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The Coupled Action Potential Pulse (Appulse)–Neural Network Efficiency from a Synchronised Oscillating Lipid Pulse Hodgkin Huxley Action Potential

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Abstract

Assumptions of almost instantaneous activation of progressive ion channels to produce the Hodgkin Huxley Action Potential are based upon the belief that electrostatic charge can travel from one channel to the next at the speed of the Action Potential: empirical evidence from channel spacing, ionic radii and diffusion coefficients demonstrate this is not the case. Evidence exists from ion channel studies and entropy measurements of a Synchronous Oscillating Hodgkin Huxley Action Potential Lipid Pulse Structure that is both efficient and fulfils entropy measurement results. Depending upon the spatial arrangement of ion channels and other membrane components: the pulse may be HH or Lipid 'soliton' or both. This is a more consistent model for membrane pulse transmission than either HH or a soliton: it is consistent with observed studies in both unmyelinated and myelinated axon and the principle may be applied both to axons and muscle, including cardiac muscle.

Keywords: *Soliton; Neural Network Transmission Efficiency; Synchronised Oscillating Pulse; Hodgkin Huxley; Action Potential; Membrane; Myelin*

Abbreviations: APPulse: a coupled oscillating lipid pulse formed from the entropy of the Hodgkin Huxley Action Potential; (HH AP): where speed of flow is defined by the lipid pulse and activation of the ion channels by mechanical forces; Soliton: Self-reinforcing oscillatory wave; HH: Hodgkin Huxley Action Potential; AI: Artificial Intelligence; BNN: Brain Neural Network: this is an accurate model Neural Network of the brain neural connections taking into account noise, error, plasticity, histology, genetics and formation; DT: Distinct Time: this is the time during which an individual or multiple memory patterns within BNN retain integrity before reorganisation by plasticity;

Introduction

Work by Hodgkin Huxley [1] forms the basis of the textbook accepted mechanism for the action potential (AP); more recent work has shown that a 'soliton'[2,3] lipid mechanical pulse [4,5] is formed from the entropy fluid-mechanics of the lipid membrane, and moves at a rate almost indistinguishable from that of the HH AP. Furthermore antidromic collisions have been shown to be inconsistent [6]. Incompatible entropy measurements [7-9,34] show that a moving AP pulse has entropy production followed by loss of entropy. This compatible with a lipid pulse, but a lipid pulse requires initial energy.

The speed of spread across the membrane is dependent upon a rate limiting 'threshold' biophysics. This is the absolute moment before hyperpolarisation is irreversible and is implied by HH to be the electrostatic opening of gates. HH equations show resistance to charge flow as being cross membrane–as in an electrical circuit–but it must also include flow from one channel to the next to instigate hyperpolarisation. Calculation on ionic diffusion coefficients, ionic radii and single patch clamp studies are incompatible with the theoretical speed of HH AP suggesting that HH alone cannot account for membrane depolarisation activation speed. Moreover efficiency [10] requirements [11,12,34] indicate a more complex model. Efficiency and accuracy is of importance when considering computation in the BNN. Unlike spiking back propagation proposed by AI, a living BNN is composed of many independent living neurons each with its own distinct

transmission capabilities where computation is affected by continuous plasticity. In such environment accuracy, precision, efficiency and error reduction, during set time intervals, must be consistent. The ability to measure AP speed, accuracy of potential information transfer and entropy accurately in small 1 μm neurons is almost impossible: the study of neural networks is important as they allow us to fill in our knowledge from associative studies.

The 'Soliton' (a self-propagating oscillatory wave) [2] and HH [1] may not be inseparable and may form the basis for a coupled Action Potential Lipid Pulse–a coupled oscillatory structure that synchronises and ameliorates the efficiency accuracy and precision of information and has a mean-field behaviour. Thus it opens the possibility for more accurate development of computational models of accurate timing.

In this model depolarisation of a membrane to threshold produces a HH digit that provides the entropy *E* for a lipid 'soliton' pulse that decays with loss of entropy *e*. If the lipid soliton pulse encounters an ion channel and E remains high enough to provide entropy to mechanically distort and open the ion channel to threshold the oscillation continues. This model demonstrates that initial entropy formed from a HH digit is subsequently transferred to the lipid and dissipated during passage of the lipid pulse phase. During the falling phase and refractory of the AP heat loss has been measured experimentally giving evidence of an entropy pulse [7,8].

Methods

The basis of this study was the examination of two contradictory theories of nerve impulse propagation the HH and soliton theories. Each theory was subsequently deconstructed for its merits and each for its faults.

Using basic maths the speed distance diffusion timing between ion channels was calculated for the sodium ion from referenced sources.

An examination was undertaken into the historical significance of research into the action potential and its flow across a membrane and its relevant inclusion into a scheme for Action Potential flow with respect to recent discoveries of ion channel activity. Where inconsistencies became apparent, a combined mathematical model was created consistent with measured entropy, ion channel activation, lipid pulses, Hodgkin Huxley action potential, and efficiency error considerations. A theory was elucidated of an oscillating synchronous APPulse that was fully consistent with measured results.

The principles from the APPulse were then applied first to myelinated nerve fibre transmission and the to muscular activation including cardiac muscle to ascertain the value of the APPulse consistency as a principle of conduction.

Where figures were taken for calculations from sources [7-10,18,26-28] the greater error figures were used to give the best possible correlation to the Hodgkin Huxley action potential. No physical experimentation was performed by the author.

Results

The Elusive HH rate-limiting Threshold – flow ignition; the leading edge

If the on-going depolarisation is caused by a build-up of charge that opens an ion gate what initially opens the first ion gates to produce threshold? For continuous excitation charge must reach progressive ion gates to open them. Unlike electrical circuits where electrons spread across the metallic sheet of a capacitor at near light speed positive ions have a limited radius and must physically move into position to affect the next channel – the HH resistance R therefore is a factor of membrane resistance and flow resistance to the next ion protein before threshold is reached.

In HH terms the threshold is the potential caused by the capacitance potential of the main digit caused by ionic charge and creating all-or-none equilibrium activation. The capacitor aspect of HH is accepted as creating the digit potential; of contention is how in HH the charge flow from the digit can cause the continuous spread of on-going threshold depolarisation in the time measured by the speed of the propagating action potential. Ions require time to flow by diffusion through and along the membrane surface before further propagation from consecutive ion channels are opened before exponential hyperpolarisation. The ability of ionic charge flowing through one

ion gate to affect another is dependent upon how fast that charge may spread. Each ion channel and direct pathways between channels represents an individual resistance to this process so that total charging time T of the capacitance representing the exponential rising phase of the digit:

$T = \sum t.$

Where t is the mean time taken for adequate charge to spread from one channel to the next and activate exponential threshold.

Patch clamp studies demonstrate that single ionic activation channels are typically greater than 1 μm apart [33] usage of pipettes with a tip diameter greater than 2 μm results in unpredictable results when more than one channel becomes is covered by the pipette. Time taken for the propagation of the ionic driven action potential along the membrane is dependent upon the speed that ionic charge may be transferred from one ion channel to another by diffusion or by the ionic radius of the ion so that in membrane ion channel threshold terms mean speed is defined by:

$$S \propto \frac{\sum I+D}{t}$$

where S is the speed, I is the ionic radius and D is the diffusion distance.

For release of ions from one ion channel protein to affect another, cause hyperpolarisation and charging of the membrane requires time t. The time taken for ionic charge to spread from one point to another can be calculated from the rate of diffusion and the ionic radius of the ion.

The time for diffusion of an ion can be calculated approximately using the formula:
$T \approx x^2/D$

D is the measured diffusion coefficient of the ion, T is the time taken and x is the mean distance. The ionic diffusion coefficient of Na+ is 1.33×10^{-5} cm^2/s [26,27]. Substituting mean distance between ion channels of 1 μm gives an approximate diffusion time of 0.3 ms, which marks the maximum speed of transfer of the first ion charge out of the proximal ion channel to the first ion to the distal channel.

The ionic charge surrounding charged atoms that additively combine to give the digit potential hyperpolarisation has an effective distance time spread that is fixed by the ionic radii in which to stimulate other molecules (ion gates). The ionic radius (the distance over which each charge may be measured effectively) is only 116 pm for Na^+ [27].

Thus the speed from diffusion is insufficient to affect distal ion channel activation timing and continuous flow cannot be achieved by an HH model alone as an AP it would require a diffusion coefficient of 5000 cm^2/s or 5787 days to travel a metre [32]. In the HH model an AP can never travel faster than the speed defined by the speed of diffusion and the ionic radii combined.

Proximate Action

As an AP progresses along the axon it is possible to see that hyperpolarisation may occur as ion channels are increasingly surrounded by ions emanating from their own source but not how threshold may be achieved in the time taken for moving depolarisation in consideration of the diffused-charge distance. The conclusion is that HH cannot dictate the forward moving depolarisation speed but only defines the shape from threshold to hyperpolarisation, in other words the large entropy, E.

For a typical axon of 1 μm a typical speed is 1 ms [29]. Charging of the membrane by this method would generate a propagating action potential with a velocity of 0.0005 m/s: allowing an error in diffusion coefficients and measured ionic radii of 20 times, this figure is no greater than 0.01 m/s It is therefore highly unlikely that charge from the digit leading edge is the responsible instigator for activating threshold during AP active flow. Another process therefore must bring the action potential to threshold during AP flow and regulate speed of APPulse.

APPulse

A membrane is made up of many ion protein channels that may have more than one activation method; in addition there are lipid channels [15] and a mechanical wave accompanies the action potential [12]. Membranes possess many electromechanical properties that must change the basic HH flow of ionic charge. It is probable that in the flowing AP the remaining entropy e of the lipid pulse soliton activates the channels to threshold potential by mechanical means than by the ionic charge flowing through the channels to hyperpolarisation. This would be analogous to the hysteresis mechanical effects on substances like a rubber band that once deformed takes time to restore to its original shape, thus delaying closing for the measured refractory period.

Coupled Action Potential Pulse

Many occurrences of synchronous oscillation appear in nature and a mathematical treatment of them has been reported elsewhere [16]. Nerve impulses are concerned with computation and is it the theory of computation that must be matched to any theory of Action Potential Pulse–the coupled action potential pulse [15] should therefore be been seen in the light of its increased computational possibilities.

A coupled action potential pulse is formed when a threshold is reached by mechanical stimulus with a HH digit that provides the energy required for on-going entropy of the pulse. Entropy: The total entropy *E* of the system where an initial burst of entropy is dissipated *e* resulting in enough entropy to open the ion gates restarting the process.

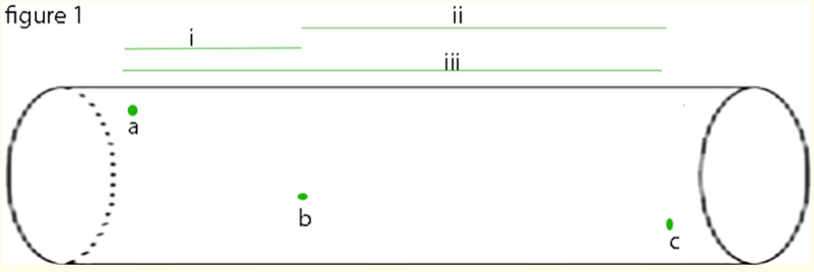

Figure 1: *An illustrative, uniform axon containing three widely spaced ion protein channels.*

For the benefit of a clear description the axon has been standardised as follows:

a. The axon is uniform such that speed along the axon by the lipid pulse is constant.
b. The protein channels are gates that reach threshold at a voltage of *V* and produce a digit of Entropy *E* distance along the axon is proportionate to time.
c. In this axon there are no lipid channels or other proteins except the three ion channel proteins.

Figure 1 demonstrates that on depolarisation at a, an action potential digit of entropy *E* is created. A Lipid pulse wave is subsequently created by Entropy *E* that continues along the axon. Entropy loss e from *E* causes a proportionate decrease in amplitude but not in speed according to wave theory[30] that causes the entropy to decrease by dissipation over distance d such that *Ed*(b) = *E*-*e*. This residual entropy *Ed*(b) is above threshold t and causes depolarisation at b to complete the circle. In this model the entropy of the AP digit must be sufficient to produce a lipid pulse of such entropy *E* to arrive at b and take b above threshold for the APPulse combination to continue. In smaller diameter neurons dissipation will be greater due to entropy being a function of membrane area.

Continuance of the pulse depends upon the entropy provided by the HH cycle–the digit and the entropy loss *e* of the lipid pulse. If the entropy provided by the ion protein channels is sufficient only to provide a pulse of entropy *E* that produces less entropy than that required by t then continuance of the pulse will fail.

A random placement of ion channel proteins produces a mean oscillating synchronised APPulse from the HH depolarisation and the lipid soliton. This Oscillation between randomly placed ion channels produces a structure where the dynamic at any finite point on the axon produces a differential APPulse that has an absolute value (the threshold *t*) a variable entropy (an initial burst *E* followed by a level of decreasing entropy *e*) depending upon the specific dynamics of the membrane. Both *t* and e are factors of importance when considering the interference that occurs when two APPulse structures collide. At that moment the concentration and distribution of ion protein channels and the lipid surface of the membrane will considerably change the transmission properties of the pulse and at that location the dynamics may allow either an HH AP a Lipid Pulse or an oscillating synchronicity.

Entropy

This model fully concurs with experimental entropy measurements[11] where high entropy is produced upon initiation of an action potential [9] and subsequently entropy is reduced as the soliton releases energy [17] The APPulse will continue efficiently over long distances because of the oscillating addition of entropy from HH,-and its ion transport systems.

Collision Theory

In Figure 1 If the dissipation of entropy from the lipid pulse e is reduced such that the entropy *E* from the depolarisation at is sufficient to produce a lipid pulse with entropy *E* sufficient to depolarise c then ion channel *b* is redundant. If the HH refractory period *r* is small, for the ion channels and the distance *d* between channels is large then where *e* is near 0 (as in a large axons) lipid antidromic pulses will penetrate, combine and continue as in wave theory and depolarise further down the axon resulting in pulses that pass as solitons.

In contrast if *r* is large and *e* is large then the HH cycle becomes the driving force and antidromic pulses will be voided by the refractory periods.

This model demonstrates that spatial dynamic qualities of the membrane will affect the transmission and the refractory period may or may not exist according to membrane composition. In addition experiments on large size neurons suggest that soliton-wave HH interference combinations may be possible under certain circumstances [21,22,35].

Myelinated axons

The Oscillating Synchronous pulse has implications for myelinated conduction of a pulse and clinical implications for diseases where de-myelination is implied.

A myelinated axon is an almost perfect environment for an oscillatory synchronised HH lipid pulse system between the entropy producing node of Ranvier and the lipid myelin sheath. At the node of Ranvier is a concentrated area of ion protein channels [23] followed by the axon with diminished channels and covered by the lipid myelin sheath. The myelin sheath is of a shape that is both a near perfect insulator in the outer layers and an almost perfect lipid transmission zone in the inner folds. The overlapping ends of the myelin form a perfect compression seal. The shape is rectangular when extruded forming a surface for a lipid soliton pulse that would spread from one corner to another of high to low entropy, minimising distortion at the first node of Ranvier and maximising pulse entropy at the next. The myelin spiral therefore transmits from the inner folds and is mechanically supported by the outside folds. The outside rigid sheath minimises entropy loss e while the inner layers maximise the flow of entropy *E*. In the myelin sheath the HH cycle becomes redundant allowing for faster pulse transfer. Any disruption to the mechanical properties of the sheath will affect its dynamic properties and may cause disruption to the pulse. This concurs with the experimental data on heat production by Tasaki [11] and others where myelinated axons demonstrate bursts entropy at the nodes of Ranvier and subsequent entropy is decreased in the myelin.

Timing

An Oscillating Action Potential Pulse suggests that it is the lipid pulse phase that provides the overall speed of the pulse. The time *t* entropy *E* and decay *e* values of this pulse are therefore different for each axon part giving a distinct profile. The shape profile of this

pulse is almost infinitely variable depending upon the dynamics of the axon: both the lipid structure, the HH components and the other elements forming the membrane. The t *E* and *e* curve therefore is almost certainly non-linear but theoretically stable for any point on the axon. Computationally this can be reduced to a variable threshold value for HH and the continuance of the APPulse at any distance along the axon. Timing is therefore oscillatory between the two structures giving a set point and mean structure.

Discussion

Without an electrostatic charge sufficient to open ion channels, the only consistent mechanism for the activation to threshold during depolarisation is mechanical displacement caused by the entropy from a lipid soliton. As written, the Hodgkin Huxley equation explains the initial entropy but cannot account for the continuous depolarisation or speed of propagation as it does not take into consideration the spatial membrane dynamics of the ion channels.

This coupled pulse has all the control elements of the Hodgkin Huxley system including a refractory period except:

1. Less ionic protein channels are required.
2. The system is stable and regular and better able to consistently pass information critical to a Neural Network.
3. Less ionic current is required to operate the system.
4. Less energy is required as the entropy provided by the HH depolarisation is used to 'power' the APPulse pulse. Entropy measurements are consistent with theory.
5. Unlike HH alone on-going soliton pulse activation of the ion channels permits an action potential pulse of the correct speed.
6. It is consistent with patch clamp studies and entropy measurements.
7. It is more efficient as it only requires a pulse of entropy from an ion channel followed by dispersion by the lipid pulse.
8. It is a more consistent explanation of myelin pulse. Saltatory conduction is thought to function to drive the local electric current accompanying the action potential at one node to the next site of initiation, which because of the insulation provided by the myelin, is the next node of Ranvier. Current is limited to diffusion and ionic radii as above and a 1mm distance between nodes would need more than 8 minutes (calculated from diffusion and ionic radii). In addition axon bundles would inevitably have interference with salutatory conduction leading to noise and error.
9. The capacitance theory of myelin states that the thickness of the myelin is important: the ionic radius of Na^+ is 116 pm so a thicker membrane than this does not increase capacitance: there is a limitation to the ability of electrical circuitry being applied to biological membranes.

The principle of the APPulse provides a consistent mechanism that can easily be applied for myelinated nerves and muscle including cardiac muscle.

Evolutionary studies show that evolution is not static and unidirectional [20] but over millions of years adaptive elements are used efficiently as possible. Functional computation may be different from the action-reaction due to neuronal stimulation of muscles in animals with simpler nervous systems and the facilitation of impulses may differ between axons of different species.

The theoretical joining of the 'soliton lipid' mechanism and the HH AP adds considerably to the efficiency of a pulse and increases the accuracy and efficiency when considering computation – especially at smaller diameters of axon-and would logically follow from evolution where initially, a lipid facilitated conduction system became a more advanced hybrid membrane for neural computation. Such a system would combine the efficiency and consistency of speed of a wave moderating the speed of impulse with the control of an ion flux system extending the distance over which the soliton would flow by addition of entropy and allow for ameliorated computation. This dualism of soliton-HH would greatly enhance efficiency and reduce variability of impulse latency in finite time.

Addition of proteins, other ion channels, ions and membrane components adds distinctiveness to each axon pathway so that dynamic timing between one synapse to another becomes a distinct variable profile of the specific axon. In addition this mechanism

provides a pathway for computation in the BNN that exceeds theories of spiking circuits and allows for computation consistent with cognitive tests on the speed of thought.

There is a direct, consistent and plausible pathway between an Oscillating Action Potential Lipid Pulse, computation and a Brain neural Network capable of associative learning and cognition. The AP Pulse is also a direct consistent and more plausible explanation for transmission within a myelinated axon.

The AP Pulse is also a consistent model for transmission of conduction of pulses over all conductive membranes and almost certainly applies to muscle contraction and the *Cardiac action potential* of the heart.

Conclusion

The Hodgkin Huxley equation explains the initial entropy of an action potential, but does not account for its continuous depolarisation or its speed of propagation. The coupled action potential pulse accounts for this because it is an oscillating lipid pulse synchronised with the Hodgkin Huxley Action Potential.

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