Field dynamics in atrioventricular activation. Clinical evidence of a specific field-to-protein interaction

Manel Ballester-Rodés,⁎, Francesc Carreras-Costa, Teresa Versyp-Ducaju, Montserrat Ballester-Rodés, Davendra Mehta

a Department of Medicine, Faculty of Medicine, University of Lleida, Catalunya, Spain
b Cardiac Imaging Unit, Hospital de la Santa Creu i Sant Pau, Universitat Autònoma de Barcelona, CIBERCV, Spain
c Consorci Educació Barcelona, Diputació de Barcelona, Spain
d Consultant in Organic Chemistry, Biochemistry and Enzymology, Barcelona, Spain
e Al Sabah Arrythmia Institute, Mount Sinai St. Luke’s Hospital, New York, USA

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This work is dedicated to Paco Torrent-Guasp MD (1931–2005), who unveiled the double helical structure of the heart, to Mae-Wan Ho (1941–2016) for her dedicated long-standing work to synthesize the complex scientific principles of Physics of Biology and to James Oschman for his effort to establish a solid scientific basis for Energy Medicine.

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ABSTRACT
The atrioventricular node (AV) is considered the electrical connection between the atria and ventricles. There is an electrical pause between activation of the atria and the ventricles (PR segment), but to date the mechanism responsible for this interruption remains unclear. The present communication focuses on the hypothesis that magnetic field dynamics could provide the answer. Proof of this hypothesis is that in Wolff-Parkinson-White syndrome, where there is physical connection between the atria and ventricles (bundle of Kent), there is electrical AV continuity, no PR segment is detected, and catheter ablation of the abnormal bundle restores AV discontinuity. Spontaneous initiation of the heart at the level of the sinus node, the pacemaker of the heart, could also be explained via field dynamics. The known transmembrane pacemaker protein CHN4, present in both sinoatrial and AV nodal cells, could interact with field information to provide specificity in an electronic key-to-lock mechanism interaction.

“How is that electricity coming from the atria suddenly stops at the atrioventricular node before it gets to the ventricles, as if having a break for coffee?” Paco Torrent-Guasp MD

The atrioventricular (AV) node is considered the electrical connection between the atria and the ventricles. From the electrocardiographic point of view, it shows as a pause between the end of electrical atrial activation (P wave) and the beginning of ventricular activity (QRS wave), the so-called PR segment, which usually lasts 50–120 ms. This provides the necessary delay between atrial and ventricular contraction to optimize cardiac function.

The mechanism responsible for such AV is unclear. Initially suggested that there is a decremental electrical conduction, studies using sensitive techniques have shown that there appears to be an interruption of the flow of electricity across the AV node [1–3]. How to explain that electrical wavefront from the atria suddenly stops at a certain point at the AV node before proceeding to the ventricle? The hypothesis...
presented herein is that the phenomenon can be understood considering field dynamics.

Field dynamics

A field is an area of influence. The cardiac field is considered the most powerful source of magnetism in the body [4]. Thanks to the pioneer efforts of several investigators in the 20th century we now know the clinical relevance of magnetic fields in human biology. Harold Saxton-Burr measured electromagnetic fields in multiple living systems, and showed that the information necessary for tissue growth, development, repair, renewal and death is in the field [5]. He emphasized that the electromagnetic field is not a by-product of the biological process but is the first occurring event. Robert O. Becker showed that magnetic fields are an integral part of human biology. For example, bone healing is based on magnetic field dynamics [6].

Fields provide information for tissues to work properly (form, function, renovation, repair, death) [5,6], but how they contain such information is uncertain. Another intriguing aspect is that they cannot be traced in space or time (non-local) [7,8]. In addition, fields are coherent (in phase) [9] and deeply interconnected, tying one part of the universe to every other part [10,11]. Nobel laureate Erwin Schrödinger coined the term entanglement to describe this feature [12].

Fields interact with body tissues, that capture information due to their coil array structure (which operate as antennas exquisitely sensitive to weak magnetic fields) and converts magnetism to electricity via Faraday's law [13].

Electrophysiology of AV node: the field hypothesis

The heart has its own electrical conduction system. Spontaneous activation of the sinoatrial node, the pacemaker of the heart, spreads the electricity through the atria (P wave) after which the electrical activity comes to a halt (PR segment) before the ventricles activate (QRS and T waves). No satisfactory explanation is put forward as to how electricity, that stops at the boundary of the AV node, proceeds to activate the ventricles. Sequential magnetic fields captured by the conduction system of the heart and converted to electricity could explain the continuity of the electrical flow from the atria to the ventricles (Fig. 1). In this way, field dynamics would fill the gap of the electrical conduction of the heart at the level of the AV node. Proof of this assumption is that PR segment is existent when abnormal muscular fibres (bundle of Kent) that connect the atria with the ventricles remain after birth (Wolff-Parkinson-White syndrome). After catheter ablation the electrical gap re-established [14] (Fig. 2).

The natural, “spontaneous”, excitation of the heartbeat

Field dynamics at the AV node could also be applicable to the origin of the “spontaneous” initiation of the cardiac impulse in the sinoatrial node, the natural pacemaker of the heart. The molecular basis for pacemaker activity has been well established: a family of hyperpolarization-activated cation channel proteins (HCN) [15] elicit an electrical current called “funny current” which proceeds to activate the atria [16]. Expression of HCN4 is observed in both the sinoatrial and AV node [17]. Therefore, the “unknown mechanism” that activates HCN4 and starts cardiac pacemaker activity are likely to be non-local fields. Field-to-protein interaction would act as a specific electronic key-lock mechanism involving the initiation of activation of both sinoatrial and AV node. Recently, a protein complex has been described as a key piece of how animals detect magnetic fields [18] that would be specific for each species [19]. The required specificity of such electronic interaction would be provided by the CHN4 protein. The intriguing differences in PR between species [20,21] (Fig. 3), could be explained by species-specific field dynamics [22]. Therefore, the AV delay would be determined by specific field concatenation for each species.

The atrioventricular dynamics in atrial arrhythmias

Explanations of several rhythm disturbances in the setting of AV disconnection are needed. These should consider: a) impairment of field-to-protein interaction as the source of disease, either because the field is abnormal (solar and geomagnetic environment [23], the HCN4 protein cannot be adequately synthetized [24], or sinoatrial tissue disease of the node (rheumatic disease) which could impair the generation of the funny current); b) local regulation of the rhythm (autonomic nervous system or drugs such as digoxin) in the field-to-protein feedback and rhythmicity; c) in atrial fibrillation, rhythm irregularity is also reflected at the ventricular level; incoherent cardiac field could directly affect both sinoatrial and AV node and provide an explanation;
d) the ratios between atrial and ventricular rhythms observed (2:1, 3:1, 4:1) in certain fast atrial rhythms is intriguing. In fact, heart beat is far from a linear phenomenon, but has a multifractal, chaotic, non-linear nature [25,26]. Understanding synchrony of AV field dynamics in atrial arrhythmias presents a formidable challenge to be solved by experts in non-linear dynamics: fractal analysis of the heart beat has been shown to predict the onset of paroxysmal atrial fibrillation [27].

In summary, the sequence of AV could have its origins in fields converted to electricity. The specific field-to-electricity interface could be the pacemaker protein, HCN4, expressed in both sinoatrial an AV node cells. This could explain activation of the sinoatrial node and the AV “jump” via field dynamics and clarify the basis for different AV delays between species.

References


Fig. 2. Schematic representation of the effect of anomalous atrioventricular fibers (bundle of Kent) in a patient with Wolff-Parkinson-White before (left figure) and after radiofrequency catheter ablation (right figure) (8). Reappearance of AV electrical discontinuity confirms that in normal hearts AV node does not conduct electricity from the atria to the ventricles. A) Cardiac field stimulating the sinoatrial node. B) Ventricular field stimulating AV node.

Fig. 3. PR segment in different species prompted Meijler to suggest that only electricity could not explain these differences (20, 21). Field dynamics proposed to be species-related could provide the answer (22). The timing of atrial to ventricular activation (the PR segment) would be determined by the specific atrio-ventricular field concatenation for each species.

PR interval [ms]

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