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Study and Analysis of Human Nervous System and Effects of External EM Signals on Conduction in Human Nerves

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Abstract: Biological effects of microwaves or in general any EM irradiation is mostly analysis in terms of thermal effects. On this ground mostly exposures from mobile phone towers are said to be safe but some of the field studies reports strongly support the ill effects associated with it. Scientists and researchers are not of clear opinion about the mechanism, how the effects can be visualized. In the study reported here, the behavior of the nerve conduction is realized is in the form of electrical circuit, basically in the form of RC Transmission line. This paper shows simulation results of the effect of the continuous exposure of External EM Field on the nervous. Simulation is based on the MATLAB® and analysis of effect of mixing of AC signal with the membrane potential is done. Another simulation result of the affect of variation of frequency on the conduction behavior is also simulated in this paper. Results indicate that the reported bio-effects may be not of direct nature, like in case of ionic radiations but may be due to the malfunctioning of the never communication network due to EM- exposure. Further development of the experimental model is under process.

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I. INTRODUCTION

Effects of microwaves on bio-system or in general EM- irradiation is mostly analysis in terms of thermal effects. On this ground mostly exposures from mobile phone towers are said to be safe but some of the field studies reports strongly support the ill effects associated with it. Scientists and researchers are not of clear opinion about the mechanism, how the effects can be visualized. In the study reported here, the behavior of the nerve conduction is realized is in the form of electrical circuit by the biomedical engineers. Nerve conduction depends upon excitation and de-excitation of Na and K ions. Effect of the presence of any external electric signal on this conduction process was studied. Researchers have developed mechanism for the penetration of EM signal inside the skin of humans but only thermal affects of these signals are in reports.

SAR is used to define and find the penetration of the EM field inside the skin. In this paper, based on the concept of EM field penetration, we are presenting here the simulation results of direct impose of the penetrated signals on the nerves. These effects are measurable and show significant variation in the signal transmission through nerves. This work may lead to strengthen the non-thermal affects of the EM waves on bio-systems. Further development of the experimental model is under process.

II. BASIC NEURON STRUCTURE

The nervous system is a well organized network of specialized and excitable cells and tissues which can receive sensitive information and send it to the brain. Proper working of the nervous system is a must for the survival of the living organism. Structure of the neuron is shown in the Fig.1. The fundamental part of the nervous system is the neuron. The neuron may be compared to electrically excitable cell which receives and transmits electrical as well as chemical signals to targets (i.e. cells, tissue and organs).

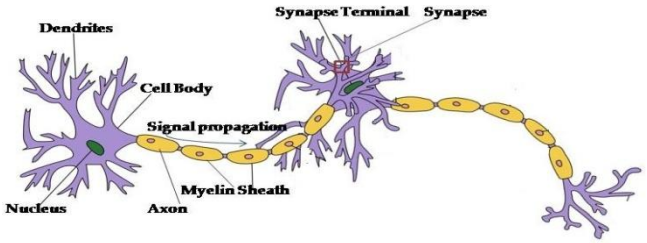


Fig 1: Nerve Diagram of Relevant Structure [1]

The human nervous system is anatomically divided into two parts namely the central nervous system (CNS) and peripheral nervous system (PNS). The CNS consists of the brain, spinal cord and complex neural networks. It receives sensitive information in form of signals from the organs, spinal nerves and peripheral nerves which are available in all parts of the body. This information is analyzed and then processed by the brain which formulates proper response. A response command is now generated and relayed out via spinal and peripheral nerves to be executed [2, 3].

III. PHYSICS OF NERVOUS SYSTEM

Most of the neurons in the nervous systems have an insulating layer around their axons, called the myelin sheath.

The sheath is formed by supporting cells, called Schwann cells that are wrapped around the axon.

Between Schwann cells are small regions of exposed axon called nodes of Ranvier. These nodes contain the voltage-gated ion channels which allow action potentials to propagate down in the axon, as result, signal jumps from node to node. This method (known as called salutatory conduction) allows signals to propagate much faster in myelinated neurons in comparison to those without a myelin sheath. The neuronal membrane and myelin sheath forms an insulating layer between the conducting axoplasm and extracellular fluid, as illustrated in Fig. 2.

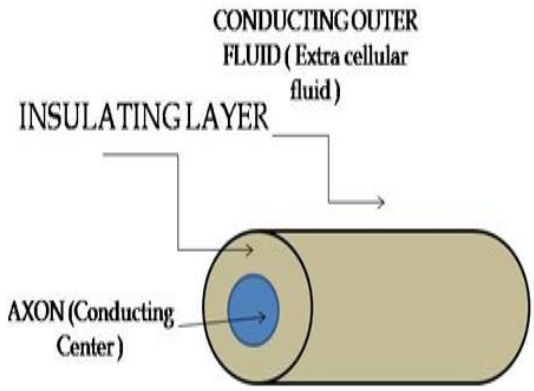


Fig. 2. Axon as an insulated wire.

When the action potential appears in a part of the axon, the voltage change that occurs there causes nearby charges to move toward it or away from it, as depicted in Fig. 3.

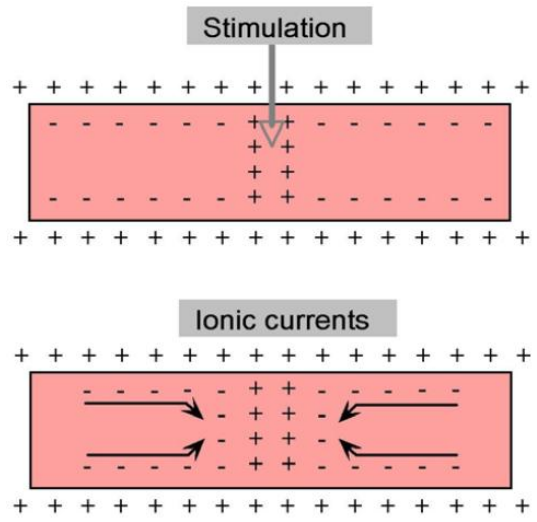


Fig. 3. Movement of charges inside the axon in response to a stimulus.

The movement of these charges, i.e. their electrical current, represents how fast the action potential travels along the length of the axon. These currents are limited not only by the electrical resistance they encounter but also by the membrane capacitance (the way they interact with charges across the membrane).

3.1. RESISTANCE

In the neuron, there are two substances that exhibit electrical resistance: the axoplasm itself and the cell membrane plus myelin sheath (if present). The electrical resistance R along the length of the axon follows the same principles as a wire where the resistivity ρ is a constant that depends on the medium and *l* and are the length and radius of the wire, respectively.

$$R = \rho l/A = \rho l/\pi r^2$$

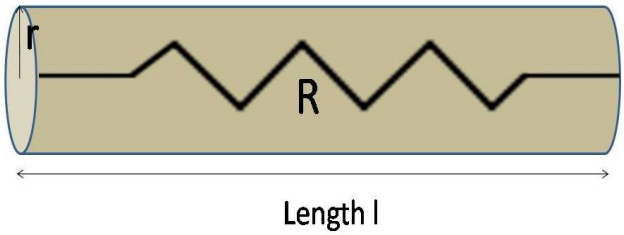


Fig. 4: A wire of length *l* and radius *r* has resistance R

For both myelinated and unmyelinated neurons, the resistivity ρ of the axoplasm is 2.0 Ω·m. For example, if the average neuron has an axon 1 mm long and a 5 μm radius, we can use Eq. (1) to find that the resistance of the axoplasm $R_{axoplasm} = 2.5X10^{7}$ Ω. This huge value indicates that axons are actually poor electrical conductors.

The cell membrane is also permeable to charge; its resistance is not infinite, even when myelinated. Rather than depending on cross-sectional area, the resistance through the membrane depends on the surface area of the axonal membrane:

$$R = \rho/A_{surface} = \rho/2\pi rl$$

For an unmyelinated axon (UA), $\rho_{UA} = 0.20$ Ω·m. So, again for an average axon 1 mm long with radius 5 μm, $R_{UA} = 6.4 \cdot 10^{6}$ Ω. Myelinated axons (MA) have a much higher resistivity, $\rho_{MA} = 40.0$ Ω·m, so $R_{MA} = 1.3 \cdot 10^{7}$ Ω.

3.2. CAPACITANCE

Capacitor consists of two conductors side by side, separated by some insulating substance called the dielectric. The ability to store charge comes from the attraction that charges in one plate experience toward the charges in the other (known as induction). A simple capacitor requires a voltage to be applied across the conducting metal plates first, in order to move the charges from one plate to the other. If both sides were electrically neutral to begin with, then

moving positive charges from one plate automatically implies that a net negative charge of identical magnitude is left behind.

The amount of charge Q that can be drawn from one to the other depends on the voltage V apllied across the plates, the separation d between the two plates, and the total surface area A between them:

$$Q = \varepsilon AV/d$$

The amount of charge stored for every volt applied across it, i.e. Q/V, is referred to as *capacitance*. The capacitance of a parallel plate capacitor is therefore

$$C=\varepsilon A/d$$

Here ε is a constant varies depending on the dielectric material present between the conducting plates. This constant is known as the permittivity. Again, the equation makes sense because the larger the surface area between the plates will store more charge. Furthermore, the smaller their separation will cause greater the attraction between the charges, which also increases the capacity for charge storage.

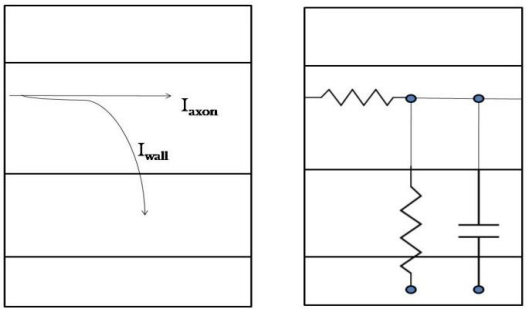
For a lipid bilayer, $\varepsilon = 5\cdot10^{-11}$ F/m (F is the symbol for Farad, the SI unit of capacitance) and d = 50 Å = $5\cdot10^{-9}$ m. Thus, the capacitance per unit area for an unmyelinated axon is

$$C/A = \varepsilon/d = 10^{-2}\ F/m^2 \quad \text{(unmyelinated axon)}$$

For myelinated axons, the myelin sheath contains a membrane that wraps around the axon a couple of hundred times. This multilayer arrangement increases the thickness of the lipid bilayer by a factor of 200 (1 μm total thickness), so capacitance per unit area for a myelinated axon is:

$$C/A = \varepsilon/d = 5X10^{-5}\ F/m^2 \text{ (myelinated axon)}$$

Summary of Electrical Properties



Physical System *Physical Model*

Fig.5. The physical model shows wires, two resistors, and a capacitor that approximate the physical flow of charge through real axons

The electrical properties of neurons are summarized by the diagram and tables below.

Table 1: Dielectric Properties of Neurons

	Unmyelinated Axon (UA)	Myelinated Axon (MA)
axoplasm resistivity	$\rho_{axoplasm} = 2.0\ \Omega\cdot m$	$\rho_{axoplasm} = 2.0\ \Omega\cdot m$
wall resistivity	$\rho_{UA} = 0.20\ \Omega\cdot m^2$	$\rho_{MA} = 40.0\ \Omega\cdot m^2$
wall capacitance/area	$C/A = 10^{-2}\ F/m^2$	$C/A = 5\cdot10^{-5}\ F/m^2$

IV. HODGKIN HUXLEY MODEL

The Hodgkin-Huxley model was developed by Alan Lloyd Hodgkin and Andrew Huxley in 1953 and they received the Nobel prize for this in 1963. This model characterizes the initiation and propagation of neural signals in giant axons of squids and describes very well the dynamic behaviour of channel kinetics. This model is a mathematic model which explains the experimental results of the voltage clamp experiment. With this mathematic model, the prediction of stimuli response is possible.

4.1 Voltage clamp experiment

In this voltage clamp experiment Hodgkin and Huxley measured the membrane voltage V_m by using an intracellular micropipette electrode and an electrode in the extra cellular fluid. They were able to control the V_m , also insert an external current for the purpose of generating an action potential in squid axons. They only could measure potential changes that means they could not detect influx or efflux currents [3].

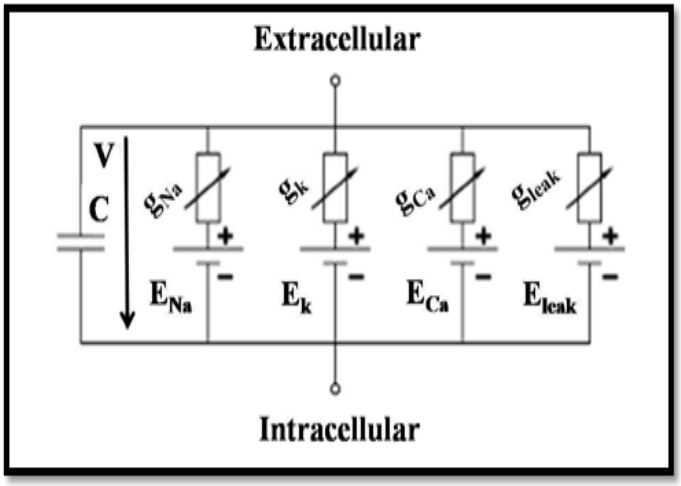


Fig. 6: Equivalent electric model for Hodgkin-Huxley Model

Here each component of an excitable cell has a biophysical analog, as shown in Figure 5.

4.2. Electric analog for cell membrane

The ions inside and outside a neuronal cell are separated through the cell membrane. The membrane behaves here like a capacitor. Discharging the capacitor corresponds to influx currents and charging is correspondence to efflux currents. Because of the different ionic concentrations of K, Na, etc inside and outside the cell, which are separated through the membrane and the closed ion channels, an electric potential difference between inside and outside layer occurs. This difference is known as membrane potential.

4.3: Electric analog for ion flows and ion concentrations

The ion flow through open ion channels because of different ion concentrations corresponds to electric flows. In squid axons ion flows for sodium ions, for potassium ions and a leakage ion flow exist, which are labeled with I_{Na}, I_K and I_L. The level of ion currents is membrane-voltage-dependent, so the ion currents obey the Ohms law. The higher the current is, the higher is the electric conductance. The g_{Na} is equal to the inward Na^+ influx and g_{Na} is equal to outward K^+ efflux and g_L stands for the leakage conductance. The currents and conductance are also shown in Fig.6 Furthermore in Fig. 6 the symbol E is displayed. E stands for an electric battery and represents the electrochemical gradient of the ion concentration and potential. Because this electrochemical gradient is the driving force for the ions it can be replaced with a battery E.

V. ACTIVITIES OF NERVOUS SYSTEM

The nervous system utilizes intricate networks of neurons to receive, process and exchange vast amounts of information. Neurons are able to respond to stimuli by generating and conducting electrical impulses, which are known as action potentials. These electrical signals can then be communicated to other neurons/ neural networks. The cellular membrane separates its internal environment from the external environment and regulates permeability of ions and molecules. The selectivity of the membrane allows exchanges of nutrients and wastes needed to sustain metabolism and allows passage of electrical currents created by the movement of ions through the membrane.

The cellular membrane is made of a thin polar membrane made up of two lipid layers with embedded integral proteins which span the membrane are the almost 2% of all membranes. These proteins provide structures for essential cellular function; including: channels, transporters, pumps, receptors, enzymes, structural support and many others. Proteins act as pumps, channels or transporters from one side of the impermeable membrane to the other. These proteins regulate the fluctuation of cellular ionic concentration and therefore the conduction of electrical current in and out of the membrane, which is fundamental to in neural communication [3, 4].

Ion channels are intrinsic membrane proteins which contain aqueous pores that can be opened or closed. When open, these channels permit selected ions one side diffusion in the membrane which creates the electrochemical gradient. While when closed, they are impermeable for these ions flow. Ion channels can allow single or multiple types of ions when opened. These ion channels can be identified by the type of stimuli that causes the channel gates to open or close. Some channels are opened by particular chemicals inside or outside the cell, such as neurotransmitters. Other channels may be sensitive to changes in voltage across the membrane, and still others may respond to various kinds of sensory stimuli [1].

Each ion channel also shows selectivity in the ions to which they are permeable. Some are constructed to only permit a specific type, such as sodium, potassium, chloride ions, etc. Others have fewer restrictions permitting broader groups of ions, such as mono-valent cations or all cations to pass through. These two characteristics, sensitivity and ion selectivity, are commonly used to describe and classify different types of ion channels. A specific category of channels, named as leak channels are selective ion channels which are always open, do not have gates and do not require stimuli. Sodium, potassium, chloride ions, etc all have individual channels, abundant throughout\ the body, which have prominent roles in neural firing and muscle contraction [4].

5.1 ACTIVE TRANSPORTERS

Active transporters are a class of intrinsic plasma membrane proteins which selectively transportations across the membrane and against their concentration gradient [1]. Active transporters bind with ions to form complexes, which are then trans-located across the membrane and released. This process takes several milliseconds causing ion translocation by active transport to be much slower than ion movement through ion channels. Actively transporting ions uphill also requires the consumption of energy. Neural transporters fall into two classes based on their energy sources: ATPase pumps and ion exchangers. ATPase pumps receive energy directly from the hydrolysis of ATP. Ion exchangers utilize the concentration gradients of other ions as an energy source to move a desired ion across the membrane. This type of transporter carries one ion up its electrochemical gradient while simultaneously carrying another ion down its electrochemical gradient. Abundant

examples include the Na+/K+ pump and the Na+/Ca+ exchanger. [1, 6, 8].

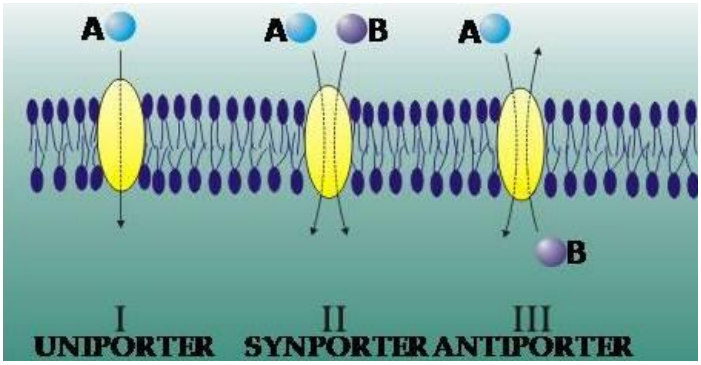


Fig 7: ATPase pumps and ion exchangers

5.2 RESTING POTENTIAL AND ACTION POTENTIAL

As we know, neurons are excitable cells which can generate electrical signals to respond stimuli. In order to achieve this phenomenon an electric potential is maintained across the cell membrane at rest. Without the resting membrane potential, no nerve impulse can occur. A nerve cell is considered in a state of rest or equilibrium when it is maintaining a consistent membrane potential and not generating any kind if of change in it . The electric potential across the membrane during this state is known as the resting membrane potential. The following are the factors which allow a nerve to maintain a resting potential across its membrane.

5.3 THE INTRACELLULAR AND EXTRACELLULAR ENVIRONMENTS

The intracellular and extracellular environments of human's neurons consist mostly of water, proteins, amino acids, phospholipids, inorganic ions and organic ions as shown below in figure ……. The intracellular proteins, phosphate groups of ATP and other organic molecules are negatively charged at the pH of the cell cytoplasm and impermeable to the membrane. This presence of non-diffusible negatively charged molecules make the intracellular environment negatively charged in comparison with surrounding extracellular environment. At rest, the intracellular senvironment has a high concentration of potassium ions and membrane impermeable anions, while the extracellular environment has a high concentration of sodium and chloride ions. These concentrations of chloride, potassium and sodium ions will remain consistent at rest and slightly fluctuate during an excited state [2, 4, 9].

Table 2: Mammalian intracellular and extracellular ionic concentration

ION	EXTERNAL (mM)	INTERNAL (mM)	PERMEABLE TO MEMBRANE
K+	5	125	YES
Na+	120	112	NO
Cl-	125	5	YES
A-	0	108	NO
H2O	55,000	55,000	YES

VI. FACTORS THAT MOVE IONS

6.1 Passive Forces

There are two forces which move ions and influence ionic equilibrium across the membrane without requiring external energy, the chemical concentration and electrical gradient. Together these two forces make up the electrochemical gradient and describe the ability of an ion to move across a membrane [3, 4, 9].

Ionic diffusion is a result of chemical concentration gradient. Diffusion is the random movement of particles from high concentration field to low concentration field. If the cellular environment of a neuron could be left unregulated, this force would move sodium and chloride ions into the cell and potassium out of the cell. However, the membrane repels this diffusive force, storing energy in the form a chemical potential across the membrane [1, 3, 9].

The ions exert electrostatic force on one another which generates electrostatic gradient. In a neural environment there is an extracellular excess of positive ions separated from the negatively charged intracellular fluid by the cellular membrane. A difference in charge across the cellular membrane causes an electrical gradient to exist across the membrane [1, 3, 9].

6.2 Active Forces

Diffusive and electrostatic forces transport ions according to the direction of the electrochemical gradient. However, to maintain a proper resting potential the membrane must also have the ability to move ions against the gradient. In neurons, Na+/K+ ATPase pumps help actively in transportation of sodium and potassium ions

against their electrochemical gradient to maintain resting potential [1,9].

6.3 Sodium–Potassium Pump

The sodium–potassium pump is a carrier protein which actively extrudes three sodium ions from the cell as it transports two potassium ions into the cell. This process is energy dependent because it acts against the sodium and potassium concentration gradients. Energy is provided in the conversion of ATP to ADP and Pi by the carrier protein ATPase enzyme [1]. Each nerve cell has leak channels which passively allow sodium or potassium to flow opposite to their concentration gradient. The cell membrane has many potassium leak channels, which allow potassium ions to leave the cell. The sodium-potassium pump counteracts this effect by restoring potassium into cell. This counteraction of the sodium-potassium pump permits nerve cells to have a relatively constant intracellular concentration of sodium and potassium ions and a constant membrane potential [1].

6.4 Resting Membrane Permeability

The concentration differences between ions on the inside and outside of the cell generate potential across the membrane and influence the direction in which ions want to flow. However, ionic mobility is limited by the permeability of the cell membrane to specific ionic species. Together, the electrochemical gradient and membrane permeability influence ionic fluctuation and distribution across the membrane. The most influential membrane permeable ions in nerve communication are sodium and potassium. The cellular membrane is made up of more potassium channels than any other type of ion channel, which also makes it the most permeable to potassium [4, 9].

Sodium and potassium ion channels are highly selective of the ions that can pass through. Each channel protein spans the lipid membrane, with an aqueous pore connecting the external medium to the internal cytoplasm [3, 7].

6.5 Membrane conductance

Membrane conductance of a particular ion depends on ion's ability to carry current across the membrane. Conductance quantifies the ease at which a particular ion responses to a change in membrane potential. Conductance is analogous to the reciprocal of the resistance of an electrical circuit to current flow, as shown below:

$$i_k = G_k\,(E_m - E_k)$$

Here

G_k is the conductance of the membrane to potassium ions, measured in Siemens.

$(E_m - E_k)$ is the driving force which governs net movement of potassium ions across the membrane, measured in Volts, and i_k is the outward membrane current, measured in Amperes.

Similar equations can be written for sodium and chloride.

$$I_{Na} = G_{Na}\,(E_m - E_{Na})$$

The membrane conductance of an ion is very closely related to its membrane permeability, but is not identical. Conductance is proportional to the rate at which the specific ions are crossing the membrane; this rate is depends on both permeability and the number of available ions [4]. Membrane permeability remains consistent in the presence of low and high concentrations of permeable ions, while conductance varies according to the concentration of permeable ions present. Together, these membrane properties provide helpful insight when evaluating the state ionic flow.

Concentration gradients cause an unequal distribution of ionic charge that generates a bioelectrochemical potential across the membrane, similar to a tiny battery with the positive pole outside of the cell and the negative pole inside. Concentration gradients are maintained by homeostatic processes such as the sodium-potassium pump [1, 3, 4].

VII. Equations

7.1 Nernst equation

The equilibrium potential of an ion is described by the Nernst Equation:

$$E_{ion\,x} = \frac{RT}{zF}\,ln\,\frac{[Xion]out}{[Xion]in}$$

Table 3: Axon ion concentration and their Nernst's Potentials

ION	EXTERNAL (mM)	INTERNAL (mM)	NERNST POTENTIAL (Mv)
K+	20	400	-75
Na+	440	50	55
Cl-	560	40	-66

7.2 Goldmann equations

The Goldmann equation expresses the contributions of ionic concentrations and membrane permeability on membrane potential. The Goldmann equations describing a membrane which is permeable to potassium, sodium and chloride can be written as:

$$Vm = \frac{RT}{F} ln \frac{P_K[K^+]_{out} + P_{Na}[Na^+]_{out} + P_{Cl}[Cl^-]_{out}}{P_K[K^+]_{in} + P_{Na}[Na^+]_{in} + P_{Cl}[Cl^-]_{in}}$$

Here P_{ion} is the membrane permeability to a particular ion. Both the Goldmann and Nernst equations serve as useful tools in determining nerve cell characteristics from intracellular and extracellular ionic concentrations.

VIII. EQUIVALENT ELECTRIC MODEL

Neurons are electrically excitable cells. This characteristic allows neurons to respond to stimuli by generating and conducting electrical impulses, known as action potentials. Action potential generation facilitates the onward propagation of nervous information to other neurons. This process provides the basis for all nervous communication.

8.1 Action Potential

The cell body's dendrites are a common postsynaptic site for coupled presynaptic neurons. These presynaptic neurons provide incoming information which is either excitatory or inhibitory in nature. The dendrites pass all incoming information to the cell body via electrical impulses where, if sufficient stimulation occurs, an action potential is generated. This action potential is conducted form the "trigger zone" in the cell body down the axon to the axon's ending terminal. An action potential reaching the axon terminal endings will trigger the release chemical or electrical signals into the synaptic gap. The neuron releases these signals from the presynaptic side of the synaptic gap; these signals now travel across the synapse onto the coupled neuron's postsynaptic site, restarting the entire process again.

8.2 Generation of an Action Potential

When a stimulus current of a sufficient magnitude is passed through the cellular membrane of an electrically excitable cell, a change in membrane potential, which is called an action potential, is generated. However, if a stimulus current is not of sufficient magnitude to generate an action potential, a smaller change in membrane potential, called a graded potential, is generated. Thus, nerve cells have capacity to generate a graded potential or action potential which is dependent on stimulus current magnitude [5]. The mechanism of generation of these potentials is also different. Graded potentials cannot activate voltage-gated ion channels and are therefore subjected to a dependent continuous relationship between stimulus magnitude and membrane potential. Action potentials drastically change membrane permeability by activating voltage-gated ion channels, allowing a discontinuous relationship between stimulus magnitude and membrane potential [5]. The membrane response during a graded potential and action potential is shown in Fig 8.

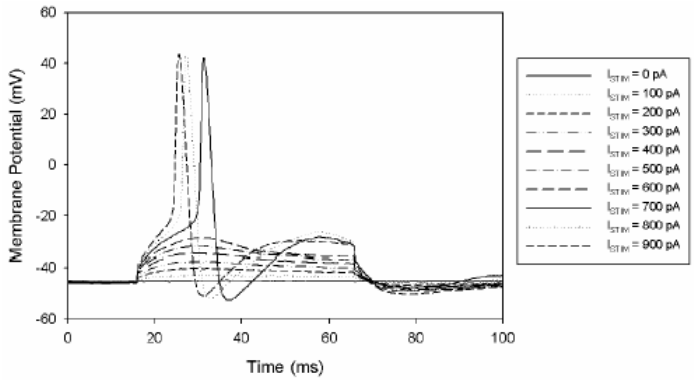


Fig 8: Graded and Action potential

The distribution in charged ions across the cell membrane fluctuates during the progress of an action potential; it generates change in trans-membrane voltage described in Fig 8. This fluctuation is due to shifts in ionic permeability and results in ionic movement.

Intervals of membrane permeability change can be divided into four basic phases: rest, depolarization, repolarizaton, and hyper-polarization. The changes in ion permeability during each phase are a result of activation and deactivation of voltage-gated ion channels [5, 10].

8.3 Voltage-Gated Ion Channels

Voltage-gated ion channels are those ion channels which open or close with changes in transmembrane potential. Voltage-gated sodium channels respond to stimulus currents to initiate an action potential; once an action potential appears voltage-gated sodium and potassium channels continue these changes in ion permeability until the membrane is repolarized back to resting potential [3].

These channels have gates which regulate ions flow. Voltage-gated sodium channels have two independent gates, which are known as the m and h gate. The m gate (also known as sodium activation gate) is located on extracellular side of the membrane protein and is closed at resting potential. The h gate (or sodium inactivation gate) is located on the intracellular side the membrane protein and is open at resting potential. The n gate (or potassium activation gate) is located on extracellular side of the membrane protein and is closed at resting potential. These gates respond to particular changes in membrane voltage and open or close at different rates [3,4]. The response of these channels and their gates during different phases of an action potential in a human neuron is briefed below.

8.4 Phases of an Action Potential

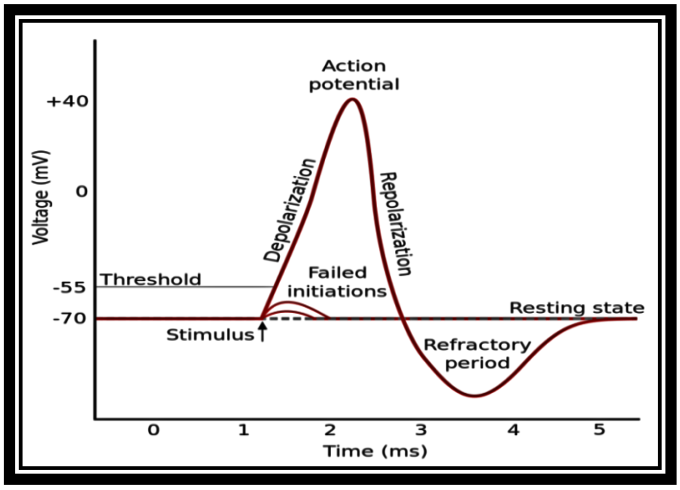


Fig 9: Phases in an Action Potential

8.4.1 Resting phase

The resting phase occurs when the nerve cell is in a steady state. It maintains resting membrane potential. In this state, the cell has the capacity to respond to sufficient stimulus current and generate an action potential. The resting potential of a human neuron nerve cell exists between the Nernst potential for potassium (-80mV) and the Nernst potential for sodium (+58mV). Potassium's potential has a significant influence on resting potential because of its dominance in membrane permeability over sodium. During this phase, the m gate and n gate are closed while the slow h gate is open [4].

8.4.2 Depolarization

The depolarization phase (or rising phase) occurs once when a stimulus current of sufficient magnitude flows through the cellular membrane of a nerve cell in resting state. This stimulus current increases membrane potential above the threshold. The threshold is the value of the minimum membrane potential which generates an action potential. Once threshold is reached, the fast acting m gate rapidly opens, the slow acting n gate begins to open and slow acting h gate start to close. This change in membrane sodium permeability allows extracellular sodium to flow down its electrostatic gradient, into the negative intracellular environment of the neuron. This massive sodium influx increases the membrane potential to a peak near sodium's Nernst potential, marking the end of the depolarization phase [4].

8.4.3 Repolarization (Refractory)

As the membrane potential reaches its maximum, the repolarization phase, or falling phase, begins. The slow acting h gate closes, inactivating the inward flux of sodium ions into the cell and ceasing the increase in membrane potential. Now the potassium channels' slow n gate is fully open, which allows potassium ions to flow down their electrostatic gradient from the newly positive intracellular environment of the neuron and into the more negative extracellular environment. The potassium n gate repolarizes the cell membrane potential, remaining open until resting potential is reached [4].

8.4.4 Hyperpolarization

During the repolarization, phase the membrane potential becomes more negative due to the efflux of intracellular potassium ions. The membrane potential continues to decrease and undershoots resting potential while the n gate fully closes and h gate beings to open again [4].

IX. PROPERTIES OF AN ACTION POTENTIAL

9.1 Threshold

To generate an action potential, a stimulus current must have sufficient strength to elevate membrane potential from resting potential to threshold potential. A nerve cell's threshold potential is the minimum value of membrane potential that will produce an action potential. In case if a stimulus current is not of sufficient magnitude to reach the threshold potential then a graded potential is generated [5].

Graded potentials open a small portion of voltage-gated sodium channels, slightly increasing sodium permeability. However, the resulting influx of sodium ions due to gradded potential cannot overcome the opposing efflux of potassium ions. A net outward membrane current is produced, ceasing further depolarization and possible generation of an action potential. At threshold potential, the minimum amount of voltage-gated sodium channels becomes open in order to overcome the efflux of potassium ions and produce a net inward current. This permits further depolarization causing sodium influx to dominate, resulting in an action potential [5]. The value of threshold potential for a particular neuron is influenced by the density of voltage gated sodium channels in the plasma membrane and the sensitivity of those channels. A high density of voltage-gated sodium channels will surely require a smaller portion of channels to open to generate an influx of sodium which overcomes potassium efflux. An increased sensitivity in voltage-gated sodium channels will also requires a smaller portion of channels to open to generate an influx of sodium which overcomes potassium efflux. The lower the threshold potential, the more excitable a neuron is to incoming nerve impulses and the more actively it propagates signals to other neurons [3].

9.2 Summation

Neurons receive postsynaptic potentials from many other neurons, located at the synapse sites. To generate an action potential, these signals must generate a resultant potential exceeding the threshold potential when reaching the neuron's triggering zone. These individual postsynaptic potentials decay with time and space when traveling from the synapse sites to trigger zone. However, in certain conditions, postsynaptic potentials can summate spatially or temporally to combine potential and combat decay [11, 12].

Spatial summation occurs when graded postsynaptic potentials, generated at different synapse locations, occur within a single space constant of another (The space constant is the distance a particular potential will occupy before it decays). Similarly temporal summation takes place when graded postsynaptic potentials occur within a single time constant of another (The time constant is the amount of time a particular potential will take to decay). Potentials which summate combine amplitudes to form a single impulse with an elevated magnitude. This elevation expands the distance postsynaptic potentials can occupy before decaying to zero. Summation of postsynaptic potentials increases the ability of a neuron to produce a resultant potential which exceeds threshold at the trigger zone and generate an action potential [12]. Spatial and temporal summations are demonstrated in Fig.9 and Fig. 10 respectively.

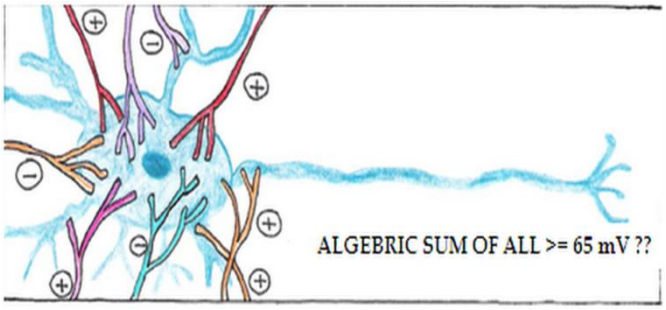


Fig 10: An action potential is generated if the spatial and temporal sum of all exxcitory and inhibitory connections reaches to the threshold value

9.3 The Refractory Period

Immediately following the occurrence of an action potential, a nerve cell has a reduced ability to generate a second action potential and is said to be refractory. For a brief interval, known as the absolute refractory period, a second action potential cannot be generated by any magnitude of stimulus. For intervals which are greater than the absolute refractory period, there is the relative refractory period in which a second action potential can be produced, but the threshold potential is elevated. Threshold elevation is dependent on a complex history of previous stimulation and also on response cycles of the axon. Absolute refractory periods occur on the order of a few milliseconds, while relative refractory periods have shown lasting effects, up to many minutes [5]. Together absolute and relative refractory periods limit the maximum frequency at which neurons can conduct information.

X. AFFECTS OF EXTERNAL EM SIGNAL ON NERVE CONDUCTION

10.1 Response time

Matlab® Simulation of Hodgkin-Huxley model considering external EM signal embedded with the membrane potential shows effective increment in the response time with increment in frequency of the External Signal [11]. It shows that propagation of information through a nerve is not independent of the external field. This simulation show delay in the reaction to any stimuli that happens on human body.

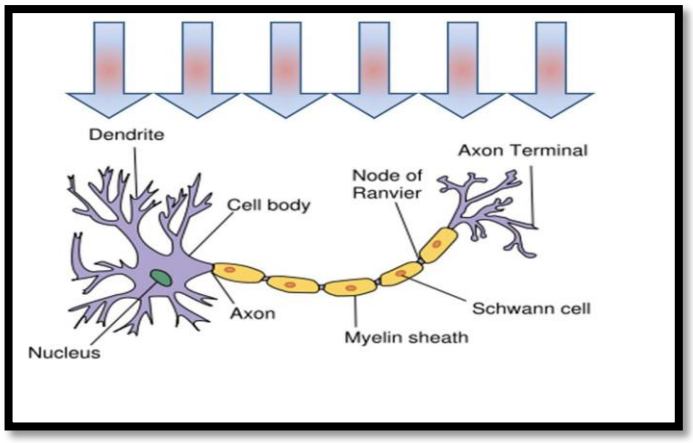


Fig. 11: Nerve under continuous exposure of external EM Field

10.2 Action potential

It is clear from the simulation results that there is a significant decrement in the peak of the membrane potential with increment in the frequency. This result may tend to the loss in the information which actually supposed to be reached to the brain. Loss of the information leads to non-proper response or non-response situation for the brain.

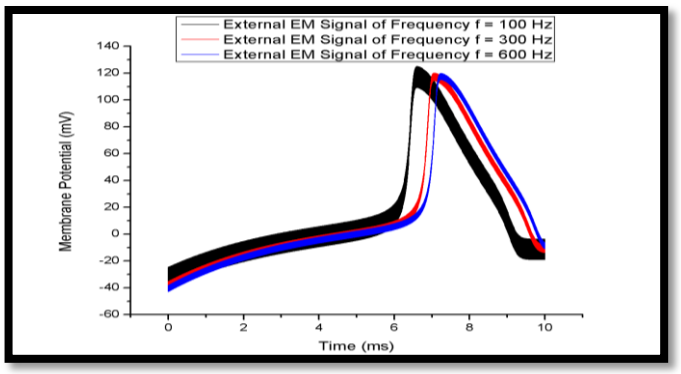


Fig 12: Affects of the external EM signals of different frequencies on human nerve conduction

XI. CONCLUSION

Nerve conduction is an essential process of a living body. Its mechanism can be understood with well established Hodgkin-Huxley model. Propagation of information through a nerve is an electrical process which is explained in this paper. Effects of the external environment signals on this process are still not reported but Simulation results of combined effects of external signal along with the membrane potential show that it is being affected under the continuous exposure of EM radiations surrounding us.

XII. FUTURE WORK

Experimental results of these simulation conditions are key factor to verify the relation and identification of pro and cons of these effects. Authors are working in this direction so that it can be established whether these external radiations really affects the working of human nervous system or not.

Controlled external signals for specific requirements may result as solution of some biomedical problems too. This can be done only after once we know exact relationship between external signal and behavioral response of nervous system for it.

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