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The Coupled Cardiac Action Potential Pulse (CAPpulse) – Synchronised Oscillating Mechanical Pulse Cardiac Action Potential

A S Johnson*

117 BIS Avenue Perpignan, 66410 Villelongue de la Salanque, France

***Corresponding Author**: A S Johnson, 117 BIS Avenue Perpignan, 66410 Villelongue de la Salanque, France.

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Abstract

Fundamental to the functioning of the heart its efficiency and lifetime durability is the control of its speed of contraction. Contemporary thinking supposes that contraction occurs by the Cardiac Action Potential (CAP) that can be measured easily and has its origins from trans-membrane 'ion-currents' propagating through ion channels along the surface of a membrane. However, as this study demonstrates mathematically and empirically, cable theory is an imperfect model for describing the AP and for subsequent electromechanical coupling in the heart. Assumptions of almost instantaneous activation of progressive ion channels to produce the Cardiac Action Potential are wrongly based upon electrostatic charge travelling from one channel to the next at or near the speed of transmission: empirical evidence from channel spacing, ionic radii and diffusion coefficients demonstrate mathematically that this is not the case. Further scrutiny is therefore required, particularly as one in four deaths is caused by cardiac irregularity. However, evidence exists from ion channel studies and entropy measurements of membranes for a synchronous oscillating cardiac action potential mechanical pulse structure that is efficient and fully explains the speed of transmission: this paper proposes this model as a foundation for consideration in further studies of the heart.

Understanding of the activation spread and speed of contraction is essential in any study or clinical application of the heart. The Coupled Cardiac Action Potential Pulse explains these mechanisms. This is a more consistent model for cardiac pulse activation: it is consistent with observed studies and the principle may be applied to other types of muscle.

This paper is a derivative of: The Action Potential Pulse (APPulse) – Synchronised Oscillating Mechanical Pulse Action Potential [1].

Keywords: *Synchronised Coupled Oscillating Pulse; Cardiac Action Potential; Membrane; Mechanical Pulse CAP*

Abbreviations

AP: Action Potential; CAPPulse: a coupled oscillating mechanical pulse formed from the activation of muscle; HH AP: Hodgkin-Huxley Action Potential. Speed of pulse is defined by a lipid pulse and initial activation of the ion channels to instigate potential threshold leading to full depolarization is by predominantly mechanical forces, with an electrical component providing activation in membrane-areas of very high channel density - which almost certainly includes an entropy component created by cardiac muscle contraction.

Introduction

One in four deaths is caused by cardiac irregularity; ventricular arrhythmias are a major cause of sudden death, which accounts for approximately half of cardiac mortality; speed of contraction and force developed are general quantifiers of cardiac health.

The Cardiac Action Potential conduction system was adapted from the elegant theoretical model of Hodgkin and Huxley [2] that forms the basis of the textbook accepted mechanism for the action potential (AP) along the membrane of nervous tissue. Evolution of their model has progressed according to the availability of experimental data with the assumption that their work was essentially correct, although

it was accepted that the speed of transmission could not be defined. Their paper assumed cable theory in the action of propagation to provide the speed of impulse along the axon although little or no biological evidence exists to corroborate this.

Similar membrane constituents make up the membrane structures that power the Cardiac Action Potential (CAP). The mechanisms that power both the AP and CAP can be thought to be analogous but are by no means similar because cardiac muscle is fundamentally a moving structure that during the CAP-contraction changes size and shape - although similarities exist in the membrane channels and fundamental materials of the membranes. Previous work has shown that a 'soliton [3,4]' lipid mechanical pulse [5-8] may be formed from the entropy fluid-mechanics of the lipid membrane, and moves at a rate almost indistinguishable from that of the HH AP in axons and the membranes of sarcolemma are similar.

Entropy measurements and the large spacing between ion channels discredits cable theory leaving the only plausible explanation for speed of propagation to be a combined and synchronised oscillating pulse [1], making a closer critical look at the CAP a necessity.

The basis of this paper is to examine the contemporary mechanism proposed for the Cardiac Action Potential CAP specifically the CAP actinomyosin coupled contraction in relation to the speed of systole with the hindsight of knowing that ion channel propagation alone cannot explain speed of the CAP. Timing between systole and diastole and indeed pulse rate are critical indicators of heart health and the fundamental mechanism essential groundwork before any understanding of biochemical or pharmacological investigation.

The discovery of the APPulse [1] led to the reinvestigation of the inter-channel distance between ion channels in the heart throughout the cardiac syncytium which are of a similar magnitude. Similar mechanisms may be shown to mathematically exist to the APPulse where continuous depolarisation along the axon membrane is impossible without a further mechanical element. This forms the elements of the Coupled Cardiac Action Potential Pulse (CAPPulse). Speed of contraction and synchronous timing is fundamental to the proper and efficient functioning of the synchronicity of cardiac muscle. Any change in this synchronisation leading to inefficiency may lead to enlargement, deterioration or even failure. To understand the controlling mechanisms that define the speed and synchronicity of function it is necessary to deconstruct the flow of the Cardiac Action Potential which forms the basis of conduction and to evaluate those elements necessary for initiation and those that mediate effectiveness. For this to happen an accurate model must be formed that can then be used to evaluate the processes that affect the mechanical operation, pharmaceutical modifiers and applied experimental techniques.

The cardiac impulse (CI) is generated in the sinoatrial (SA) node. It traverses and activates the atria, before converging on the atrioventricular (AV) node for distribution to the ventricles via a specialised conduction system; the bundle of His; the main bundle branches to each ventricle, and the Purkinje fibre network. Originating in pacemaker cells the CI travels along the His –Purkinje system composed of specialised cells responsible for the synchronous activation of the ventricles and timing of systole [9,10].

Contemporary orthodoxy separates the cardiac action potential and the His –Purkinje system from the contraction of the underlying muscle although it has been noted that the speed of the action potential, in normal function, is tied precisely to that of the contraction and that contraction once instigated may proceed when the His-Purkinje system is incapacitated [11]. Experimental studies show that the Purkinje system can be arrhythmogenic during electrolyte imbalance, after exposure to various drugs, and in myocardial ischemia, during which Purkinje cells can survive in anaerobic conditions [12,13]. In addition, catheter ablation where the conduction system of the Purkinje tissue is severed can often prevent cardiac arrhythmias.

The Cardiac Action Potential differs significantly in different regions of the heart, reflecting a differentiation of functionality that provides both synchronised and concatenated contraction of disparate areas [14]. There are many reviews of the mechanisms involved of Pacemaker cells, Purkinje cells and the corresponding pathways of activation that are outside the scope of this paper.

An understanding of the physiology of ion handling in cardiac cells is critical for understanding the functioning of the excitation-contraction (EC) coupling of cardiac muscle. Central to this is the conventionally accepted mechanism of Ca induced Ca release (CICR).

During a CAP the voltage dependent L type Ca channel is activated – resulting in a small influx of external Ca into the cytosol. This Ca bids to the cytosolic Ca ryanodine receptor RyRs located in the sarcoplasmic reticulum. Ryanodine receptors (RyRs) exist as three mammalian isoforms (RyR 1–3) of which RYR2 is predominant in cardiac muscle. RyRs are located in the sarcoplasmic/endoplasmic reticulum membrane and are responsible for the release of Ca^{2+} from intracellular stores during excitation-contraction coupling in both cardiac and skeletal muscle. RyRs opens channels leading to an out-flux of Ca from the SR – the main intracellular store of Ca. The released Ca then binds to Troponin C causing a cascade of conformational changes in the myofilaments and ultimately causes muscle contraction. During the relaxation phase Ca release is terminated and the released Ca is recycled back to the SR by the SR CA ATPase or extruded by the cell by the Na/Ca exchanger thus lowering cytosolic Ca concentration and allowing dissociation of Ca from the myofilaments permitting muscle relaxation [9].

Recent studies [11,15] have shown that many membrane channels are also mechanically gated or in the case of more than one stimulus mechanically modulated [11]. Lipid Channels may also form under mechanical stimulation [16].

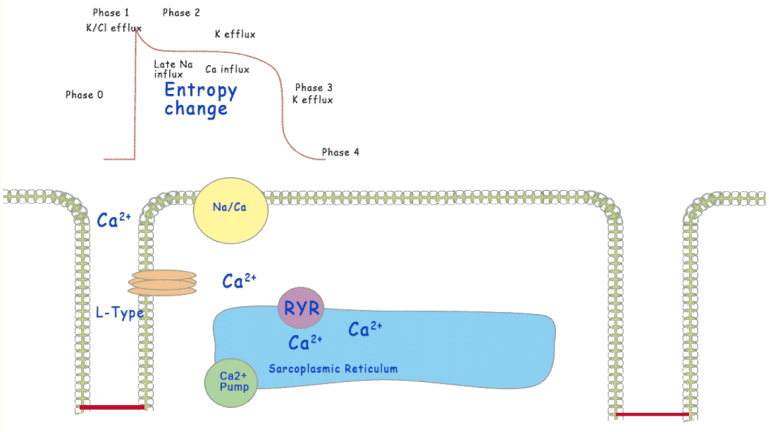


Figure 1: *Resting State.*

In the conventional model of Excitation Contraction coupling, excitation of the adjacent membrane causes potential sensitive channels to open unrelated to the likely physiological impact of contraction from myofilaments situated within the local or adjacent syncytia. Neither is the CAP considered to be anything greater than a potential difference acting upon potential-sensitive channels: neglecting the impact and possibility of a lipid pulse travelling along the membrane or of the undeniable effect of the myofilament contraction upon the spatial histology of the syncytium, sarcoplasm, lumen and the ensuing mechanical effect this would have upon the internal structure and function of the SR, T tubules and the cellular Ca constitution.

This paper examines the relationship between the ion channels and the membrane spread of the CAP critically in relation to the timing of the opening of the channels to enable full depolarisation. In the CAP the speed of spread across the membrane is dependent upon a rate limiting 'threshold'. This is the absolute moment before hyperpolarisation is irreversible and is implied to be the electrostatic opening of Na gates – phase 0. Rapid Na^+ channels are stimulated to open, flooding the cell with positive sodium ions. For this hyperpolarisation to occur there must be a mechanism to open successive channels across the surface of the membrane. Cable theory suggests resistance to charge flow as being cross membrane – as in an electrical circuit – but it must also include flow of ion charge from one channel to the next for hyperpolarisation to occur. By contrast to an electrical circuit where spread of electrostatic charge across a capacitor membrane is almost instantaneous positive ions have to physically move – capacitance therefore is a useful but inaccurate model. No electrons are

involved in the process and the difference in potential is achieved by relative potentials of positive ions. Positive ions are of a defined size and require a finite time to travel not only through the membrane but also to adjacent channels in order to activate and eventually hyperpolarize the membrane. It is the mathematical relationship between the distance and the time taken that defines the spread across the membrane and which can be shown to be reliant upon further factors. This relationship is easy to calculate from ionic radii and diffusion coefficients both of which are available.

This paper firstly describes the general, mathematical relationship between the CAP and contraction in terms of membrane potential and examines the possibility of CAP spread and hyperpolarisation along the sarcolemma by a coupled mechanical synchronized pulse. This coupled synchronised oscillating potential-mechanical membrane compression pulse is comprised of; the potential difference across the membrane, a lipid pulse allowing the potential to spread and hyperpolarize and 'electro'-contraction-coupling caused by contraction of the myocytes and the deformation of the syncytial membrane leading to a synchronised CAP Pulse where contraction: a. deforms the membrane causing mechanical opening of the channels and a CAP, b. compression of the sarcoplasm and sarcoplasmic reticulum within causing expulsion of Ca leading to myocyte contraction.

Implications of this model include an explanation of Delayed after depolarization (DAD) caused by enforced delay between the CAP and the on-going contraction pulse causing a state of duplication of the CAP and defibrillation.

Methods

An examination was undertaken into the historical significance of research into the cardiac action potential, its flow across a membrane and its relevant inclusion into a scheme for cardiac action potential flow with respect to recent discoveries of ion channel activity.

The maximum speed of hyperpolarisation of the membrane was calculated using the method-values published from single Na and Ca ion channel studies ionic radii and diffusion coefficients.

The speed of activation of the myocyte was calculated from the point of hyperpolarisation by a calculation of the diffusion coefficients, ionic radii and the approximate length of T tubules thought to provide intracellular Ca^{2+} to act on tropomyosin complexes and induce myocyte contraction.

A model of membrane activation and myocyte contraction was formed that was consistent with the experimental studies and the calculated results.

Where inconsistencies became apparent, mathematical models were created consistent with measured entropy, ion channel activation, lipid pulses and efficiency.

Calculation was undertaken on ionic diffusion coefficients to show the time required for ions to travel from one channel to the next to activate hyperpolarisation. Diffusion mathematics is well defined and documented and beyond the scope of this paper, calculations are given in results. Ionic radii of ions were also evaluated to verify the extent to which one charge can affect another at distance. Methods of single patch clamp studies were researched to determine the average distances between ion channels [17,18]. Calculations were then made to determine whether charge from one channel alone could open a neighbouring channel and thus be responsible for the CAP and cardiac contraction [16,19,20].

Results

For continuous excitation and hyperpolarisation 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 ionic radius and must either be adjacent to, or physically move into position to affect the next channel. This follows for all electrostatically operated channels.

In cable theory 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 the Cardiac action Potential is accepted as creating the digit potential; of

contention is how the charge flow from the initial digit can cause the continuous contiguous depolarisation along a membrane from one ion channel to the next in the time measured by the speed of the propagating cardiac action potential. Ions require time to flow by diffusion through and along the membrane surface before activation and further propagation of consecutive ion channels are opened creating exponential hyperpolarisation phase 0 of the CAP Na intake. It is this process that defines the speed of the CAP linked to the speed of cardiac contraction before repolarisation and recovery. 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 [13,21] with 1.5 µm taken as standard for pipettes in single ion channel clamping research [13]. Usage of pipettes with a tip diameter greater than 2 µm results in unpredictable results when more than one channel becomes covered by the pipette. This empirical measurement has therefore long been established and accepted as being the distance between adjacent channels. 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 [19,20] and that for Ca^2+ 0.79 × 10-5 cm^2/s.

For the sodium channels: Substituting mean distance between ion channels of 1 µm gives an approximate diffusion time of 0.3ms, 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 picometres(pm)or Na^+ [21,22] and 114 pm for Ca^{2+}

Thus, the speed from diffusion is insufficient to affect distal ion channel activation timing and continuous flow cannot be achieved by a CAP model alone as a CAP it would require a diffusion coefficient of about 5000 cm^2/s assuming a distance of 1.5 μm. Using $T \approx x2/D$ and substituting Ca diffusion coefficient of 0.79×10^{-5} then it would take a Ca ion 7325 days to travel a metre [11,15] or more simply take about 1 year for each heartbeat.

CAP Channel Proximate Action Hyperpolarisation

Instigation of depolarisation is by pacemaker cells. For a CAP to exist as an entity and to progress along a sarcolemma depolarising and causing hyperpolarisation the channels would have to be much closer together. It is possible to accept that channels may open as a result

of ions proximate to the sites channel receptors – within ionic radii or, even allowing for charitable error double ionic radii. However, it is not possible at initial activation threshold for ionic charges to spread across the membrane in sufficient quantity and time to activate ion channel receptors in the time taken for moving depolarisation as demonstrated by phase 0 of the CAP – the distances in consideration of the diffused-charge distance are too great.

The conclusion is that ionic charge alone cannot dictate the forward moving depolarisation speed but only defines the shape from threshold to hyperpolarisation, in other words the large entropy, E.

It is therefore highly unlikely that charge from the digit leading edge of the CAP is the responsible instigator for activating threshold hyperpolarisation during CAP active flow. Another process therefore must bring the action potential to threshold during AP flow and regulate speed of CAPPulse, and myocyte contraction and be responsible for the mechanical time/force physiological qualities of the heart. The CAP alone is not therefore responsible alone for the activation of either the sarcoplasmic reticulum or of myocyte contraction.

The Coupled Cardiac Action Potential Synchronised Oscillating Mechanical Pulse (CAPpulse)

Cardiac muscle is a mechanical entity, its function is to contract, exerting pressure: as a direct consequence of contraction physical changes take place within the cellular structure. Cells in the human body are not fixed but are composed of membranes, protein and compounds almost all of which exhibit elasticity and subsequently hysteresis in relaxation. In the APPulse [1] it was described how a coupled lipid action potential pulse could better describe a model for an action potential than that of Hodgkin Huxley and could furthermore better explain myelinated transmission. Similarly, the sarcolemma of a cardiac syncytium is a lipid membrane where a lipid pulse could explain CAP hyperpolarisation. However cardiac muscle itself is a moving component pressurizing the syncytium many times during contraction which undoubtedly has an effect both on the CAP travelling along the membrane and the spread of Ca through the sarcolemma to the sarcoplasmic reticulum and almost certainly in Ca activated Ca release.

A model is therefore proposed combining these three factors, CAP, lipid pulse and mechanical contractive cellular changes. A preliminary discussion of the soliton pulse is described elsewhere [1,3,16,25]. Many occurrences of synchronous oscillation appear in nature and a mathematical treatment of them has been reported elsewhere [26,27,28].

A coupled cardiac 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.

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

a. The sarcolemma is uniform such that speed along the sarcolemma 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 sarcolemma is proportionate to time.

c. In this sarcolemma, there are no lipid channels or other proteins except the three ion channel proteins.

Figure 2 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 sarcolemma. Entropy loss e from E causes a proportionate decrease in amplitude but not in speed according to wave theory [17] 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 CAP digit must be sufficient to produce a lipid pulse of such entropy E to arrive at b and take b above threshold for the CAPPulse combination to continue. 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.

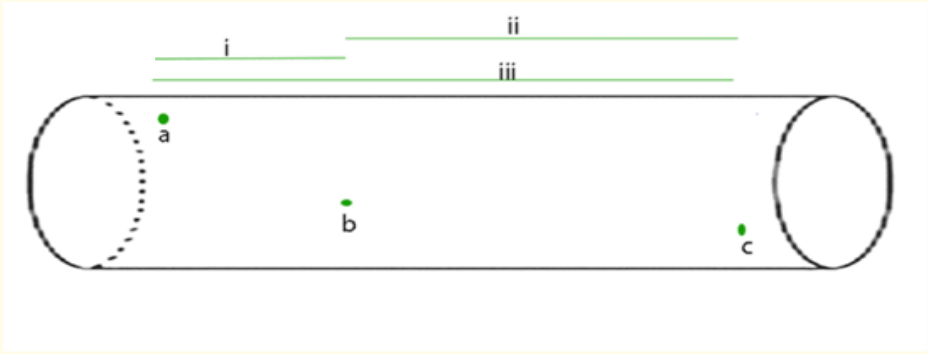


Figure 2: *An illustrative, uniform sarcolemma containing three widely spaced ion protein channels.*

A random placement of ion channel proteins produces a mean oscillating synchronised CAPPulse 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 sarcolemma produces a differential CAPPulse 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. 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.

Timing

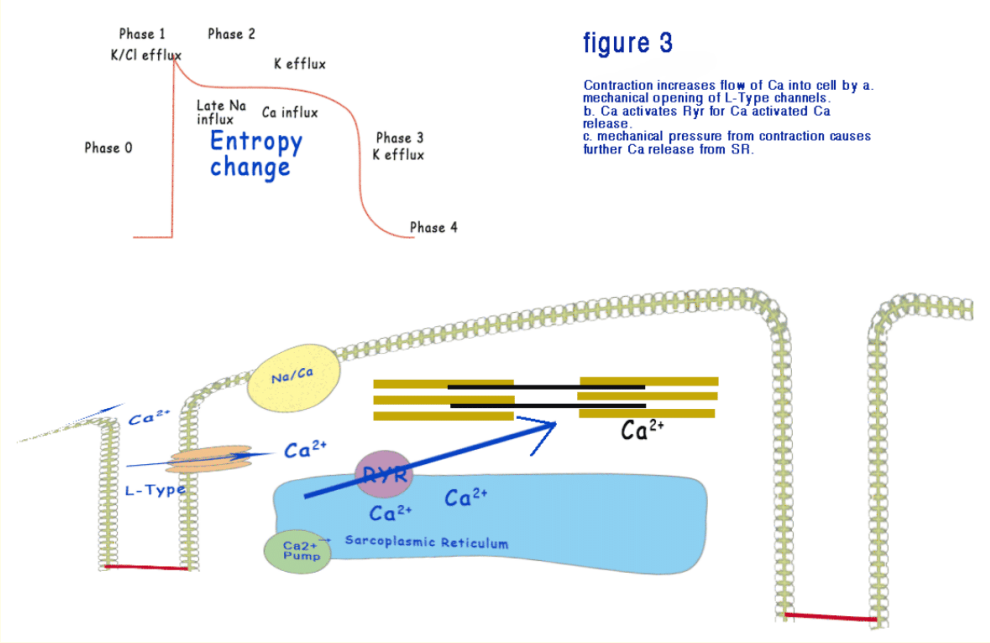
In a Coupled Oscillating Cardiac Action Potential Pulse, it is the lipid pulse phase that provides the overall speed of the pulse. The time t entropy E and decay e values of this pulse are therefore different for each sarcolemma (part) giving a distinct profile. This lipid pulse is also affected by the cardiac muscle mechanical contraction and is almost certainly bound to it unless the membrane is ruptured or malformed producing further coupling. The shape profile of this pulse is almost infinitely variable depending upon the dynamics of the sarcolemma explaining differenced in recorded CAP: 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 defined stable point on the sarcolemma. Computationally this can be reduced to a variable threshold value for HH and the continuance of the CAPPulse at any distance along the sarcolemma.

Timing is therefore oscillatory between the three structures at a set point and coupled by entropy provided by the HH CAP and the mechanical component.

Ca activated Ca release by CAPPulse:

The exact sensor for intra-SR release of calcium has not been elicited but is suspected to be the ryanodine receptor described briefly above it is also known that many channel proteins exhibit mechanical opening under pressure [11,15] including the L type Ca channel and ryanodine [11]. Histologically the SR is a connection of tubules within a finite space with the SR placed in close proximity to the T-tubules allowing for close connectivity between a CAP pulse and the SR and rapid pulse transfer. Partial de-tabulation of the muscle causes a depressed contractile force and slower twitch kinetics [29].

There are two main ways to change the strength of cardiac contraction: by altering the amplitude or duration of the Ca transient, and by altering the sensitivity of the myofilaments to Ca. Myofilament Ca sensitivity is enhanced dynamically by stretching the myofilaments (as the heart fills with blood), resulting in a stronger contraction. This is due, in part, to the transverse filament lattice compression that occurs on stretch, which enhances the actin–myosin interaction. This lateral compression is an important self-regulatory mechanism by which the heart modifies to alter diastolic filling.



A sarcolemma is made up of many ion protein channels that may have more than one activation method; in addition there are lipid channels [30], and a mechanical wave pulse accompanies the action potential [23,25]. Membranes possess many electromechanical properties that must change the basic flow of ionic charge. It is probable that in the flowing CAP the remaining entropy e of the lipid pulse soliton activates the channels to threshold potential by mechanical means as well as 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 the measured refractory period.

After activation of any part of the syncytium by the sarcolemma, internal pressure in any part of the SR will rapidly transfer causing a spread of Ca greater than by diffusion alone. It is this pressure that opens Ca channels and precipitates Ca activated Ca release; in this model, it is unnecessary for large potential changes within the syncytium for SR-Ca activated Ca release. The combination of potential change and pressure opens the SR channels. In this model, any proximate change in charge in any region of the SR instigates an immediate pressure curve to open other Ca channels. The rate of activation is proportional to the pressure and the levels of Ca. Closing may be timed by hysteresis.

This model is consistent with the Frank Starling effect [31] where the stroke volume increases with the filling of the heart due to the extra pressure within the syncytia promoting an increase in internal fluidity.

Conclusion

It is an empirical impossibility that the CAP is the correct activator of cardiac muscle; whilst the CAP is alluringly simplistic it is unacceptable as a foundation for research into an organ responsible for the death of a quarter of all human life.

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 in coupled action with mechanical contraction. The Hodgkin Huxley equation explains the initial entropy but cannot account for the continuous depolarisation speed of propagation or contraction, as it does not take into consideration the spatial membrane dynamics of the ion channels or explain activation.

The Coupled Cardiac Action Potential Pulse is an oscillating pulse powered by the entropy of the HH cycle that flows through a lipid membrane between channels. This pulse flows down the T-tubules opening the Ca channels that in turn activate the channels in the sarcoplasmic reticulum. In this respect, there is little evidence of excitation – contraction coupling but of coupled excitation-pulse contraction coupling. Spread of Ca from the SR is by initial diffusion and then by pressure pulse fluid movement within the lumen with mechanical forces opening channels. Ca is recycled by the Na/Ca and Ca ATPase within the membranes.

Although the CAP permits clinicians to view certain aspects of cardiac activity, what is seen is a ghosting of actual cardiac function. Ironically in a healthy heart the CAP records correctly and mirrors the CAPPulse, however during cardiac arrhythmia or DAD for example, what is recorded is the CAP and not the activity of full cardiac function of the pulse and flow of activation or activity within the sarcoplasm.

Synchronicity is of importance to the heart to maintain cellular integrity; like all materials undergoing stress movement within the membranes and proteins of the heart must be kept within normal limits to prevent stress injury. Cardiac muscle in desynchronised or syncopated rhythm, like any material, produces stress and repetitive strain. Membranes subjected to repetitive action are susceptible to injury.

Under normal working conditions the feedback oscillation from the coupled CAP – pulse – contraction complex maintains a synchronised heart, however it is not difficult to see that where the CAP is cut from the pulse contraction or the CAPPulse from the contraction any split will produce two contractions; these extra contractions then become the basis of arrhythmias and DAD. Any slight de-synchronisation will inevitably damage tissue further.

This coupled pulse has all the elements of the Hodgkin Huxley system except:

a. Less ionic protein channels are required.

b. The system is stable.

c. Less ionic current is required to operate the system.

d. Less energy is required as the entropy provided by the HH depolarisation is used to 'power' the APPulse pulse. In addition, cardiac contraction itself contributes to its entropy.

e. Unlike HH alone on-going soliton pulse activation of the ion channels predicts a cardiac action potential pulse of the correct speed.

f. It is more efficient as it only requires a pulse of entropy from an ion channel followed by dispersion by the lipid pulse – the lipid pulse also is coupled to the cardiac contraction.

The theoretical joining of the 'soliton lipid' mechanism, the HH AP and contraction adds considerably to the efficiency of a pulse and increases the accuracy and efficiency. Such a system would combine the efficiency and consistency of speed of a wave pulse moderating the speed of impulse with the control of an ion flux system extending the distance over which the soliton mechanical pulse would flow by addition of entropy. The dualism of soliton-HH would greatly enhance efficiency and reduce variability of impulse latency in finite time even without contraction.

Clinical Implications

Clinicians use their knowledge of cardiac physiology to predict the actions of pharmaceuticals on heart rate/force: this model changes the application of treatments to better reflect the actions of the cardiac muscle allowing more accurate treatments to become available.

Knowledge of the CAPPulse should provide information for a new type of synchronised pacemaker to control arrhythmias.

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