Neurons: A Numerical Approach

Neuronas: Un Enfoque Numérico

Sui-Nee Chow
School of Mathematics-CDSNS, Gatech
Atlanta, GA 30332-USA
Teodoro Lara (teodorolara@cantv.net)
Departamento de Física y Matemáticas, NURR-ULA
Trujillo-Venezuela

Abstract

We introduce a model for the electrical behavior of brain cells, based on a model introduced in [8]. This model basically makes analogies between electrical circuits and the way the body and synapse of brain cells work. Numerical simulation is implemented seeking for synchronization; what the numerical results show is synchronization in case of little, strong interaction (excitation), strong inhibition, some excitation, some inhibition, and mixture of these states.

Key words and phrases: cellular neural networks, Singular Perturbation, synchronization, neurons.

Resumen

Se introduce un modelo para el comportamiento eléctrico de las células cerebrales, basado en un modelo introducido en [8]. Este modelo básicamente hace analogías entre circuitos eléctricos y la manera en que trabajan el cuerpo y las sinapsis de las células cerebrales. Se implementa la simulación numérica buscando sincronización; los resultados numéricos muestran sincronización en los casos de poca, fuerte interacción (excitación), fuerte inhibición, alguna excitación, alguna inhibición, y mezclas de estos estados.

Palabras y frases clave: redes celulares neuronales, perturbación singular, sincronización, neuronas.
1 Introduction

Biological membranes play fundamental roles in many processes of life. Much of their activity is electrical, and the membrane potential, i.e., the voltage across a membrane, is one of the physical states of nerve cells that can be measured in vitro. The flow of various ions (charged chemical molecules) through membranes establishes electrical currents that cause changes in the membrane potential. These are observed to be pulses of voltages and are called action potentials. Neuron physiology describes the electrical properties of membranes. Models of nerves are based on the Nerst equation that determines membrane potential of a cell from the ion concentrations near it. See [8]. For quite some time a great deal of effort has been dedicated to the study of electrical behavior of brain cells; different models have come out since the Hodgkin-Huxley model was proposed ([5]). In the next section we will take a look to some of them, including the foregoing.

The model we study is based in one proposed in [8], and what it does is to represent each cell as two electrical circuits; one for the body cell and one for the synapse. The body cell is viewed as a voltage controlled oscillator and the synapse as a low pass filter. We set some coupling based in our model of CNN considered in [13]; and test it for arrays of $3 \times 3$, $5 \times 5$ and $10 \times 5$ cells. The results in each case are very alike. Indeed for strong interaction or strong inhibition, synchronization is observed; moreover in some cases of mixed excitation and inhibition synchronization is also observed. We include some pictures of the $3 \times 3$ and $5 \times 5$ cells showing the mentioned situation.

2 Neurons

A neuron consists of dendrites that receive signals, a cell body that synthesizes incoming signals and generates new ones, an axon that transmits new signals away from the cell body, and a synapse that transmits the signals to other cells. Neurotransmitters (chemical molecules) released at synapses in response to changes in membrane voltage communicate these changes to the environment of neuron. Attempts to describe a nerve cell’s electrical behavior have been based on electrical circuit analogies and their mathematical models. The Hodgkin-Huxley (HH) model (1952) is a major success resulting from this approach. This model was derived from experimental studies of the squid giant axon. The theory provides the analog circuit studied most in neurophysiology. This circuit is shown in Figure 1. The model is formulated for a membrane and accounts for $Na^+$ (sodium), $K^+$ (potassium), and $L^+$
Figure 1: Hodgkin-Huxley Circuit

(leakage) ion channels. The equation is

\[ C\dot{v} = g_{Na}(E_{Na} - v) + g_{K}(E_{K} - v) + g_{L}(E_{L} - v) + I \]  

(1)

where \( v \) is the membrane potential, \( I \) is input current, \( E_{Na}, E_{K}, E_{L} \) are the sodium, potassium and leakage resting potentials respectively, with \( E_{Na} = 55mV, E_{K} = -75mV \), \( C \) is the membrane capacitance, \( g_{Na}, g_{K}, g_{L} \) are the, respectively, sodium, potassium, and leakage ion conductances, and they are defined as \( g_{Na} = \frac{1}{R_{Na}}, g_{K} = \frac{1}{R_{K}}, g_{L} = \frac{1}{R_{L}} \), with \( R_{Na}, R_{K}, R_{L} \) the resistance of the membrane to the flow of ion \( Na, K, L \) respectively. There is a way in which these conductances depend on \( v \), but it involves three more differential equations. See [8] for details.

Various other models have been formulated that describe important features of the HH model and at the same time they are more tractable for mathematical analysis and numerical simulations; among others we can mention the FitzHugh-Nagumo (FHN) model and a simplification of it due to Keener ([10]). The FHN model was introduced in the late 50s it involves a Tunnel Diode (TD) and it is typical of various “flush and fill” circuits; the circuit is depicted in Figure 2. More can be said about this model but that is not the main objective of this work. And the corresponding equations, by
Figure 2: FitzHugh-Nagumo Circuit

\[
\begin{align*}
L \dot{I} &= E - v - RI, \\
C \dot{v} &= I - g(v)
\end{align*}
\]

where \( L \) is inductance, and \( g \) is N-shape function. However, tunnel diodes are obsolete, hard to work with and expensive. They have been replaced by sophisticated integrated circuits that are inexpensive, stable and reliable. J. Keeneer in [10] has developed a circuit, similar to the FHN circuit, but based on the operational amplifier (op-amp). See Figure 3.

Figure 3: Operational Amplifier
Op-amps are useful for research in a variety of technical fields who need to build simple amplifiers but do not want to design at the transistor level. Op-amps are designed to perform a basic function, which is to give a reliable output voltage that depends solely on the difference of the input voltages. Integrated circuits technology allows the construction of many amplifier circuits on a single composite chip of semiconductor material. See [12] for instance for details.

The model described in [10] is given by

\[
\dot{I} = \beta V - I - V_0, \quad \epsilon \dot{V} = I_0 - I - G(V),
\]

where

\[
\dot{I} = \frac{dI}{d\tau}, \quad \tau = \frac{t}{C_2R_4}, \quad V = \frac{(R_1 + R_4)v}{V_+R_4}, \quad V_0 = \frac{R_1V_0}{R_3V_+},
\]

\[
I = \frac{R_1}{R_4V_+}((R_4 - R_3)I_2 - v_0), \quad I_0 = \frac{R_1i_0}{V_+},
\]

\[
\epsilon = \frac{R_1C_1}{(R_1 + R_4)C_2}, \quad \beta = \frac{R_4}{R_3} \frac{R_3 - R_1}{R_4 + R_1},
\]

and for \( s = \frac{R_4 - R_1}{R_4 + R_3} \), the function \( G(V) \) is defined as

\[
-G(V) = \begin{cases} 
1 - V, & \text{if } V \geq \frac{1}{s+1} \\
sv, & \text{if } -\frac{1}{s+1} \leq V \leq \frac{1}{s+1} \\
-1 - V, & \text{if } -\frac{1}{s+1} \geq V.
\end{cases}
\]

In Figure 4 there is a representation of such a circuit. More details can be seen in [10]. Now we spend a bit of time talking about Voltage Controlled Oscillators since they will be used later in modeling neurons.

**Definition 2.1.** Voltage Controlled Oscillators, (VCOs), are oscillators whose frequency is modulated or controlled by an input voltage. Current is ignored in VCOs and the model is given in terms of the input and output voltages alone.

The situation is as follows \( V_{in} \rightarrow [VCO] \rightarrow V(x(t)) \); where \( V_{in} \) and \( V \) are input and output voltages respectively, they are related in a somewhat complicated way. The form of \( V \) for a VCO might be a step function, a triangular or sinusoidal wave; in general \( V \) is taken to be continuously differentiable and periodic, the phase of the signal, \( x(t) \), is unknown. When \( V_{in} \) is in operating range of the VCO, the output phase is related to the controlling voltage by

\[
\dot{x} = \omega + \sigma V_{in},
\]
\(\omega\) is called the VCO’s center frequency and \(\sigma\) is called the sensitivity, in general \(\sigma = 1\) for suitable scaling of the voltages. Now we visualize the cell body as being a VCO and then an analog circuit for a synapse is given; combination of this produces a basic neuron model.

Neurons operate in either a repetitive firing mode or an excitable mode which is a similar behavior of a VCO. The VCO feedback loop is modeled in terms of the phase \(x_V\); which is determined by the equation \(\dot{x}_V = e_0 + \omega_0\) where \(e_0\) is the acquisition voltage and \(\omega_0\) is the VCO’s center frequency. We view \(V\) as being a cell’s membrane potential having the form described above.

Now we find a circuit analog to the synapse, that will be called a SYN circuit ([10]). In order to do so, we introduce the notion of filter; in general a filter may be considered to be a signal processing device which operates on an input signal to produce an output signal bearing a prescribed relationship to the input signal; there are different type of filters, we shall mention only the low pass filter. The low pass filter is the one which passes the package of wave energy from zero frequency up to a determined cut off frequency and rejects all energy beyond that limit. The output \(W\) voltage of a low pass filter is determined by solving the equation

\[
RC\dot{W} + W = S(V); \tag{5}
\]
Neurons: A Numerical Approach

with \( S(V) = \max(V, 0) \).

An action potential generated in the cell body passes down an axon that terminates in a synaptic bouton; neurotransmitter is then released to interact with the postsynaptic membrane. Neurotransmitter kinetics are analogous to a low-pass filter. There is a threshold effect also where an action potential must reach a certain strength before it can cause release of neurotransmitter. Therefore, the first device in a SYN circuit is a diode. The diode takes the positive part of the action potential as causing neurotransmitter release, so we consider 0 as being the transmitter release threshold. In the above equation, \( S(V) = V_+ \equiv \max(V, 0) \equiv d(V) \) and equation (5) now becomes

\[
RC \dot{W} + W = d(V).
\]

If we ignore chemical kinetics, \( RC = 0 \), then \( W = d(V) \). The neurotransmitter can be excitatory, adding to the postsynaptic potential, or inhibitory. We assume this is modeled by adding the effect of neurotransmitter to the postsynaptic potential and then trimming the sum to fit the physiological limit of the postsynaptic membrane. This is accomplished by combining a voltage adder (+) with a linear amplifier. The amplifier output is described by its characteristic function that we will denote as \( P \). In general, \( P \) can be any bounded, continuously differentiable monotone increasing function. In some cases it is convenient to take \( P(u) = \tanh(u) \) and in neighborhood of origin \( P(u) = u - u^3/3 \), \( d(V(y)) \) is the super-threshold part of input voltages. Combining the above two equations the following system appears:

\[
\dot{X} = \omega_0 + P[V(X) + W],
RC \dot{W} = -W + d(V(y));
\]

(6)

where \( y \) represents phase of input voltages from sites outside the cell. The above equation is called voltage-controlled oscillator neuron or VCON and equation (5) synapse analog or SYN. If we assume \( RC = 0 \) then system (6) reduces to only one equation

\[
\dot{X} = \omega_0 + P[V(X) + d(V(y))],
\]

(7)

and for \( RC \neq 0 \), and by assuming \( BC \) is small, say, \( 0 < BC \ll 1 \), the system becomes

\[
\dot{X} = \omega_0 + P[V(X) + W],
\epsilon \dot{W} = -W + d(V(y)); \quad \epsilon = RC.
\]

This equation (\( RC = 0 \)) is described by a circuit corresponding to a first order Phase-Locked Loop ([15]) or PLL, but to do this we need to introduce
some concepts of electrical circuits. The first of them is Phase Detector (PD); which is a form comparator providing DC output signal proportional to the difference in phase between two input signals. Although a linear response would be ideal, in practice the response of phase detectors is nonlinear and periodic over a limited phase range.

![Phase-Locked Loop Diagram]

A PLL is basically an oscillator whose frequency is locked onto some frequency component on an input signal $V_i$. This is done with feedback control loop (Figure 5). What it does is synchronize the frequency of an output signal generated by an oscillator with frequency of a reference signal by means of the phase difference of the two signals. Sometimes between the PD and the VCO a low-pass filter is located. In case of no filter it is called a first order PLL. The frequency of this component in $V_i$ is $\omega_i$ (in rad/sec) and its phase $\theta_i$. The oscillator signal $V_0$ has frequency $\omega_0$ and phase $\theta_0$. The phase detector (PD) compares $\theta_0$ with $\theta_i$, and it develops a voltage $V_d$ proportional to the phase difference. This voltage is applied as a control voltage $V_c$ to the VCO to adjust the oscillator frequency $\omega_0$. Through negative feedback, the PLL causes $\omega_0 = \omega_i$; and the phase error is kept to some small value. Thus, the phase error and the frequency of the oscillator are “locked” to the phase and the input signal. PLLs are used primarily in communication applications.

Our task now is to give a coupled system for a 2D array of cells by using the foregoing model.
3 Coupling

In this section we shall give expression for coupling of \( M \times N \) neurons distributed in a 2D array.

**Definition 3.1.** A neighborhood of cell \( C(ij) \) in a CNN is defined as

\[
N^{ij} = \{ C(i_1j_1) : \text{Max}\{|i - i_1|,|j - j_1|\} \leq 1; 1 \leq i_1 \leq M, 1 \leq j_1 \leq N. \}
\]

This is the same neighborhood considered in [8], [13], [2], and [3] as well; also we impose periodic boundary conditions exactly in the same way as we did in [13].

The connections between cells are described by the coefficients of an \( n \times n \)-matrix \( A \) and its components are given in terms of coefficients of a cloning template \( \tilde{A} \) (by means of the periodic boundary conditions) that will be specified shortly; \( A \) will be taken to be symmetric since if \( a_{ij} \) is the \( ij \) coefficient of \( \tilde{A} \), it represents the strength of input from neuron \( j \) to neuron \( i \), which also can be assumed as same strength from neuron \( i \) to neuron \( j \). The connections from external stimuli are given by coefficients of an \( n \times n \)-matrix \( B \) and they are given by cloning template \( \tilde{B} \); if \( \tilde{B} = (b_{ij}) \) then \( b_{ij} < 0 \) means inhibitory stimulus, \( b_{ij} > 0 \) means excitatory stimulus and \( b_{ij} = 0 \) is not stimulus. The cloning templates \( \tilde{A} \) and \( \tilde{B} \) are 3 \( \times \) 3-matrices. In general they are given as (see [13])

\[
\tilde{A} = \begin{pmatrix} a & b & c \\ b & d & e \\ c & e & f \end{pmatrix}, \quad \tilde{B} = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & b_{22} & b_{23} \\ b_{31} & b_{32} & b_{33} \end{pmatrix},
\]

and the corresponding matrices \( A \) and \( B \) as

\[
A = \begin{pmatrix}
A_1 & A_2 & 0 & \cdots & 0 & A_M \\
A_M & A_1 & A_2 & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & \ddots & \ddots \\
0 & \cdots & A_M & A_1 & A_2 & 0 \\
0 & 0 & \cdots & A_M & A_1 & A_2 \\
A_2 & 0 & 0 & \cdots & A_M & A_1 \\
\end{pmatrix}, \quad B = \begin{pmatrix}
B_1 & B_2 & 0 & \cdots & 0 & B_M \\
B_M & B_1 & B_2 & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & \ddots & \ddots \\
0 & \cdots & B_M & B_1 & B_2 & 0 \\
0 & 0 & \cdots & B_M & B_1 & B_2 \\
B_2 & 0 & 0 & \cdots & B_M & B_1 \\
\end{pmatrix}.
\]
A and B are $n \times n$-matrices as we said previously, with $n = MN$; $A_i, B_i; i = 1, 2, 3$ are $N \times N$-matrices given as

$$A_1 = \begin{pmatrix}
d & e & 0 & \cdots & 0 & b \\
b & d & e & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & 0 & 0 \\
0 & \cdots & b & d & e & 0 \\
0 & 0 & \cdots & b & d & e \\
e & 0 & 0 & \cdots & b & d
\end{pmatrix},$$

$$B_1 = \begin{pmatrix}
b_{22} & b_{23} & 0 & \cdots & 0 & b_{21} \\
b_{21} & b_{22} & b_{23} & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & 0 & 0 \\
0 & \cdots & b_{21} & b_{22} & b_{23} & 0 \\
0 & 0 & \cdots & b_{21} & b_{22} & b_{23} \\
b_{23} & 0 & 0 & \cdots & b_{21} & b_{22}
\end{pmatrix},$$

$$A_2 = \begin{pmatrix}
e & f & 0 & \cdots & 0 & c \\
c & e & f & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & 0 & 0 \\
0 & \cdots & c & e & f & 0 \\
0 & 0 & \cdots & c & e & f \\
f & 0 & 0 & \cdots & c & e
\end{pmatrix},$$

$$B_2 = \begin{pmatrix}
b_{32} & b_{33} & 0 & \cdots & 0 & b_{31} \\
b_{31} & b_{32} & b_{33} & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & 0 & 0 \\
0 & \cdots & b_{31} & b_{32} & b_{33} & 0 \\
0 & 0 & \cdots & b_{31} & b_{32} & b_{33} \\
b_{33} & 0 & 0 & \cdots & b_{31} & b_{32}
\end{pmatrix},$$

$$A_3 = \begin{pmatrix}
b & c & 0 & \cdots & 0 & a \\
a & b & c & 0 & \cdots & 0 \\
\vdots & \ddots & \ddots & \ddots & 0 & 0 \\
0 & \cdots & a & b & c & 0 \\
0 & 0 & \cdots & a & b & c \\
c & 0 & 0 & \cdots & a & b
\end{pmatrix},$$
The resulting equation, for the case of no chemical reaction ($\epsilon = 0$), is

$$\dot{X} = \omega + P[V(X) + AV(X) + BV(Y)],$$  \hspace{1cm} (9)$$

and for $\epsilon \neq 0$, the equation will be

$$\dot{X} = \omega + P[V(X) + AV(X) + W]$$

$$\epsilon \dot{W} = -W + BV(Y),$$ \hspace{1cm} (10)$$

where $X = (X_1, \ldots, X_M)^T$, $X_i = (X_{i1}, \ldots, X_{iN})^T$, $X$ describes the phases of the whole network; after this order is set we may write $X$ as $X = (X_1, \ldots, X_n)$; with this in mind, the rest of parameters in the system are written as $\omega = (\omega_0, \ldots, \omega_n)^T$; $\omega$ is the center frequencies vector, $W = (W_1, \ldots, W_n)^T$ is the output voltage vector coming out from the network, in particular $W_i$ is the output voltage coming out of cell $i$; $P, V : \mathbb{R}^n \rightarrow \mathbb{R}^n$ are given as $P(X) = (P(X_1), \ldots, P(X_n))^T$, $V(X) = (V(X_1), \ldots, V(X_n))^T$; $V(X_i), 1 \leq i \leq n$ is voltage output of VCON at cell $i$; $V(X) + = (d(V(X_1)), \ldots, d(V(X_n)))^T$; $Y = (y_1, \ldots, y_n)^T$ and $y_j, 1 \leq j \leq n, n = MN$, is phase of input voltage coming from cell $j$; it might be taken as $y_j = \nu_j t$, where $\nu_j$ is external voltage frequency put into the network at site $j$. Because we are working in a $M \times N$ array and above there are elements in $\mathbb{R}^n$. This same order is considered in [2, 3] for the study of CNN. Without danger of confusion we shall write $P, V$, and $W$ as $P, V$, and $W$ respectively. Then (9)-(10) can be rewritten, respectively, as

$$\dot{X} = \omega + P[V(X) + AV(X) + BV(Y)].$$ \hspace{1cm} (11)$$

$$\dot{X} = \omega + P[V(X) + AV(X) + W]$$

$$\epsilon \dot{W} = -W + BV(Y).$$ \hspace{1cm} (12)$$

This is the system that appears in [8]; our next step is to modify it and implement some numerical computations seeking for synchronization. The first thing to change in (12) is the center frequency $\omega$; it is natural to expect that it will change with time and in an oscillatory way; so we consider...
\[ \omega = \omega(t) = (\omega_1(t), \ldots, \omega_n(t))^T \quad \text{and} \quad \omega_i(t) = 0.5 \sin(4\pi t); \ i = 1, \ldots, n. \] Actually, the particular form of \( \omega_i \) is taken to fit numerical expectations, but it can be chosen in a more general way, say, \( \omega_i(t) = A \sin(Bt) \). We also consider \( V(X_i) = 15 \cos(\pi X_i/6); \ i = 1, \ldots, n. \) Now (12) looks like

\[
\begin{align*}
\dot{X} &= \omega(t) + P[V(X) + AV(X)_+ + W] \\
\epsilon W &= -W + BV(Y)_+.
\end{align*}
\] (13)

Notice that the second equation in the above system does not involve \( X \). However, the VCO model suggests that the synapse voltage should depend on \( X \), since the voltage \( V(X) \) coming out of cell body passes through the axon to the synapse. The synapse receives this stimulus through the axon in an oscillatory way, so we wish to make the second equation in (13) reflect this oscillatory dependence. We therefore introduce the following vector and diagonal (and constant) matrix, respectively,

\[ G(X) = (3X_1 - X_1^3, \ldots, 3X_n - X_n^3)^T, \quad D = \text{diag}(d_1, \ldots, d_n); \]

we will consider \( d_i = 2, \ i = 1, \ldots, n \) but other values can be assumed as well. It is important to notice that for \( X_i, \ i = 1, \ldots, n; \) small, \( G(X) \) remains bounded. We rewrite (13) as

\[
\begin{align*}
\dot{X} &= \omega(t) + P[V(X) + AV(X)_+ + W] \\
\epsilon W &= -W + BV(Y)_+ + DG(X).
\end{align*}
\] (14)

4 Numerical Simulation

In this section we implement some numerical computations seeking for synchronization; the definition of synchronization we use is the one given in [1].

The first numerical simulation with this model is done for nine cells; different types of cloning templates and \( \epsilon \) are considered. The first implementation is considering (8) as

\[
\begin{align*}
\tilde{A} &= \begin{pmatrix} 0.008 & -0.1 & 0.005 \\ -0.1 & 0 & 0.02 \\ 0.005 & 0.02 & -1 \end{pmatrix},
\tilde{B} &= \begin{pmatrix} 2.35 & 0.01 & 1.1 \\ 0.7 & 0.7 & 1 \\ 0.25 & 0.5 & 1 \end{pmatrix}.
\end{align*}
\]
This is a case of strong interaction ($b_{ij} > 0$): we take $\epsilon = 0.1$ and initial conditions $X(0) = (0.75, 1, 1.5, 1.75, 0.5, -0.5, 1.8, 0.9, 1.3, 0.75, 0.9, 1.4, 1.75, 0.4, 1.9, 1.75, 0.9, 1)$. Some plotting is given in Figure 6. As we can see from this picture, synchronization is present; of course the plotting of other components also show synchronization.

It is important to mention here that numerical simulations suggest that initial conditions, $\epsilon$ and $\tilde{A}$ may be chosen more or less arbitrary and synchronization (or non synchronization) is not affected; that is, little changes in the above mentioned parameters do not change the synchronization (or non synchronization) of the overall system.
Next we introduce some null stimuli \((b_{ij} \geq 0)\); that will be called 'some excitation'. Actually, we consider \(\epsilon = 0.01\) and (8) as

\[
\tilde{A} = \begin{pmatrix} 0.8 & -1 & -0.5 \\ -1 & 0 & 0.02 \\ 0.5 & 0.02 & -1 \end{pmatrix}, \quad \tilde{B} = \begin{pmatrix} 2 & 0.1 & 1 \\ 0.25 & 0 & 1 \\ 2 & 0.1 & 1 \end{pmatrix}
\]

and use the same initial conditions as before. After numerical implementation, we observe that synchronization is lost; some of the plotting are given in Figure 7. However it is possible to give cloning templates such that some excitation also produce synchronization; that will be done in the case of 25 cells.

Figure 7: Nine Cells, \(\epsilon = 0.01\), Some Excitation.
The next case into consideration is strong inhibition; that is; \(b_{ij} < 0\), again we take \(\epsilon = 0.1\) but as we mentioned before it can take another value; (8) is chosen as

\[
\tilde{A} = \begin{pmatrix}
0.008 & 0.01 & 0.005 \\
0.01 & 0 & 0.02 \\
0.005 & 0.02 & 0.001
\end{pmatrix}, \quad \tilde{B} = \begin{pmatrix}
-2 & -0.1 & -1 \\
-0.7 & -0.7 & -1 \\
-0.25 & -1 & -1
\end{pmatrix};
\]

we observed from Figure 8 that synchronization is present again.

Notice that we represent only a few plots; but all of them were tested and we found the same results as indicated. Again, by moving the coefficients of \(\tilde{A}\) the results remain unchanged.

Now we test it for same \(\epsilon\) and \(\tilde{A}\) as before, some inhibition is assumed

Figure 8: Nine Cells, \(\epsilon = 0.1\), Strong Inhibition
$(b_{ij} \leq 0)$; actually we consider

\[
\tilde{B} = \begin{pmatrix}
2 & 0.1 & 1 \\
0.25 & 0 & 1 \\
2 & 0.1 & 1
\end{pmatrix};
\]

numerical results show that synchronization is gone; we mention also that this case was tested for time greater than 40, nevertheless the system still does not show synchronization. If synchronization takes place that should be reached in a short interval of time.

The same array of nine cells is considered but now we introduce excitation and inhibition in the same cloning template: some $b_{ij}$ are positive, some are negative, and some others are zero; $\epsilon$ is assumed to be 0.1 and

\[
\tilde{A} = \begin{pmatrix}
0.8 & -0.1 & 0.5 \\
-0.1 & 0 & 0.02 \\
0.5 & 0.02 & -1
\end{pmatrix}, \quad \tilde{B} = \begin{pmatrix}
0 & -0.1 & 1 \\
0.5 & -0.7 & 0 \\
0.25 & 0 & -1
\end{pmatrix}.
\]

In this particular case synchronization is lost (some plotting are given in Figure 9). However, as we shall see later (case of 25 cells), it is possible to give templates, in a situation as above where synchronization is found.

The case $\epsilon = 0$ is not so important since it means no chemical kinetics, but in practice chemical kinetics is almost always present in any neuronal process; in any case we implemented this case for different choices of the templates. The first we looked at was

\[
\tilde{A} = \begin{pmatrix}
0.8 & -0.1 & 0.05 \\
-0.1 & 0 & 0.02 \\
0.05 & 0.02 & -1
\end{pmatrix}, \quad \tilde{B} = \begin{pmatrix}
2 & 0.1 & 1 \\
0.7 & 0.7 & 1 \\
2 & 0.1 & 1
\end{pmatrix};
\]

and initial conditions

\[
X(0) = (0.75, 1, 0.1, -0.5, 0.5, -1, 1.8, -1, 0.1);
\]

synchronization is found.
Next we try with $\tilde{A}$ as the foregoing and $\tilde{B}$ assumed to be

$$\tilde{B} = \begin{pmatrix} 2 & 0.1 & 1 \\ 0.7 & 0.7 & 1 \\ 2 & 0.1 & 1 \end{pmatrix};$$

synchronization is present again, but as we said before the case of $\epsilon = 0$ is not so relevant for this model.

Next we consider an array of twenty five cells; a $5 \times 5$ array. For this case we will try different values of $\epsilon$ (small ones) but as said before this does not seem to affect the behavior of the whole system. The initial data considered is generated for the formula below; again changing the initial data does not affect the behavior of the set of cells, therefore we keep this same initial conditions
along the simulations. If the initial data is denoted by

\[ X(0) = (X(0)_1, X(0)_2, \ldots, X(0)_{50}) \]

we take

\[ X(0)_i = \cos\left(\frac{2\pi}{50}\right) \text{ if } 1 \leq i \leq 25 \]

and

\[ X(0)_i = \cos\left(\frac{3\pi}{50}\right) \text{ if } 26 \leq i \leq 50. \]

This formula may look strange but it is just a way to produce fifty numbers without write them one by one (for \( n \) cells there are \( 2n \) equations).

Figure 10: Twenty Five Cells, \( \epsilon = 0.1 \), Strong Excitation.

The first case under consideration is strong excitation (\( b_{ij} > 0 \)); for this
we pick $\epsilon = 0.1$ and (8) given by

$$
\tilde{A} = \begin{pmatrix}
0.3 & 0.1 & 0.25 \\
0.1 & 0 & 0.02 \\
0.25 & 0.02 & 1
\end{pmatrix}, \quad \tilde{B} = \begin{pmatrix}
1.35 & 1.55 & 2.1 \\
1.8 & 1.7 & 1 \\
1.25 & 1.85 & 2.35
\end{pmatrix}.
$$

In this case, as we expect, synchronization is present; this situation is depicted in Figure 10. Again we only show a few plots but all the components of the solution were tested with similar results.

By considering the same cloning templates as above and $\epsilon = 0.01$ the same results are obtained, i.e.; synchronization is reached.

![Figure 11: Twenty Five Cells, $\epsilon = 0.1$, Some Excitation.](image)

We add now some null stimuli, that is, some $b_{ij} = 0$; specifically we
consider (8) to be
\[
\begin{align*}
\tilde{A} &= \begin{pmatrix} 0.08 & -1 & -0.5 \\ -1 & 0 & 0.02 \\ -0.5 & 0.02 & -1 \end{pmatrix}, \\
\tilde{B} &= \begin{pmatrix} 1.35 & 0.1 & 0 \\ 1.8 & 0.7 & 1 \\ 0.25 & 0 & 1 \end{pmatrix}; \\
\epsilon &= 0.1.
\end{align*}
\]
We found no synchronization even changing values of \(\epsilon\) and \(\tilde{A}\); in Figure 11 this situation is depicted.

As we did in \(3 \times 3\) array, let us consider strong inhibition, here \(\epsilon = 0.02\) and (8) given by
\[
\begin{align*}
\tilde{A} &= \begin{pmatrix} 0.3 & 0.1 & 0.25 \\ 0.1 & 0 & 0.02 \\ 0.25 & 0.02 & 1 \end{pmatrix}, \\
\tilde{B} &= \begin{pmatrix} -0.3 & -0.1 & -1 \\ -0.7 & -0.7 & -1 \\ -0.25 & -1 & -0.5 \end{pmatrix};
\end{align*}
\]

Figure 12: Twenty Five Cells, \(\epsilon = 0.02\), Strong Inhibition.
and as we expect, synchronization is found; plotting corresponding to this case is given in Figure 12.

By introducing some inhibition \((b_{ij} \leq 0)\), say,

\[
\tilde{B} = \begin{pmatrix}
-0.3 & -0.1 & -1 \\
-0.7 & 0 & -1 \\
-0.25 & -1 & -0.5
\end{pmatrix},
\]

\(\tilde{A}\) as before and \(\epsilon = 0.05\) synchronization is found. Again the value of \(\epsilon\) is not so important; in the particular case under consideration we chose other values of \(\epsilon\) with the same results.

The following is the case of some excitation and some inhibition in the same templates, with \(\epsilon\) set equal to 0.1; (8) is

\[
\tilde{A} = \begin{pmatrix}
1.3 & 0.1 & 0.25 \\
0.1 & 0 & 0.02 \\
0.25 & 0.02 & 1
\end{pmatrix}, \quad \tilde{B} = \begin{pmatrix}
1.35 & -1.55 & 2.1 \\
1.8 & 1.75 & -1 \\
1.25 & 1.85 & -0.35
\end{pmatrix};
\]

we get synchronization again; in Figure 13 the above case is depicted.

Choosing another expression for \(G\), say,

\[
G(X) = (\alpha \sin(X_1), \ldots, \alpha \sin(X_n))^T
\]

(wave-like function) we also implemented numerical simulation with results essentially similar to those already mentioned. It is possible to have synchronization for \(X\) but no synchronization for \(W\). What this indicates is that even in the case of very similar electrical behavior of the body of different cells, the behavior of the corresponding synapses may not be similar. The reason for this may lay in the way the brain reacts to different stimuli, for instance, a smelling sensation makes some cells of the brain respond while some others do not.
Moreover, there are no rigid separate functions for each particular brain region, but at the same time the brain does not function as a homogeneous mass. Rather, different brain regions have different and flexible roles in a coordinated, integrated brain; more details can be seen in [4]. In 1962, David Hubel and Torsten Wiesel ([9]) showed that neurons in a particular region do not all behave in the same way; instead, groups of neurons become active under very specific conditions. Another factor to be taken into consideration is the shape of the neurons: there are at least fifty basic neuronal shapes in the brain which can affect the efficiency of signaling ([4]). Small cells are excited more easily than larger ones (because the smaller cells have a higher resistance, and so any current produced as an incoming signal is transformed into a larger voltage). Size, then, and the number and length of processes that extend from a cell are critical factors in its behavior. Therefore the seemingly easy metaphor of hardware and software does not really work as an analogy.
Finally we would like to mention that even when numerical implementation of finite array of cells and fixed and unchanged connections among them may seem unrealistic, that is not always the case; for instance in [14] it is shown that the muscles in the lobster stomach, whose movements cause the lobster stomach to digest food, are controlled by a total of twenty neurons grouped together in a hard-wired assembly; that is, the connections among them are fixed and unchanging. It is an intriguing fact that the output of this group of neurons is not fixed and invariant: the rhythms of contraction of the stomach muscles that they produce are enormously versatile.

We have presented a modified version of the model of electrical behavior of neurons given in [8] by using the model of CNN previously studied. As we just have seen, numerical implementations of this model show that it is possible to have synchronization in short a period of time even in cases where some stimuli are zero; still there is much work to be done in this direction; we believe this is a starting point.

References


