ISSN 2347-3487

Brain Interconnections

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ABSTRACT

The interaction brain-machine is now an indissoluble fact. The repercussions this fact will have in future are presently unimaginable. As mentioned in [1] the nerve axon can now be modeled by a cable or a planar high speed interconnection, no matter what kind of interconnection will be used. The brain development is clearly shown in a stupendous book of the SOCIETY FOR NEUROSCIENCE treating the brain and the nervous system. An extraction of the brain development chapter is here included to show the relationship among the physiological brain connections [2] and the high speed electrical connections [1]. On the other hand, because of the massive volume of information storage now a day the synchronization clock speeds are all in the range of GHz almost reaching the THz. At these very high frequencies the behavior of the interconnects are more like that of a transmission line, and hence distortion, delay, and phase shift-effects caused by phenomena like cross talk, ringing, and overshoot are present and may be undesirable for the performance of a circuit or system. Thus, the interconnects do not have to be considered like simple conductors or lumped elements. All this gives rise to a new emerging discipline known as signal integrity. This discipline is extremely important to maintain the signal quality on microstrip circuits [3]. In this discipline the correct timing and signal quality preservation preventing transients and false switching are studied in order to avoid excessive delays.

Keywords

Signal Integrity, distortion, delay, phase shift-effects, cross talk, ringing and overshot.

ISSN 2347-3487

1. INTRODUCTION

Extraction of [2]

Three to four weeks after conception, one of the two cell layers of the gelatin-like human embryo, now about one-tenth of an inch long, starts to thicken and build up along the middle. As this flat neural plate grows, parallel ridges, similar to the creases in a paper airplane, rise across its surface. Within a few days, the ridges fold in toward each other and fuse to form the hollow neural tube. The top of the tube thickens into three bulges that form the hindbrain, midbrain and forebrain. The first signs of the eyes and then the hemispheres of the brain appear later. How does all this happen? Although many of the mechanisms of human brain development remain secrets, neuroscientist are beginning to uncover some of these complex steps through studies of the roundworm, fruit fly, frog, zebra fish, mouse, rat, chicken, cat, and monkey.

Many initial steps in brain development are similar across species, while later steps are different. By studying these similarities and differences, scientists can learn how the human brain develops and how brain abnormalities, such as mental retardation and other brain disorders, can be prevented or treated.

Neurons are initially produced along the central canal in the neural tube. These neurons then migrate from their birthplace to a final destination in the brain. They collect together to form each of the various brain structures and acquire specific ways of transmitting nerve messages. Their processes, or axons, grow long distances to find and connect with appropriate partners, forming elaborate and specific circuits.

Finally, sculpting action eliminates redundant or improper connections, honing the specificity of the circuits that remain. The result is the creation of a precisely elaborated adult network of 100 billion neurons capable of a body movement, a perception, an emotion or a thought.

Knowing how the brain is put together is essential for understanding its ability to reorganize in response to external influences or to injury. These studies also shed light on brain functions, such as learning and memory. Brain diseases such as schizophrenia and mental retardation are thought to result from a failure to construct proper connections during development. Neuroscientist are beginning to discover some general principles to understand the processes of development, many of which overlap in time.

Birth of neurons and brain wiring

The embryo has three primary layers that undergo many interactions in order to evolve into organ, bone, muscle, skin or neural tissue. The skin and neural tissue arise from a single layer, known as the mesoderm.

A number of molecules interact to determine whether ectoderm becomes neural tissue or develops in another way to become skin.

Studies of spinal cord development in frogs show that one major mechanism depends on specific molecules that inhibit the activity of various proteins. If nothing interrupts the activity of such proteins, the tissue becomes skin. If other molecules, which are secreted from mesodermal tissue, block protein signaling, then tissue becomes neural.

Once the ectodermal tissue has acquired is neural fate, another series of signaling interactions determine the type of neural cell to which it gives rise. The mature nervous system contains a vast array of cell types, which can be divided into two main categories:

the neurons, primarily responsible for signaling, and supporting cells called glial cells.

Researchers are finding that the density of neural tissue depends on a number of factors, including position that defines the environmental signals to which the cells are exposed. For example, a key factor in spinal cord development is a secreted protein called sonic hedgehog that is similar to a signaling protein found in flies. The protein initially secreted from mesodermal tissue lying beneath the developed spinal cord, marks young neural cells that are directly adjacent to become a specialized class of glial cells. Cells further away are exposed to lower concentrations of sonic hedgehog protein, and they become the motor neurons that control muscles. An even lower concentration promotes the formation of interneurons that relay messages to other neurons, not muscles.

A combination of signals also determines the type of chemical messages, or neurotransmitters, that a neuron will use to communicate with other cells. For some, such as motor neurons, the choice is invariant, but for others it is a matter of choice. Scientists found that when certain neurons are maintained in a dish without any other cell type, they produce the neurotransmitter norepinephrine. In contrast, the same neurons are maintained with other cells, such as cardiac or heart tissue cells, they produce the neurotransmitter acetylcholine. Since al neurons have genes containing the information for the production of these molecules, it is the turning on of a particular set of genes that begins the production of specific neurotransmitters. Many researchers believe that the signal to engage the gene and, therefore, the final determination of the chemical messengers that a neuron produces, is influenced by factors coming from the targets themselves.

As neurons are produced, they move from the neural tube's ventricular zone, or inner surface, to near the border of the marginal zone or the outer surface. After neurons stop dividing, they form an intermediate zone where they gradually accumulate as the brain develops. The migration of neurons occurs in most structures of the brain, but is particularly prominent in the formation of a large cerebral cortex in primates, including humans. In this structure, neurons slither from the place of origin near the ventricular surface along nonneuronal fibers that form a trail to their proper destination. Proper neuron migration requires mechanisms, including the recognition of the proper path and the ability to move long distances.

One such mechanism for long distance migration is the movement of neurons along elongated fibers that form transient scaffolding in the fetal brain. Many external chemical and physical factors such as alcohol, cocaine or radiation, prevent proper neuronal migration and result in misplacement of cells, which may lead to mental retardation and epilepsy. Furthermore, mutations in genes that regulate migration have recently been shown to cause some rare genetic forms of retardation and epilepsy in humans. Once the neurons reach their final location, they must make the proper connections for a particular function, such as vision or hearing, to occur. They do this through their axons. These stalk-like appendages can stretch out a thousand times longer than the cell body from which they arise. The journey of most axons ends when they meet the branching areas, calling dendrites, on other neurons. These target neurons can be located at a considerable distance, sometimes at opposite sides of the brain. In the case of motor neuron, the axon may travel from the spinal cord all the way down to a foot muscle. The linkup sites, called synapses, are where messages are transferred from one neuron in a circuit to the next. Axon growth is spearheaded by growth cones. These enlargements of the axon's trip actively explore the environment as they seek out their precise destinations. Researchers have discovered that many special molecules help guide growth cones. Some molecules lie on the cells that growth cones contact, while others are released from sources found near the growth cone. The growth cones, in turn, bear molecules that serve as receptors for the environmental cues. The binding of particular signals with its receptors tells the growth cone whether to move forward, stop, recoil or change direction. Recently researchers have identified some of the molecules that as cues and receptors. These molecules include proteins with names such as cadherin, netrin, semaphoring, ephrin, neuropilin and plexin. In most cases, these are families of related molecules; for example there are at least 15 semaphorins and at least 10 ephrins. Perhaps the most remarkable result is that most of these are common to worms, insects and mammals, including humans. Each family is smaller in flies or worms that in mice or people, but their functions are quite similar. It has therefore been possible to use the simpler animals to gain knowledge that can be directly applied humans. For example, the first netrin was discovered in a worm and shown to guide neurons around the worm's "nerve ring". Later, vertebrate netrins were found to guide axons around the mammalian spinal cord. Worm receptors for netrins were then found and proved invaluable in finding the corresponding, and again related, human receptors. Once axons reach their targets, they form synapses, which permit electric signals in the axon to jump to the next cell, where they can either provoke or prevent the generation of a new signal. The regulation of this transmission at synapses, and the integration of inputs from the thousands of synapses each neuron receives, is responsible for the astounding information processing capabilities of the brain. For processing to occur properly, the connections must be highly specific. Some specificity arises from the mechanisms that guide each axon to its proper target area. Additional molecules mediate "target recognition" whereby the axon chooses the proper neuron, and often the proper part of the target, once it arrives at its destination. Few of these molecules have been identified. There has been more success, however, in identifying the ways in which the synapse forms once the contact has been made. The tiny portion of the axon that contacts the dendrite becomes specified for the release of neurotransmitters, and the tiny portion of the dendrite that receives the contact becomes specialized to receive and respond to the signal. Special molecules pass between the sending and receiving cell to ensure that the contact is formed properly.

Paring back

Following the period of growth, the network is pared back to create a more sturdy system. Only about one-half of the neurons generated during development survive to function in the adult. Entire populations of neurons are removed through internal suicide programs initiated in the cells. The programs are activated if a neuron loses its battle with other neurons to receive life-sustaining nutrients called trophic factors. These factors are produced in limited quantities by target tissues. Each type of trophic factor supports the survival of a distinct group of neurons. For example, nerve growth factor is important for sensory neuron survival. It has recently become clear that the internal suicide program is maintained into adulthood, and constantly held in check. Based on this idea, researchers have found that injuries and some neurodegenerative diseases kill neurons not directly by the damage they inflict, but rather by activating the death program. This discovery, and its implication that death need not inevitably follow insult; have led to new avenues for therapy. Brain cells also form too many connections at first. For example, in primates, the projection from the two eyes to the brain initially overlaps and then sorts out to separate territories devoted only to one or the other eye. Furthermore, in the young primate cerebral cortex, the connections between neurons are greater in number and twice as dense as an adult primate. Communication between neurons with chemical and electrical signals is necessary to weed out the connections. The connections that are active and generating electrical currents survive while those with little or no activity are lost.

Critical periods

The brain's refining and building of the network in mammals, including humans, continues after birth. An organism's interactions with its surroundings fine-tune connections. Changes occur during critical periods. These are windows of time during development when the nervous system must obtain certain critical experiences, such as sensory, movement or emotional input, to develop properly. Following a critical period, connections become diminished in number and less subject to change, but the ones that remain are stronger, more reliable and more precise.

Injury, sensory or social deprivation occurring at a certain stage of postnatal life may affect one aspect of development, while the same injury at a different period may affect another aspect. In one example, a monkey is raised from birth up to six months of age with one eyelid closed. As a result of diminished use, the animal permanently loses useful vision in that eye. This gives cellular meaning to the saying "use it or lose it". Loss of vision is caused by the actual loss of functional connections between that eye and neurons in the visual cortex. This finding has led to earlier and better treatment of the eye disorders congenital cataracts and "crossed-eyes in children. Research also shows that enriched environments can bolster brain development during postnatal life. For example, studies show that animals brought up in toy-filled

surroundings have more branches on their neurons and more connections than isolated animals. In one recent study, scientists found enriched environments resulted in more neurons in a brain area involved in memory. Scientists hope that new insights on development will lead to treatments for those with learning disabilities, brain damage and even neurodegenerative disorders or aging.

```
% THE RINGING AND OVERSHOOT OF A SIMULATED MICROSTRIP TRANSMISSION LINE %


clear all
warning off


%% ACQUIRING DATA.
CO2=0;
%Note1: CO is the value for the connector in cm.


IV=9;
JV=59+CO2;
KV=3+3;
LV=59+CO2;
freqmi=0*1e9;
freqma=3*1e9;
freqce=(freqma+freqmi)/2;
freqstep=0.01*1e9;


%Permitividad de la microcinta.
epsrm=10.5;


%Permeabilidad en el vacio.
muz=4*pi*1e-7;


%Permitividad en el vacio.
epsz=8.854e-12;


cz=1/(sqrt(muz*epsz));


%Espesor del dieléctrico.
W1=0.001882;
```

ISSN 2347-3487

```
%Valor en x de la micro cinta.
ddx=W1/3;

%Valor en y de la micro cinta.
ddy=ddx;

dt=ddx/(sqrt(2)*cz);

nsteps=input('Enter the number of timesteps: ');
sel= questdlg('¿Graficar en 2D o 3D?', 'INFO', '2-D', '3-D', '3-D');

nfreqs=((freqma-freqmi)/freqstep)+1;
freq(1:nfreqs)=freqmi:freqstep:freqma;
freqi(1:nfreqs)=freqma:-freqstep:freqmi;

arg(1:nfreqs)=2*pi*freq(1:nfreqs)*dt;
args(1:nfreqs)=2*pi*freq(1:nfreqs);

H=0.000635;
W1=0.001882;
Wf1=0.001882*1e3;

[ls1, cs1, Zo1, vpm] = minocodi(epsrm, epsz, H, W1);

Lvp1=((dt*vpm)*ls1)/(2*ddx);
Lp1=(ls1/2)+Lvp1;

Zo=Zo1;                          %Impedancia caracteristica.
Zs=Zo/3;%Zo;%3*Zo;%Zo/4;         %Impedancia de fuente.
Zl=0.0;%6.5*Zo;%Zo;%Zo+i*Zo;     %Impedancia de carga.
clc

v(1:IV,1:JV+1)=0;
```

ISSN 2347-3487

```
jx(1:IV+1,1:JV)=0;
jy(1:IV,1:JV)=0;
t0=20;
spread=4;
T=0;
ini=1;
sal=1;

%% CYCLE FOR
for t=1:nsteps

        T=T+1;
        pulse=1;
        v(4:KV,1)=pulse;
        jy(4:KV,2)=jy(4:KV,2)-((v(4:KV,3)-v(4:KV,2))*Lp1);
        v(4:KV,2)=v(4:KV,1)-(jy(4:KV,2)*mean(Zs));
        jx(5:KV,1:LV)=jx(5:KV,1:LV)+(dt/(ls1*ddx))*(v(4:KV-1,1:LV)-
v(5:KV,1:LV));
        jy(4:KV,1:LV)=jy(4:KV,1:LV)+(dt/(ls1*ddy))*(v(4:KV,1:LV)-
v(4:KV,2:LV+1));
        v(4:KV,3:LV)=v(4:KV,3:LV)+(dt/(cs1*ddx))*(jx(4:KV,3:LV)-
jx(5:KV+1,3:LV)-jy(4:KV,3:LV)+jy(4:KV,2:LV-1));
        jy(4:KV,LV)=jy(4:KV,LV)+((v(4:KV,LV)-v(4:KV,LV+1))*Lp1);
        v(4:KV,LV+1)=jy(4:KV,LV)*mean(Zl);

        if  t==ini
            timestep=int2str(t);
            time=num2str(t*dt);

            %GRAFICA EN 2-D
            if strcmp(sel,'2-D')

                %PLOT 1
                subplot(2,1,1)
```

ISSN 2347-3487

```
                surf(1:JV+1,1:IV,v);
                title('Amplitude of potential waveform','FontSize',
20,'FontWeight','Bold')
                zlabel('Volts','FontSize',20)
                view([0,0])

                %INFORMACIÓN DE TIMESTEPS Y TIEMPO
                text(-10,0,-0.2,['Timesteps
=',timestep],'FontWeight','Bold','FontSize',20)
                text(-10,0,-0.3,['Time(s) = ',time],'FontSize',
20,'FontWeight','Bold')

                %PLOT 2
                subplot(2,1,2)
                surf(1:JV,1:IV,jy);
                title('Amplitude of current density waveform','FontSize',
20,'FontWeight','Bold')
                xlabel('Number of cells','FontSize',20)
                zlabel('Amperes/m','FontSize',20)
                axis([0 JV 0 IV 0.0 0.2])
                view([0,0])
            end

            %GRAFICA EN 3-D
            if strcmp(sel,'3-D')

               %PLOT 1
                subplot(2,1,1)
                surf(1:JV+1,1:IV,v);
                title('Amplitude of potential waveform','FontSize',
20,'FontWeight','Bold')
                zlabel('Volts','FontSize', 20)

                %INFORMACIÓN DE TIMESTEPS Y TIEMPO
                text(-((JV+1)/(5)),0,-1.5,['Timesteps
=',timestep],'FontSize', 20,'FontWeight','Bold')
```

```
                text(-((JV+1)/(5)),0,-2.0,['Time(s) = ',time],'FontSize',
20,'FontWeight','Bold')
                axis([0 JV+1 0 IV 0.0 2.0])
                view([15,45])

                %PLOT 2
                subplot(2,1,2)
                surf(1:JV,1:IV,jy);
                title('Amplitude of current density waveform','FontSize',
20,'FontWeight','Bold')
                xlabel('Number of cells','FontSize', 20);
                zlabel('Amperes/m','FontSize',20)
                axis([0 JV 0 IV 0.0 0.2])
                view([15,30])
            end
            pause(0.01)
            ini=ini+sal;
        else end
        %Notas del programa:
        %view([0,0]) activa la vista en 2D
        %view([15,45]) activa la vista en 3D
        %colorbar('east') coloca una barra de color;
End
```

Conclusion

The paper has shown the electromagnetic simulation of the nerve axon when it is considered as a planar transmission line (see Fig. 1.). The simulation considers the nerve axon as a right conductor but when using printed circuit boards frequently many others non-right or multi-line conductors as synchronous impedance transformers, non-synchronous impedance transformers, right-angle bend discontinuities, low-pass filters and two-stub four-port directional couplers, may be necessarily used as demonstrated in [4].

Acknowledgements

The corresponding author would like to acknowledge the support of PROMEP-SEP, México, under grant PROMEP/103.5/11/6834, IDCA 5197, UDG-CA-424, and also to CONACYT, México by the continuous support via the PROSNI program. Besides, the corresponding author acknowledges the support of the Electronics Department, Computer and Electronics Division, CUCEI, Universidad de Guadalajara, México.

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ISSN 2347-3487

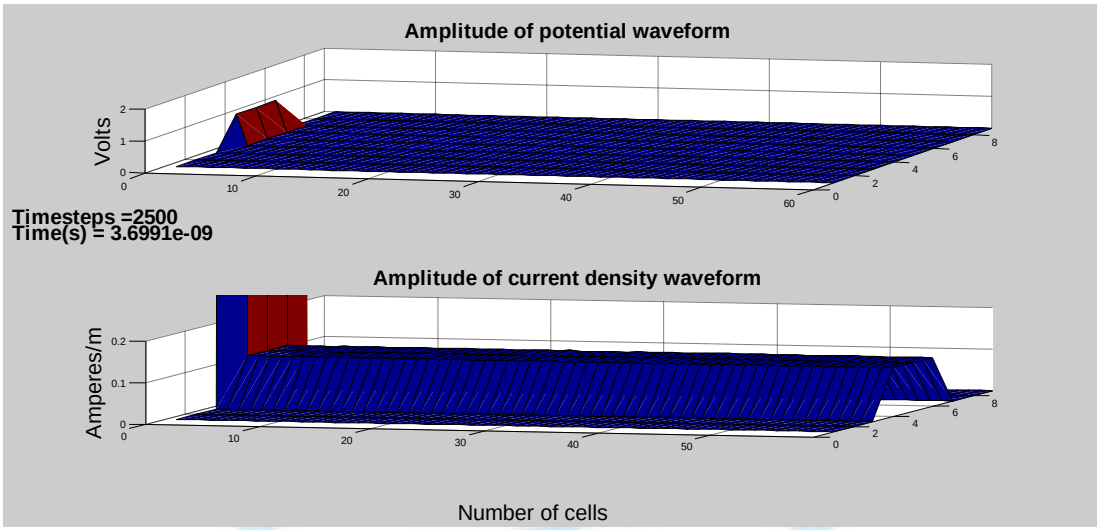

Fig.1. Amplitude of potential and current density waveforms at time(s) 3.6991e-09. The reflections have totally ceased.