foundation Grant No. DBI-9873173.

References

[1] Achermann, P., Hartmann, R., Gunzinger, A., Guggenbühl, W., and Borbély, A. A., *Electroencephal. Clinical Neurophysiol.*, **90**, 384–387 (1994).

[2] Aihara, K., Takabe, T., and Toyoda, M., *Phys. Lett. A*, **144**, 333–340 (1990).

[3] Albright, T. D., Jessell, T. M., Kandel, E. R., and Posner, M. I., *Neuron*, **25**, S1–S55 (2000).

[4] Babloyantz, A., and Destexhe, A., *Proc. Natl. Acad. Sci. USA*, **83**, 3513–3517 (1986).

[5] Bondarenko, V. E., *Phys. Lett. A*, **196**, 195–200 (1994).

[6] Bondarenko, V. E., *Int. J. Bifurc. Chaos*, **7**, 1133–1140 (1997a).

[7] Bondarenko, V. E., *Int. J. Bifurc. Chaos*, **7**, 1887–1895 (1997b).

[8] Bondarenko, V. E., *Phys. Lett. A*, **236**, 513–519 (1997c).

[9] Bondarenko, V. E., *Chaos, Solitons & Fractals*, **13**, 139–154 (2002).

[10] Bondarenko, V. E., and Rusev, A. V., "Chaotic neural network under external periodic pulsed force", in *Cybernetics and Systems '98*, edited by R. Trappl, Vol. 2, Austrian Society for Cybernetic Studies, Vienna, 1998, pp. 634–637.

[11] Destexhe, A., Sepulchre, J. A., and Babloyantz, A., *Phys. Lett. A,* **132**, 101–106 (1988).

[12] Dvorak, I., and Siska, J., *Phys. Lett. A,* **118**, 63–66 (1986).

[13] Frank, G. W., Lookman, T., Nerenberg, M. A. H., Essex, C., Lemieux, J., and Blume, W., *Physica D,* **46**, 427–438 (1990).

[14] Freeman, W. J., *Biol. Cybern.,* **56**, 139–150 (1987).

[15] Golomb, D., Wang, X.-J., and Rinzel, J., *J. Neurophysiol.,* **75**, 750–769 (1996).

[16] Grassberger, P., and Procaccia, I., *Phys. Rev. Lett.,* **50**, 346–349 (1983).

[17] Hayashi, H., and Ishizuka, S., *Brain Res.,* **686**, 194–206 (1995).

[18] Hopfield, J. J., *Proc. Natl. Acad. Sci. USA,* **81**, 3088–3092 (1984).

[19] Ishizuka, S., and Hayashi, H., *Brain Res.,* **723**, 46–60 (1996).

[20] Kandel, E. R., Schwartz, J. H., and Jessell, T. M., editors, *Principles of Neural Science*, 3rd edition, Appleton & Lange, Norwalk, Connecticut, 1991, p. 778.

[21] Koch, C., and Segev, I., *Nat. Neurosci.,* **3**, Supplement, 1171–1177 (2000).

[22] Kürten, K. E., and Clark, J. W., *Phys. Lett. A,* **114**, 413–418 (1986).

[23] Nakagawa, M., and Okabe, M., *J. Phys. Soc. Jpn.,* **61**, 1121–1124 (1992).

[24] Pijn, J. P., van Neerven, J., Noest, A., and Lopes da Silva, F. H., *Electroencephal. Clinical Neurophysiol.,* **79**, 371–381 (1991).

[25] Röschke, J., and Aldenhoff, J., *Biol. Cybern.,* **64**, 307–313 (1991).

[26] Schiff, S. J., Jerger, K., Duong, D. H., Chang, T., Spano, M. L., and Ditto, W. L., *Nature,* **370**, 615–620 (1994).

[27] Schuster, H. G., *Deterministic chaos*, Physik-Verlag, Weinheim, 1984.

[28] Searle, J. R., *Phil. Trans. R. Soc. Lond. B,* **353**, 1935–1942 (1998).

[29] Searle, J. R., *Phil. Trans. R. Soc. Lond. B,* **354**, 2069–2080 (1999).

[30] Searle, J. R., *Annu. Rev. Neurosci.,* **23**, 557–578 (2000).

[31] Singer, W., *Progr. Brain Res.,* **95**, 461–474 (1993).

[32] Singer, W., *Phil. Trans. R. Soc. Lond. B,* **353**, 1829–1840 (1998).

[33] Soong, A. C. K., and Stuart, C. I. J. M., *Biol. Cybern.,* **62**, 55–62 (1989).

[34] Stam, C. J., Jelles, B., Achtereekte, H. A. M., Rombouts, S. A. R. B., Slaets, J. P. J., and Keunen, R. W. M., *Electroencephal. Clinical Neurophysiol.,* **95**, 309–317 (1995).

[35] Stopfer, M., Bhagavan, S., Smith, B. H., and Laurent, G., *Nature,* **390**, 70–74 (1997).

[36] Theiler, J., Eubank, S., Longtin, A., Galdrikian, B., and Farmer, J. D., *Physica D,* **58**, 77–94 (1992).

[37] Theiler, J., and Rapp, P. E., *Electroencephal. Clinical Neurophysiol.,* **98**, 213–222 (1996).

[38] Tononi, G., and Edelman, G. M., *Science,* **282**, 1846–1851 (1998).