Biophysical basis for the mechanism of action potential initiation in presence of static magnetic fields

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***Abstract*—** A biophysical model accounting for the experimentally-observed response of neurons to a low-level static magnetic field is proposed. This model explains the reduced excitability observed in neurons exposed to steady magnetic flux densities of the order of 10mT. In presence of a steady magnetic field reduced excitability is manifested as an increase in the excitation threshold associated with the neural membrane. The separation of charge resulting from the Lorentz force exerted on moving intracellular ions leads to formation of an electric field in a direction perpendicular to that of action potential transmission. As a result, the ionic displacement current available for discharging the membrane capacitance and bringing the transmembrane potential to the threshold level is diminished. The quantitative approaches undertaken in order to validate the proposed model were aimed at determining the change in the excitation threshold in presence of a static magnetic field. To this end applicability of the Rattay's modified cable equation was assessed, and the variation of the transmembrane potential corresponding to the excitation threshold was examined based on recently-proposed generalizations of the Hodgkin and Huxley (HH) model accounting for action potential initiation. A further generalization of the HH model is also proposed which allows modeling the response of a neuron to a magnetic field of arbitrary time dependence. Accordingly, an expanded equivalent circuit model for the neuronal membrane with a magnetic field-dependent source term is derived from the generalized HH model. The proposed model is shown to be consistent with the biophysical basis for the dynamical mechanism of action potential initiation in class 3 pain neurons.

***Keywords*—**Action potential, Hall Effect, neuron, steady magnetic field.

I. Introduction

PUBLIC concern about the health impact of magnetic fields has persisted in the past thirty years in an unabated fashion [1],[2],[3]. Nevertheless, applications of magnetic fields in clinical medicine have continued to expand. Magnetic resonance imaging (MRI) [4], now serves as a routine diagnostic tool, although steady magnetic fields in the range of tens of Tesla have long been known to constitute a serious health hazard to living organisms [5]. Aside from MRI [6], diagnostic applications of magnetic fields encompass the use of SQUID (superconducting quantum interference device) probes to identify magnetic fields generated by cardiac [7] and neural tissues [8],[9]. Applications of magnetic fields for therapeutic purposes consist of field-induced inhibition of pain [10] as well as magnetic stimulation of neuromuscular tissue [11],[12]. In the meantime, fundamental investigations elucidating the biological effects of magnetic fields have led to a deeper understanding of the influence of magnetic fields at the cellular level [13].

While numerous studies have been conducted over a period of roughly three decades [14], [15], [16] to explain the influence of a constant magnetic field on action potential (AP) generation and conduction, a satisfactory quantitative model for the response of neuron to relatively small steady magnetic fields is yet to be advanced. While theoretical investigations have suggested that constant magnetic flux densities in the 25-100T range would be required to affect ionic currents flowing in nerve processes [14],[17], experimental studies have indicated that electrically stimulated action potentials generated in adult mouse sensory neurons present in cell cultures are to a large degree blocked in neurons positioned in a static magnetic flux density of 11*m*T [15]. Reduced excitability in the form of a decrease in the amplitude of the action potential has also been reported in isolated rat sciatic nerve in presence of a 50 Hertz, 1*m*T magnetic flux density [18]. Recently, a biophysical model for the reduced excitability in neurons exposed to steady magnetic fields has been presented [19],[20]. The slight redistribution of ionic charge resulting from the magnetic force acting on the mobile ions in the intracellular space has been introduced as the origin of a Hall electric field in a direction perpendicular to AP transmission along the axon. The Hall electric field, in turn, gives rise to a transient current density flowing in a direction perpendicular to the direction of the depolarizing current responsible for bringing the membrane potential to the action potential threshold level. The current density associated with the Hall electric field may constitute a relatively small fraction of the total stimulating current density available for depolarizing the membrane in response to the electrical stimulation of the neuron. It has been shown, however, that relatively small magnetic fields may cause a sufficient reduction in the stimulating current leading to AP blockade.

In this work the appropriate quantitative approach for modeling the response of a neuron to magnetic fields will be presented. The validity of the proposed approach is verified by demonstrating its utility in development of a quantitative biophysical model justifying the experimentally observed

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suppression of action potential in presence of small magnetic fields in the *m*T range. Based on the proposed quantitative approach, the equivalent circuit model of the neuronal membrane has been expanded to account for the reduced excitability of neurons exposed to a steady magnetic field.

II. Biophysical Model

A first order analysis on the excitability of neuron can be performed by considering the passive response of a length Δx of the axonal membrane, which can be modeled by the equivalent circuit of Fig. 1.

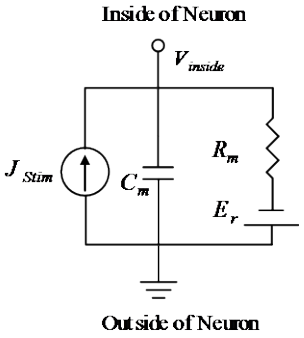

Fig.1 Equivalent circuit model of the neuronal plasma membrane characterizing the passive response of the neuron to electrical excitation in the form of a depolarizing current

This equivalent circuit includes an electromotive force, E_r, which represents the resting membrane potential, the electrical resistance of the membrane, R_m and the capacitance per unit area of the membrane, C_m . The excitability of neuron can be characterized by the magnitude of the depolarizing current necessary to bring the transmembrane potential, V_m to the threshold level necessary to initiate the action potential. In response to a depolarizing transient current density (current per unit cross-sectional area) J_{stim} the membrane capacitance is discharged giving rise to a rate of change of V_m given by

$$\frac{dV_m}{dt} = \frac{J_{Stim}}{C_m} \tag{1}$$

The typical value of the neuron excitation threshold, i.e. the required depolarization of the membrane potential for initiation of the action potential, is approximately $15mV$, which occurs over a typical time interval of $2msec$. Therefore, considering the typical value of $1\mu F/cm^2$ for the membrane capacitance, the discharging current density for generation of action potential can be estimated as follows:

$$J_{Stim} = C_m \frac{dV_m}{dt} \cong C_m \frac{\Delta V_m}{\Delta t} = 1\frac{\mu F}{cm^2} \cdot \frac{15mV}{2m\sec} = 7.5\frac{\mu A}{cm^2} \tag{2}$$

For current densities higher than the typical value estimated above, the voltage-dependent sodium channels in the plasma membrane become activated leading to further depolarization of the membrane and initiation of the action potential.

The reduced excitability of neurons exposed to a constant magnetic field can be explained by a decrease in the magnitude of the depolarizing current available to bring V_m to the threshold level. The Hall Effect provides a plausible explanation for the reduction of the depolarizing current in presence of a constant magnetic field. According to Lorentz law, when exposed to a magnetic field, moving charged particles experience a force proportional to the product of their velocity and the magnetic flux density. The influence of a magnetic field on the function of neuron can be explained based on the Lorentz force acting on the ions present in the intracellular environment. The source of ionic motion is the local electric field resulting from the electrical stimulation of neuron, which leads to flow of a drift current. In presence of a magnetic field these ions will also be subject to an induced electric field known as the Hall electric field. In particular, the slight redistribution of charge associated with the Lorentz magnetic force exerted on moving intracellular ions leads to formation of an induced Hall electric field in a direction perpendicular to that of action potential transmission along the axon. Therefore, as shown in Fig. 2 with a normal magnetic field B_z, under the conditions of electrical excitation, the transient ionic current density J_x, which is available for discharging the membrane capacitance, is reduced due to partial drift of ions along the direction of the Hall electric field (i.e. out of the page along the +y direction in Fig. 2 for positive ions).

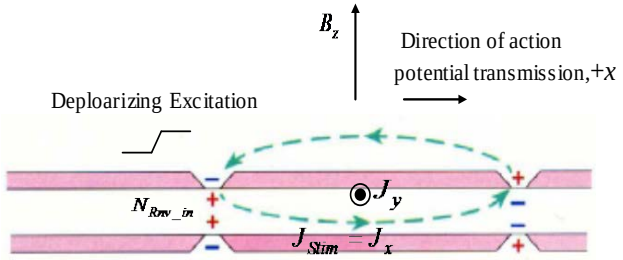

Fig.2 Influence of a static magnetic field on transmission of action potential along the axon

The general expression for the ionic drift current density J flowing in presence of an electric field E is given by

$$J = qnv_d = q\mu nE \tag{3}$$

where q, μ, n, and E represent the electronic charge, the ionic mobility, the density of the given ion, and the electric field respectively, and $v_d = \mu E$ denotes the drift velocity. Based on (3), the depolarizing current density J_x is given by

$$J_x = qn_{\text{int}}v_x \tag{4}$$

where n_{int} denotes the concentration of the intracellular ions carrying the depolarizing current and v_x is the drift velocity in the direction of action potential propagation. Given the expression for the Lorentz force $F_M = qv_xB$, the induced Hall electric field, will be represented by

$$E_y = F_M/q = v_xB \tag{5}$$

Therefore, the drift current associated with the Hall electric field, J_y, which flows in a direction perpendicular to the action potential transmission can be expressed as

$$J_y = q\mu_y n_{\text{int}}E_y = q\mu_y n_{\text{int}}v_xB \tag{6}$$

where μ_y denotes the transverse ionic mobility in a plane associated with the cross sectional area of the axon. From (4)

and (6), the ratio $\alpha=J_y/J_x$ characterizing the deflection of the depolarizing current is given by

$$\frac{J_y}{J_x} = \mu_y B_z = \alpha \tag{7}$$

α represents a dimensionless current segregation ratio. The segregation ratio J_y/J_x characterizes the segregation of the transient depolarizing current into a component flowing in the direction perpendicular to action potential transmission, J_y, and a component corresponding to the actual depolarizing transient current density J_x flowing parallel to the transmission direction.

The validity of the proposed model for the influence of a constant magnetic field on the excitation threshold of neuron has been demonstrated based on simulation of the neural transmembrane potential [19]. By implementing the Hodgkin-Huxley model equations in MATLAB™, the public-domain software HHSim [20] allows the behavior of the neuronal membrane to be simulated in response to a variety of stimuli. This graphical simulator, which provides full access to the Hodgkin-Huxley model parameters, permits application of a depolarizing current to a segment of the axon. As shown in Fig. 3, simulation of a neuronal membrane segment in response to two depolarizing current stimuli indicated that a relatively small reduction in the amplitude of the stimulus current resulting from presence of a magnetic field in the 10mT range may suppress action potential generation. The increase in the neuronal excitation threshold is basically equivalent to a decrease in the amplitude of the depolarizing current. Specifically, as suggested by (4), based on the proposed model it is expected that the amplitude of the stimulating current is reduced in proportion to the magnitude of the applied magnetic flux density. Therefore, in agreement with experimental observations [15], the simulation result indicated that relatively small magnetic flux densities may reduce excitability.

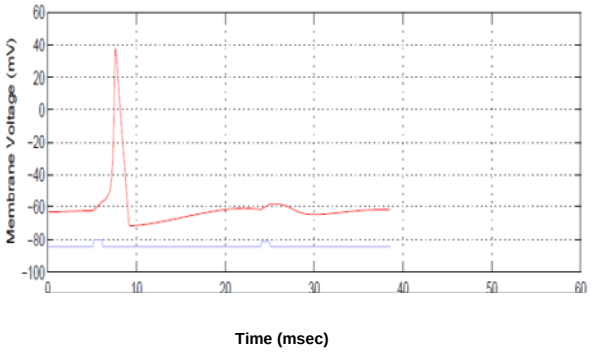

Fig.3 Transmembrane potential of a segment of neuron in response to two depolarizing current Stimuli

III. Modeling The Response Of Neuron to A Magnetic Field

A. Modified Cable Equation

A more rigorous quantitative approach to modeling the influence of magnetic fields on the excitability of neuron can be adopted by considering the electrotonic response of an axon to extracellular stimulation characterized by the cable equation. Since the cable equation represents the passive response of the membrane, the contribution of the active, nonlinear, voltage-sensitive ion channels are ignored in this approach. An efficient approach for prediction of the extracellular stimulation response, introduced by Rattay [21], employs the concept of activation function to derive a modified form of cable equation given by

$$\lambda_m^2 \frac{\partial E_x(x,t)}{\partial x} = -\lambda_m^2 \frac{\partial^2 V_m(x,t)}{\partial x^2} + \tau_m \frac{\partial V_m(x,t)}{\partial x} + V_m(x,t) \tag{5}$$

where λ_m and τ_m denote the length and the time constants of the neural membrane respectively, which are defined as:

$$\lambda_m = \sqrt{\frac{R_m}{R_i}} \tag{6}$$

$$\tau_m = R_m C_m \tag{7}$$

with R_i representing the intracellular resistance. Here, E_x is the x component of the electric field associated with extracellular electrical stimulation and/or a magnetically induced electric field. The $\lambda_m^2\, \partial E_x/\partial$x term is defined as the activating function allowing estimation of the initial change in the transmembrane potential, V_m [21]. Essentially, the activating function permits evaluation of stimulation based on λ_m and the spatially varying electric field [22], characterized by the $\partial E_x /\partial x$ term. The magnitude of the $\partial E_x /\partial x$ term in the direction of the fiber extension can be regarded as responsible for activation of the axon [23].

For a magnetic field perpendicular to the axon (direction of AP transmission) as depicted in Fig. 2, however, the magnetically induced Hall electric field is perpendicular to direction of fiber extension. Therefore, the modified cable equation cannot shed light on extracellular magnetic stimulation in this situation. Considering a more general direction for the magnetic field, however, Rattay's modified cable equation can be employed to assess the influence of magnetic field on the change in V_m. A more accurate analysis based on this approach to modeling the response of axon to magnetic stimulation, nevertheless, requires numerical simulation of the solutions to the modified cable equation.

B. Generalization of Hodgkin and Huxely Model

The role of nonlinear, voltage-dependent ion channels in establishing the onset potential, defined as the membrane potential at which an AP is triggered, becomes important around threshold. The original interpretation of Hodgkin and Huxley captured by the equivalent circuit model of the neural membrane, models the nonlinear ion channels as variable conductances. The dynamics of the transmembrane potential V_m of a spatially homogeneous section of neuron in presence of a stimulating current $I_{Stim\text{-}eff}$ injected into the cell are given by the following differential equation [24]:

$$C_m \frac{dV_m}{dt} = \frac{1}{A} \cdot (I_{Stim-eff} - I_{Na^+} - I_{K^+} - I_M - I_L) \tag{8}$$

where A is the membrane area, and

$$I_{Na^+} = g_{Na^+} P_{Na^+} (V_m - E_{Na^+}) \tag{9}$$

$$I_{K^+} = g_{K^+} P_{K^+} (V_m - E_{K^+}) \tag{10}$$

$$I_M = g_M P_M (V_m - E_{K^+}) \quad (11)$$

$$I_L = g_L (V_m - E_L) \quad (12)$$

$$I_{Stim-eff} = I_{Stim-net} + I_{syn} \quad (13)$$

Here $I_{stim\text{-}net}$ is the net stimulating current and I_{syn} is the current originating from synaptic background activity [25]. To model the background activity synaptic conductances are typically assumed to be stochastic consisting of an excitatory conductance (g_e) with reversal potential E_e and an inhibitory conductance (g_I) with reversal potential E_I, thereby giving I_{syn} as [26]

$$I_{syn} = g_e (V_m - E_e) + g_I (V_m - E_I) \quad (14)$$

I_{Na^+}, I_{K^+}, and I_M are the currents passing through the voltage-gated sodium, potassium, and M-type potassium channels respectively and the leakage current I_L is the current flowing through the passive ion channels, g_{Na^+}, g_{K^+}, and g_M are the maximal conductance of the voltage-sensitive sodium, potassium, and the M-type potassium channels respectively, and g_L is the effective conductance associated with all passive ion channels, P_{Na^+}, P_{K^+}, and P_M represent the probability that the sodium, the potassium, and the M-type potassium channels are open respectively, and E_{Na^+}, E_{K^+}, and E_L denote the reversal potentials associated with the sodium, potassium, and passive ion channels respectively. During initiation of the AP, the dominant role is played by the sodium channels. The M-type potassium channel may play an inhibitory role during AP initiation by raising the threshold for spike generation. The potassium channels, however, respond too slowly for their dynamics to impact the membrane potential. Nevertheless, the M-type potassium channel is unique because it is open at rest and even more likely to be open during depolarization.

The onset span can be regarded as a measure of the variability of the voltage threshold for action potential initiation [24]. Presence of a stochastic synaptic background activity represented by the current I_{syn} leads to a distribution of voltages at which the voltage threshold is reached; the width of this distribution represents the onset span [24]. The onset of action potential occurs when V_m reaches a threshold value V_{TH} such that below V_{TH} the transmembrane potential exhibits relaxation towards the resting membrane potential while above V_{TH} an action potential is fired.

The essential framework of Hodgkin-Huxley models for action potential generation, which is captured by (8)-(12) is appropriate for modeling the response of neuron to extracellular stimulation at threshold. However, generalizations of the Hodgkin and Huxley model, such as those represented by (13) and (14) may be necessary to account for experimental observations. For example, accounting for the inverse relationship between the onset span and onset rapidity, defined as the rate at which the membrane potential increases, requires inclusion of the synaptic background activity in the model [24].

Influence of the magnetic field on AP initiation can also be modeled through generalization of the Hodgkin and Huxley model. Based on the proposed explanation for the reduced excitability of neuron in presence of a steady magnetic field, failure to reach the excitation threshold is the result of a decrease in the amplitude of the stimulating current in proportion to the magnetic flux density. This effect can be modeled by adding a $-I_{Mag}=-\mu_y B_z I_{Stim}$ term to the right handside of (8).

IV. Expansion of Neuronal Membrane Model

In the equivalent circuit model for the neuronal membrane the influence of a constant magnetic field can be represented using a dependent current source whose magnitude is proportional to the magnetic flux density. The expanded equivalent circuit model for the neuronal membrane is shown in Fig. 4.

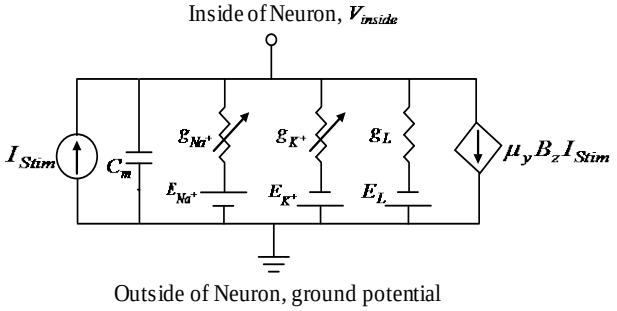

Fig.4 Expandend Equivalent Circuit Model of Neuronal membrane in Presence of Magnetic Field

This model is based on the equivalent circuit representation of the original Hodgkin and Huxley model in which the semi-permeable membrane is considered to be equivalent to a membrane capacitance separating the intracellular and extracellular fluid compartments. The voltage-dependent ion-channels are represented by variable resistances through which C_m is discharged and charged in the course of the action potential. The conductance g_L represents the effective static conductance associated with all passive ion channels and essentially models a leakage ionic current. And, the reversal potentials are represented by batteries. To account for the reduced excitability in presence of a magnetic field a magnetic-field-dependent current source with a magnitude of $\mu_y B_z I_{Stim}$ is included whose polarity is opposite to that of the stimulating current, I_{Stim}. In the expanded neuronal membrane model, therefore, the influence of the magnetic field is represented by a current-controlled current source (CCCS).

V. Compatability with Dynamical Mechanism of AP

A critical process in neural coding involves transduction of graded synaptic input into trains of all-or-none APs. Based on qualitative differences in analog-to-digital transduction properties, Hodgkin classified neurons into three distinct groups. The essential features of the spike initiation process are captured by the Hodgkin's classification, since it furnishes a fundamental account of the analog-to-digital transduction on the time scale of a single inter-spike interval (ISI). A general explanation of the biophysical basis for this classification has been recently advanced by Prescott *et al.* [27] according to which, Hodgkin's three classes of excitability represent

different outcomes in a nonlinear competition between oppositely-directed, kinetically-mismatched currents. The minimal model employed by Prescott and coworkers elucidates the biophysical basis of dynamically distinct mechanisms responsible for initiation of action potentials in each of the Hodgkin's classes. In this section the proposed model for the influence of a magnetic field on neuronal response is demonstrated to be consistent with the biophysical basis for the dynamical mechanism of action potential initiation.

The minimal two-dimensional (2D) model adopted by Prescott *et al.* [27] was derived from the Morris-Lecar model [28] by employing a slower recovery variable *w*, and a fast activation variable $m_\infty(V_m)$ associated with the transmembrane potential leading to the following system description:

$$C\frac{dV_m}{dt} = I_{Stim} - g_{fast}m_\infty(V_m)(V_m - E_{Na^+}) - \quad (8)$$
$$g_{slow}w(V_m - E_{K^+}) - g_L(V_m - E_L)$$

with *C* denoting the membrane capacitance in Farads, $C=AC_m$.

In the resulting 2D model, the voltage V_m controls instantaneous activation of fast currents (I_{fast}) such as those associated with the voltage-gated sodium channels and *w* is a function of voltage and controls activation of slower currents (I_{slow}). Both I_{fast} and I_{slow} may consist of more than one component. I_{slow} may, for example, be split into its component parts which include the delayed rectifier K^+ current, $I_{K,dr}$ and a subthreshold current, I_{sub} which may be either inward or outward. To reduce the dimensionality of the model, currents with similar kinetics are lumped together in the 2D model. Despite the minimalist framework adopted by Prescott *et al.* [27], this simple 2D model displays all of Hodgkin's three classes of excitability and permits distinguishing one class of excitability from another. The oppositely-directed, kinetically-mismatched currents and their relative magnitudes determine the dynamics of excitability in each class as follows.

Class 1 excitability has been ascribed to presence of a net inward (depolarizing) current at perithreshold potentials under steady state conditions [27], which gives rise to slow firing in response to relatively weak stimuli. In essence, absence of a slow-activating outward current at voltages below threshold causes the inward current to face no competition leading to arbitrarily slow spiking.

According to Hodgkin's classification, Class 2 neurons are incapable of maintaining spike generation below a critical frequency. Even with net current being outward (hyperpolarizing) at steady state, spike initiation can occur in Class 2 neurons because inward current may activate faster than the outward current [27]. While fast-activating inward current can guarantee repetitive spiking beyond a critical frequency, spiking cannot be maintained below a rate providing sufficient time for slow-activating outward current to drive the net current outward during the ISI.

Based on Hodgkin's classification, Class 3 neurons do not initiate spikes repetitively, typically firing only once at the onset of stimulation. Class 3 excitability has been attributed to the condition where fast-activating inward current overpowers slow-activating outward current during a stimulus transient [27], in spite of the slow-activating outward current dominating during constant stimulation. In Class 3 excitability, therefore, the outward current is sufficiently strong to suppress repetitive spiking despite presence of a fast-activating inward current. Spike initiation is only possible when the system is driven out of steady state, e.g. during a stimulus transient, when fast-activating inward current initiates a spike before slow-activating outward current finds the opportunity to counteract the positive feedback process.

In summary, Hodgkin's three classes of excitability arise from different outcomes in the course of a competition between fast- and slow-activating currents. The kinetic mismatch between currents is a critical determinant of single-spiking as occurs in class 3 excitability, or repetitive spiking faster than a critical frequency despite the net steady state current being outward at threshold (class 2 excitability).

As Class 3 neurons, nociceptors are generally electrically silent [29] and transmit all-or-none action potentials only when stimulated. Nociceptor activity, however, does not per se lead to the perception of pain. The latter requires peripheral information to reach higher centers and normally depends on the frequency of action potentials in primary afferents, temporal summation of pre- and postsynaptic signals, and central influences [30]. Magnetic field-induced inhibition of pain can be explained based on the proposed model in accordance with the biophysical basis for the dynamical mechanism of AP initiation. Specifically, in class 3 excitability the fast-activating inward current surpasses the slow-activating outward current at the onset of stimulation. In presence of a static magnetic field, there will be an additional component associated with I_{fast}, namely a fast-activating outward (hyperpolarizing) current resulting from ion flow in the direction perpendicular to the AP transmission. This component can be incorporated into (8) by adjusting the g_{fast} term. Changes in fast currents lead to modulation of the net current at perithreshold potentials leading to variations in the excitation threshold voltage. During a stimulus transient, the additional I_{fast} component may assist the slow-activating outward current in overpowering the fast activating inward current in a class 3 pain neuron leading to suppression of repetitive spiking.

VI. Disscussion

Simulation of a neuronal membrane segment in response to two depolarizing current stimuli indicated that a relatively small reduction in the amplitude of the stimulus current resulting from presence of a magnetic field in the 10mT range may suppress action potential generation. Therefore, in agreement with experimental observations [15] relatively small magnetic flux densities may reduce excitability. A value of $5m^2V^{-1}sec^{-1}$ was used for the transverse ionic mobility in the simulation, which was calculated based on (4) assuming a magnetic flux density of 11*m*T to obtain a segregation ratio of α=0.95 corresponding to a roughly five percent reduction in the amplitude of the depolarizing current. A mobility value on the order of several $m^2V^{-1}sec^{-1}$ is, however, significantly

higher than the typical values reported for the mobility of different ions within the cell. A higher than expected value of mobility, however, can be readily explained by the absence of the scattering mechanism associated with the electric field across the membrane capacitance in the plane corresponding to the cross section of the axon.

The proposed model describing the influence of a constant magnetic field on the neuronal excitation threshold, further suggests that in presence of a magnetic field, the equivalent circuit representing the neuronal plasma membrane corresponding to the essential framework of the Hodgkin and Huxley model may be modified by including a magnetic-field-dependent current source whose magnitude is proportional to the magnetic flux density. In particular, the reduction in the depolarizing stimulus current, I_{Stim} may be accounted for in the equivalent circuit representation of the membrane using a hyperpolarizing CCCS whose magnitude is given by αI_{Stim}. Expansion of the equivalent circuit model for the neuronal membrane can be of value for simulation of the effect of magnetic fields on the excitability of neuron using the SPICE circuit simulation software.

In presence of a steady magnetic field reduced excitability may be manifested as a decrease in the frequency of action potentials. The fast-activating outward (hyperpolarizing) current resulting from application of a static magnetic field can lead to a reduced firing rate in class 1 and class 2 neurons. In particular, in class 1 neurons if the inward current faces competition from a fast hyperpolarizing current component arbitrarily slow spiking may be expected. In class 2 neurons the reduction in the net fast-activating inward current by the hyperpolarizing fast current component arising from the magnetic field may lead to a reduced rate of spiking down to a critical frequency below which spiking cannot be sustained.

VII. Conclusion

A biophysical explanation for the reduced excitability in neurons exposed to steady magnetic fields was presented. The slight redistribution of ionic charge resulting from the magnetic force acting on the mobile ions in the intracellular space was introduced as the origin of a Hall electric field in a direction perpendicular to action potential transmission along the axon. The Hall electric field, in turn, gives rise to a transient current density flowing in a direction perpendicular to the direction of the depolarizing current responsible for bringing the membrane potential to the action potential threshold level. The current density associated with the Hall electric field may constitute a relatively small fraction of the total stimulating current density available for depolarizing the membrane in response to the electrical stimulation of the neuron. The validity of the proposed explanation was verified by simulations based on the Hodgkin-Huxley model. Furthermore, the equivalent circuit model for the neuronal membrane was expanded, which may allow simulation of the influence of a magnetic field on neuron excitability using the SPICE circuit simulation software.

The proposed model for the influence of a magnetic field on neuronal excitability was shown to be consistent with the biophysical basis for the dynamical mechanisms of action potential initiation. In particular, an explanation for the magnetic-field-induced inhibition of pain was provided based on the proposed model in accordance with the biophysical basis for the dynamical mechanism of AP initiation in class 3 neurons.

A rigorous proof of the validity of the proposed model, however, requires employment of experimental neurophysiological methods for measurement of the induced electric field.

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