Fluid Mechanics: Open Access
ISSN: 2476-2296

Perspective Open Access

Ballester-Rodés and Others - A New Look at the T Wave

Manel Ballester-Rodés[1]*, Francesc Carreras[2], Jagat Narula[3] and James L Oschman[4]

[1]*Department of Medicine, Faculty of Medicine, University of Lleida (Catalunya), Spain*
[2]*Cardiac Imaging Unit, Hospital de la Santa Creu i Sant Pau, Universitat Autònoma de Barcelona, Spain*
[3]*Department of Cardiology, Mount Sinai Medical Center, New York, USA*
[4]*Nature's Own Research Association, Dover, New Hampshire, USA*

Introduction

Over a century ago a Dutch physician, Willem Einthoven, developed a galvanometer that could record the voltages produced during the cardiac cycle using electrodes placed on the body surface [1,2]. Einthoven assigned the letters P, Q, R, S and T to the various deflections, a terminology that is still in use today. His seminal discovery eventually led to the clinically useful field of electrocardiography, and Einthoven received the Nobel Prize in Medicine in 1924.

Physicians rely on the electrocardiogram (ECG) for the diagnosis of a variety of cardiac diseases, which reveal themselves characteristically in the electrocardiogram. However, few physicians and few cardiologists realize that our understanding of how the electrocardiographic signals are produced and propagated to the skin surface is incomplete. The T wave is especially important clinically because it is dramatically altered when there are cardiac abnormalities, but its relationship to electrical activity at the cellular level is poorly understood [3].

An early question in cardiology was precisely how the electrical fields produced during the beating of the heart travel to the surface of the body. In 1913, Einthoven and colleagues made the simplifying assumption that the human body is a homogeneous "volume conductor" with the heart's electricity conducted through tissues, with dissolved electrolytes serving as the charge carriers [4]. This overly simplistic model was useful in the early stages of research on the electrocardiogram.

The volume conductor assumption continues to dominate electrophysiology. For example, a recent treatise on electromagnetic field effects summarizes "electrical transport within tissues" as follows: "The fundamental bioengineering perspective is that the human body is considered to be a compartmentalized (or lumped element) conducting dielectric". It consists of about 60% of water by weight, in which 33% is intracellular and 27% is extracellular. Body fluid in both the intracellular and the extracellular compartments is highly electrolytic, and these two compartments are separated by a relatively impermeable, highly resistive plasma membrane. Current within the body is carried by mobile ions in the body fluid [5].

With this approximation, the various organs and layers of tissue are "lumped" together, essentially disregarding anatomy, histology, and the dielectric properties of connective tissues. This is a classic example of "meaning invariance" [6-9]. It is a problem that occurs again and again in science when tentative assumptions, useful in the early stages of an investigation, gradually come to be taken as facts. Reliance on the volume conductor assumption has encouraged the use of approximations that affect virtually every aspect of physiology and medicine. The problem is that the diffusing hydrated ion and molecular charge transfer complex are simply too large to move fast enough through tissues to explain the speed and subtlety of living processes, including the electrocardiogram.

Controversy about the electrocardiogram began during the 1930's, when physiologists looked more carefully at the mechanisms of conduction of cardiac electricity. It was realized that the electrical pathways through the body to the sensing electrodes are anatomically intricate, and that each tissue has a different conductivity [10-12]. These factors are neglected in the volume conductor model. Confusion about the precise nature of the electrocardiogram persists to this day, and extends to many other biological phenomena involving charge transfer.

A further complication arises because the myocardium has traditionally been viewed as having a more or less homogenous morphology. This assumption dates to the 17th century, when physician William Harvey described the circulatory system [13]. His simplistic anatomical perspective, which is widely accepted to this day, was that the heart is a single homogenous muscle. The ventricles, however, had posed profound mysteries for almost five centuries, and were referred to in 1864 by the well-known British professor of Anatomy, James Bell Pettigrew as a 'Gordian Knot', a term that is often used as a metaphor for an intractable problem (as disentangling a "hopelessly impossible" knot in a rope) [14]. After some 50 years of research, Spanish Professor Torrent Guasp untangled the ventricular knot for the first time, discovering that the 3D configuration of the ventricles is a double helix, known as the helical ventricular myocardial band (HVMB) [15]. Guasp's discovery has been confirmed, and has led us to reconsider the significance of the T wave.

The Purkinje system is isolated from the surrounding myocardium and provides a fast means to simultaneously electrify both ventricles to stimulate contraction. In humans the Purkinje system starts at the level of the atrioventricular (AV) node, branches via the right and left bundle bundles and fans subendocardically, spreading in a caudal way to the right and left ventricles and then ascends towards the base of the heart. It is difficult to accurately trace the distal connections of the bundle-branch system because the Purkinje fibers penetrate the subencocardioum and myocardium for varying distances, depending on the species [16-18]. The QRS wave of the electrocardiogram corresponds to the electrical activation of the Purkinje system and is referred to as depolarization. It is thought that the electrical impulse follows a radial distribution from endocardium to epicardium, after which repolarization follows, giving rise to the T wave. Interpretation of the QRS complex follows from understanding of how depolarization takes place: from the septum, towards the apex, to both ventricles and eventually the base of the heart [19,20]. The spatial sequence of the repolarization process has never been fully explained.

***Corresponding author:** Oschman JL, Nature's Own Research Assolciation, Dover, New Hampshire, USA, Tel: +603-742-3789; E-mail: joschman@aol.com

Received July 15, 2016; **Accepted** July 22, 2016; **Published** July 29, 2016

Citation: Ballester-Rodés M, Carreras F, Narula J, Oschman JL (2016) Ballester-Rodés and Others - A New Look at the T Wave. Fluid Mech Open Acc 3: 128.

Assumptions Revisited

Interpretation of QRS and T waves of the ECG are based on several assumptions: 1) The myocardium is an homogenous structure; 2) radial distribution, from the endocardium to the epicardium, is the basis of the electrical propagation through the myocardium; 3) mechanical activation of the myocardium topographically follows sequence of its electrical activation; 4) the T wave reflects the electrical recovery phase of the myocardium, the repolarization; 5) blood flow in the aortic arch is laminar.

In the next paragraphs, several considerations are put forward that question these assumptions.

The myocardium is not an homogenous structure

Recent discovery of the myocardium as a continuous double helical structure which folds on itself has been a major breakthrough [21,22]. Manual dissection of the myocardial anatomy as a continuous single band (Figures 1 and 2) questions the classical division of the right and left ventricles as unrelated structures. Actually, the myocardium is formed by a superposition of muscular layers that are complexly interwoven [23] (Figures 3a and 3b). As an example, the interventricular septum is formed by an endocardial helical descendent muscular segment that crosses at approximately a 60º angle with the ascendant epicardial one (Figure 4). Recently, automated analysis of myocardial fibre orientation by diffusion tensor magnetic resonance multi-resolution tractography imaging has confirmed the continuous double helical ventricular myocardial fibre arrangement [24-26] (Figure 5). Thus, the complexity of ventricular myofibre direction, which had challenged anatomists and physiologists for centuries [27,28] can be explained by the spatial architecture of a double helicoid. The anatomical findings of Torrent-Guasp are therefore confirmed.

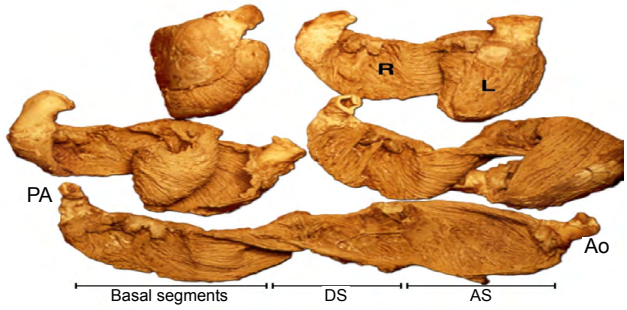


Figure 1: Different stages of the dissection of the myocardial band [21,22]. Myocardial muscle as a continuous structure that spans from the pulmonary artery (PA) to the aorta (Ao). The basal segments and right (R) and left (L) ventricles. At this point the band twists 180% and a descendent segment (DS) spirals down to the apex, where a sudden twist takes place to the ascendant segment (AS), which spirals up to the aorta.

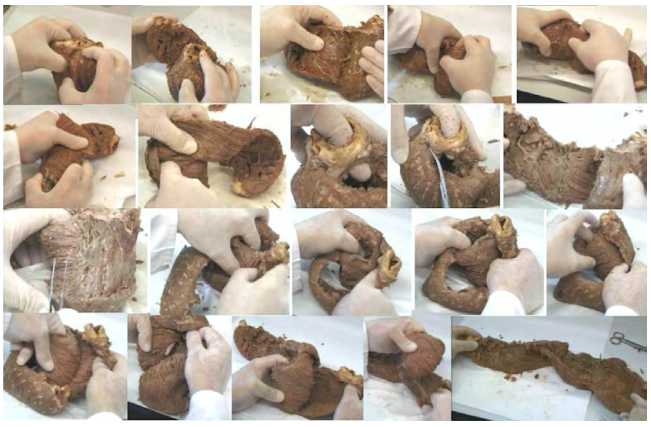


Figure 2: Detailed procedure for the dissection of the myocardial band of a bovine heart [23].

Figure 3: a) Figure shows a bovine heart that has been unrolled, coloured, rewrapped and sliced following a 4-chamber section; b) several transverse sections at the base (top) as seen from the apex, midventricle, and near the apex (bottom). Note the different layers. The right ventricle (blue) and the base of the left (red) form the base of the heart. The inner portion is the descendant segment of the band (yellow), which occupies the subendocardium and spirals down to the apex . At this level a sudden change in orientation gives rise to the ascendant segment (green) which in a spiral way ends at the aorta [23].

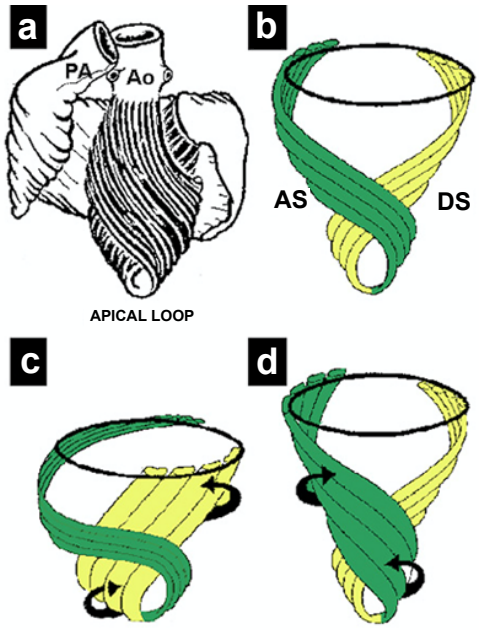


Figure 4: a, b) schematic representation of the descendant (DS) and ascendant (AS) segments of the myocardium. Explanation: ascendant and descendent segments cross at 60º angle at the level of the septum. c) Fiber arrangement in systole, showing contraction of the descending segment and elongation of the ascending segment, with downward displacement of the base of the heart. d) In diastole, the ascending contracts and the base is lifted upward.

Radial distribution is not the basis of the electrical propagation from the endocardium to epicardium

Study of propagation of the electrical impulse that triggers contraction of the myocardial fibres was developed before the helical structure of the myocardium was known. The concept of radial propagation from the endocardium to the epicardium stems from the belief that the Purkinje system penetrates the cardiac muscle from the endocardium towards the epicardium (Figures 6a and 6b). This has provided the basis of the ECG interpretation following a radial depolarization process [19]. But in fact the Purkinje system in humans is subendocardial [29] and does not penetrate the myocardium. This differs from other species (birds) in which Purkinje fibres do penetrate the full thickness of the myocardium, and explains the very high cardiac frequencies attained (1200 pm) [30].

It is well established that propagation throughout myocardial fibers is anisotropic, meaning that it preferentially follows the longitudinal arrangement of the rod-like shaped myocytes, and that propagation velocity in this direction is maximum [31]. In the face of the complex interwoven ventricular anatomy of the helical heart it seems unlikely that the endocardial stimulus follows a radial distribution to the epicardium crossing muscular layers which are differently oriented, therefore in a non-anisotropic pathway. But how can we explain the well-known timing of endocardial to epicardial propagation? The answer lies in the helical anatomy and the sequential activation, first via the subendocardial descendent segment, followed by subsequent activation of the epicardial ascendant segment, as first suggested by Torrent-Guasp [21] and shown in the laboratory using microcrystals oriented according to the helical segments [32] (Figure 7) and myocardial 3D displacement fields captured with DENSE MRI [33]. A recent computer model of propagation of the helical model [34] also supports this pattern of electromechanical propagation.

The topographic sequence of electrical activation of the myocardium (QRS) is unrelated to the sequence of electromechanical activation

Electrical propagation via Purkinje system, which is responsible for the QRS complex, is indeed a very fast event, usually 80 ms [35]. No mechanical activity is observed at that time. Therefore, QRS reflects the quick electrification process of the ventricular myocardium rather than

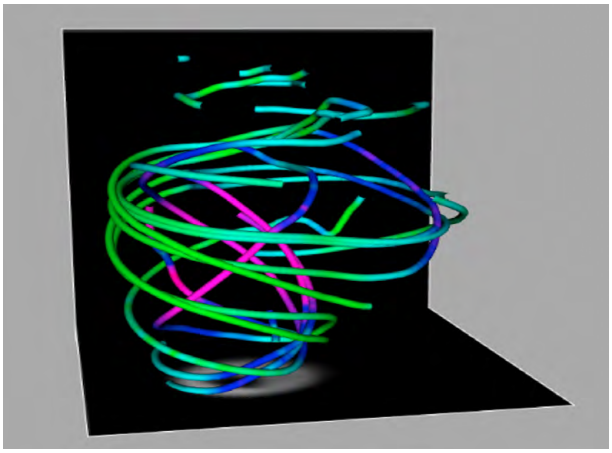
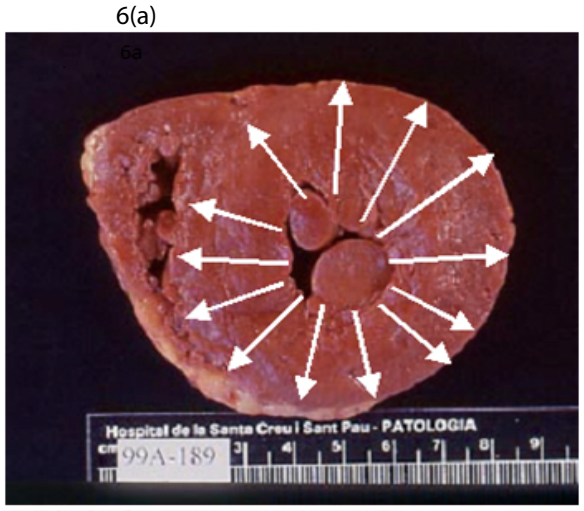


Figure 5: Simplified tractography reconstruction from diffusion tensor imaging MRI depicting the continuous double helical ventricular arrangement of the myocardial fibres [24-26].



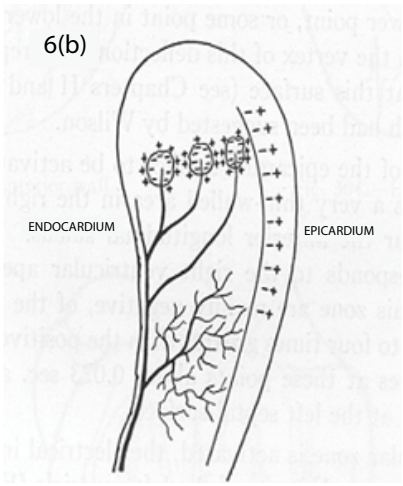


Figure 6: a) the radial theory of electrical propagation from subendocardium to subepicardium, based on b) the intramyocardial penetration of the Purkinje system thought to occur in humans [19].

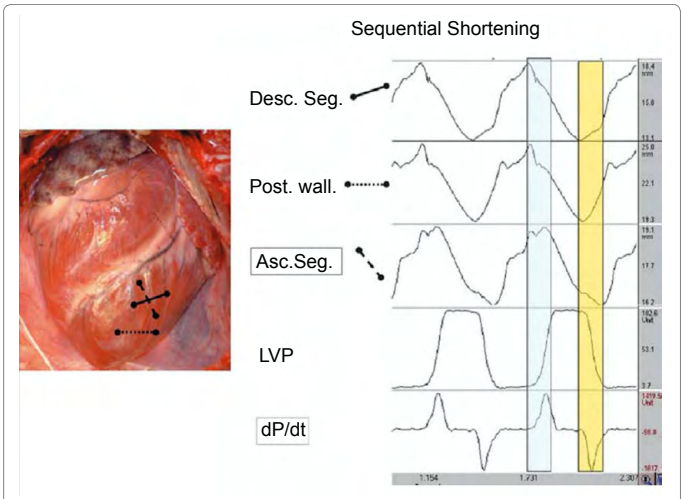


Figure 7: Explanation of the endocardial to epicardial activation in the helical heart [32]. Microcrystals have been implanted at the endocardium, aligned with the descendant segment, and at the epicardium, aligned with the ascendant segment. Note that the sequence of events of propagation has an endocardium-epicardium direction through the helical anatomy.

the effects of propagation through myocytes, which is a much slower process (300-400 ms). It is well known that electrical diffusion follows a septum-apex-base direction [36]. The rapid electrification of the heart is mediated through the conduction system fibres of the Purkinje network. Purkinje fibers are surrounded by a fine fibrous sheath that progressively loses its interaction with the working myocardium [37], and once the electrical stimulus has been delivered at the cardiac muscle, in a way that is still poorly understood [38] electromechanical anisotropic propagation through myocardium follows.

Existing evidence suggests that the starting point in the myocardium is not a single one, but two topographically different areas appear to be the recipients of the electrical stimulus: at the level of the infundibulum of the right ventricle and the base of the left ventricle [39-41]. From the moment the electrical impulse attains the myocardium, the electromechanical impulse anisotropically follows the myocardial fibre tracts, first from base of the heart, where it proceeds to the subendocardium (descendant helical segment) and later the subepicardium (ascendant helical segment) in a sequence that explains systole (counterclockwise twist of the base of the heart and clockwise twist of the apex) and diastole, which reverses these torsion movements [42-44] (Figure 8).

The T wave Coincides with the Electromechanical Activation

Correlation between the myocardial mechanical activity and the T wave reveals a temporal coincidence (Figures 9, 10a and 10b). Indeed, propagation of the electromechanical stimulus, as assessed by magnetic resonance imaging [46] and speckle tracking echocardiography [47,48] reveals a close overlap between the muscular activity and the T wave.

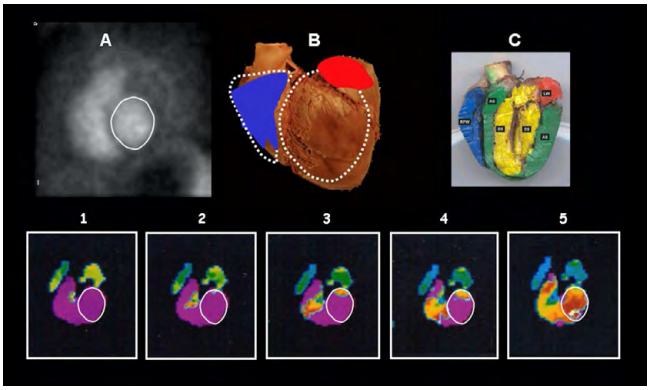


Figure 8: Base-to-apex mechanical propagation of myocardial activation as assessed by Fourier analysis of blood-pool ventricular isotopic studies [40,45]. A) Isotopic blood pool image of the ventricles in LAO view. In circle the left ventricular cavity. B) A pig heart has been unwrapped, dissected and rewrapped. In blue, the right ventricle –which corresponds to the right segment; in red, the basal portion of the left ventricle, which corresponds to the left segment. C) A four chamber section of a heart, as described in figure 3A, which illustrates the position of the basal loop of the helical heart: blue –right segment- and red –left segment. Number 1 to 5 correspond to the Fourier analysis of ventricular motion during ventricular activation: 1) the first portion to be activated is at the level of the pulmonary infundibulum, beneath the pulmonary valve. At that time no signal from the left ventricle is elicited; 2) The signal from the right ventricle increases and there is a minor dot at the basal portion of the left ventricle indicating the initial process of activation; 3 and 4) The right ventricle signal is increasingly expanding to the right ventricle and the basal portion of the left is now activated, but the signal has not propagated to the body left ventricle. 5) Eventually the full myocardium of the right ventricle is fully activated as is the left ventricle.

Blood flow in the aortic arch is laminar

Several studies have shown that both the heart and the vessels connected to it twist with each heartbeat [49]. The vortical structure and dynamics of the ventricular myocardial band continue into the aortic arch and arteries. The blood spirals through the aorta and beyond, into the arterial tree and all the way to the pre-capillaries (Figure 13a). And arterial endothelial cell orientation closely follows these blood flow patterns [50]. This type of vortical charge movement is similar to that taking place in a coil or solenoid (Figure 13b). From the electrical engineering perspective, this should amplify the electromagnetic field produced by the heart.

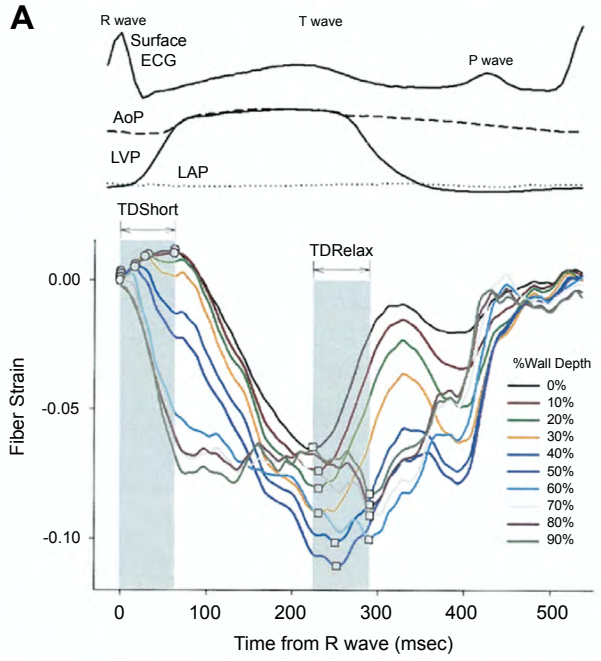


Figure 9: Time course of transmural myocardial fiber strain correlated with the T wave. Note that there is a temporal coincidence with both events [46].

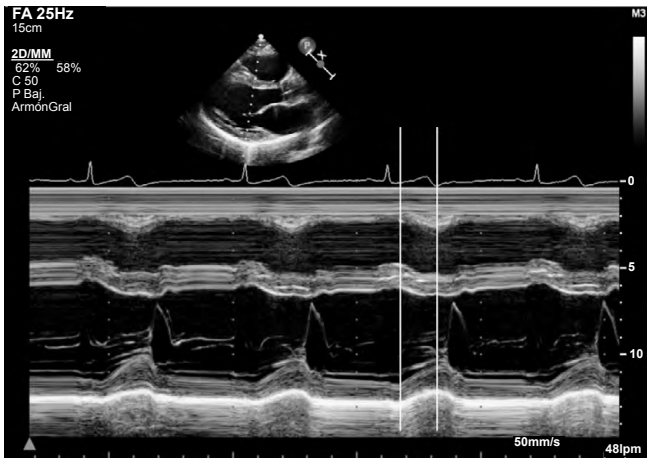


Figure 10: a) Correlation between T wave and mechanical activity as assessed by M-mode echocardiography: ventricular systolic motion coincides with T wave activity of the ECG (between vertical lines).

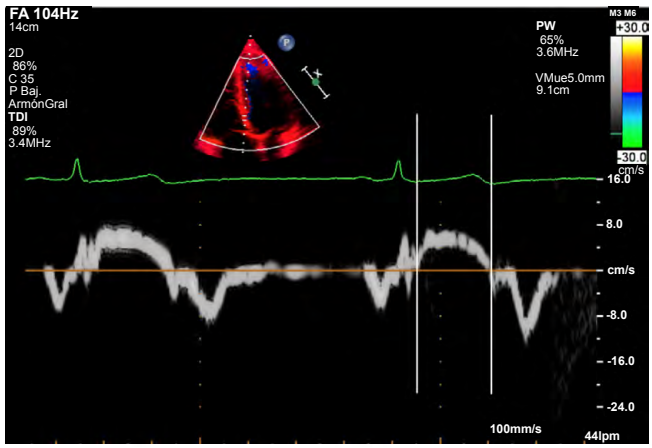


Figure 10: b) Tissue Doppler imaging of the base of the heart which reveals that ventricular motion of the septum coincides with the T wave (between vertical lines).

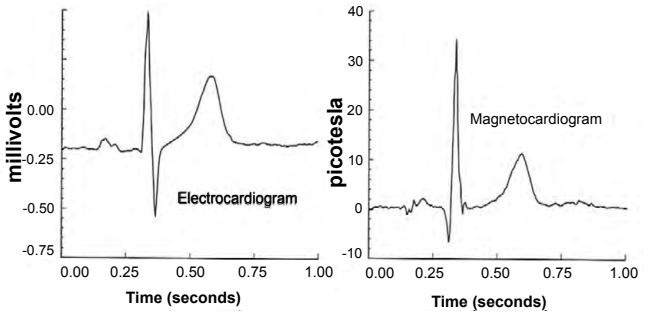


Figure 11: ECG and SQUID detection of magnetic activity associated with the cardiac cycle [55]. The identical pattern of surface ECG and the magnetocardiogram, which picks up the cardiac electromagnetic signal at a distance, reveals the magnetic nature of the ECG.

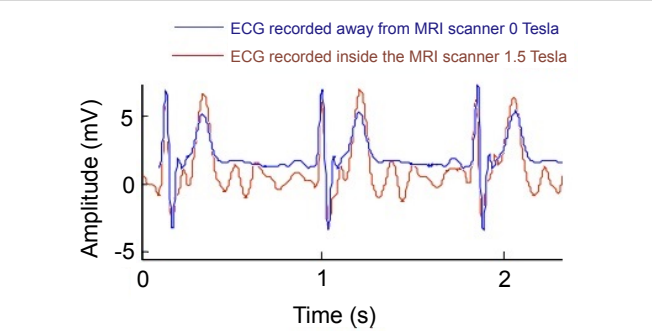


Figure 12: Magnetohydrodynamic effect on ECG and MRI. The artifact which makes it difficult to trigger the MRI scan, thought to be related to the magnetic field of the moving blood and muscle, closely overlaps the T wave [59].

Stonebridge and Brophy performed direct angioscopic examination in the lower extremities and found that the inner surface of the arteries is organized in a series of spiral folds that sometimes protrude into the lumen. These folds are probably a consequence of spiral blood flow. Vortexing blood was actually observed with fiber optics in the region of these endoluminal folds. It was suggested that this type of vortex flow may be more efficient, requiring less energy for the blood to move through the distal tapering and branching arterial system [51]. Rifled gun barrels cause bullets to spin, making them more stable in flight and therefore improving accuracy. In the vessels the blood appears to form its own grooved conduits to support torsional flow. The spiral folds are not found in excised arteries or cadavers; they are dynamic features of living tissues.

Physiological helices may help to stabilize flow by reducing turbulence, preserving energy, and protecting vessels from atherosclerosis [52].

What does the T wave Reflect?

The ECG tracing reflects changes in the electromagnetic activity of the heart. It is usually detected by electrodes placed on the chest, but an identical signal can also be detected at a distance by a sensitive magnetometer (SQUID detector) [53-55] (Figure 11). Any mechanical activity is invariably associated with an electromagnetic field [56] and electromechanical propagation through the myocardium should have its electromagnetic correlate. We believe that the T wave might reflect the electromagnetic field associated with the mechanical activity of the working myocardium and the blood motion. In fact, this has been shown in magnetic resonance studies, where myocardial and blood motion induces the so called the magnetohydrodynamic effect, a signal superimposed on the T wave of the ECG. This effect makes it challenging to synchronize the MRI scan with the ECG [57-59]. In such tracings, the magnetohydrodynamic effect waves closely overlap the T wave, as seen in Figure 12.

Cellular depolarization, repolarization and myocardial electromechanical activation: do they reflect the same phenomena?

Much of the equivocal concepts in ECG interpretation probably stem from the fact that the electrophysiological events shown in a single cell have been equated with the electromechanical events occurring at the myocardial level. The terms cellular depolarization and repolarization have been assumed to have electromechanical correlates. And this is probably a major source of confusion. In order to clarify these issues, a new way to interpret the ECG events is suggested (Table 1).

Implications

In the light of the new ventricular anatomy and function [60] interpretation of the ECG should be revisited. The terms depolarization and repolarization should best be limited to cell physiology rather than to myocardial activity. This new vision -and division- of the electrical and electromechanical phenomena brings up questions regarding the

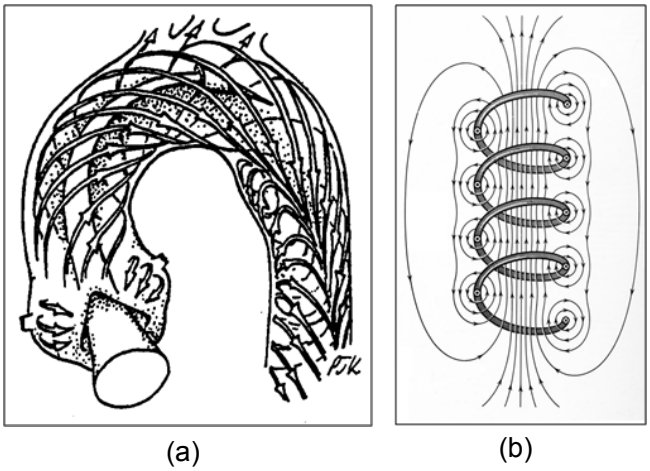


Figure 13: a) Vortical flow pattern in the aortic arch. The heart electricity is thought to be conducted through the circulatory system by ions in the blood, and the conductor itself is moving helically. b) From an electrical engineering perspective, this resembles a coil or a solenoid that amplifies the electromagnetic output.

ECG	Duration	Reflects
P wave	50 ms	Electromagnetic field corresponding to the atrial activation
QRS complex	80 ms	Electromagnetic field corresponding to the ventricular electrical delivery (via Purkinje) to sites of initial ventricular propagation
T wave	300-400 ms	Electromagnetic field associated with ventricular electromechanical propagation and/or blood motion

Table 1: Proposed interpretation of the ECG phenomena.

nature of ECG changes in health and disease and opens the way to future research.

Summary

Depolarization and repolarization are electrophysiological terms that describe changes in a single cell´s membrane potential due to ionic movements. Membrane potentials transiently change from inside negative to positive due to influx of positive ions into the cell while activated, and reverse to attain the resting state.

These phenomena have been extrapolated from a cellular level to the entire myocardium to form the basis for the interpretation of the electrocardiogram (ECG). Thus, the QRS complex of the ECG is thought to reflect the depolarization and the T wave the repolarization.

The sequence of events that explains the morphology of the QRS is the electrical activation of the myocardium beginning with the pacemaker cells in the sinoatrial node. The wave of depolarization spreads out through the atrium, passes through the atrioventricular node and down into the left and right bundle of His to the respective Purkinje fibers for each side of the heart, and then to the endocardium at the apex of the heart, then finally throughout the ventricular epicardium. The T wave is deemed to reflect the electrical events associated with recovery from this process.

The myocardium has traditionally been viewed as a homogenous morphological structure. However, discovery of the complex anatomical 3D configuration of the ventricles as a double helix, known as the helical ventricular myocardial band (HVMB), prompts a reconsideration of the origin of the T wave.

The 3D-helical model of the heart has been confirmed. Its electrical, electromechanical and functional implications are the basis for a new interpretation of the T wave. We believe that the T wave might reflect the electromagnetic field associated with the mechanical activity of the working myocardium and the vortical motion of the blood through the aortic arch.

References

1. Einthoven W (1903) Ein neues Galvanometer. Annalen Der Physik 317: 1059-1071.
2. Einthoven W (1906) Weitere Mitteilungen über das Saitengalvanometer. Analyse der saitengalvanometrischen Kurven. Masse und Spannung des Quarzfadens und Widerstand gegen die Fadenbewegung. Annalen Der Physik 326: 665-700.
3. Kootsey JM, Johnson E (1980) The origin of the T-wave. Critical reviews in bioengineering 4: 233-270.
4. Einthoven W, Fahr G, de Waart A (1913) Über die Richtung und die manifeste Grösse der Potentialschwankungen im menschlichen Herzen und über den Einfluss der Herzlage auf die Form des Elektrokardiogramms. Pflugers Archiv fur die gesamte Physiologie 150: 275-315.
5. Lee RC, Bodna EN, Betala P, Bloom-Eberwein S (2006) Electrical shock trauma. In: Barnes FS Greenbaum B (eds.) Handbook of Biological Effects of Electromagnetic Fields (3rd edn). CRC Press, Boca Raton, FL.
6. Feyerabend P (1985) Realism, Rationalism and Scientific Method. Philosophical Papers, Cambridge University Press, Cambridge, UK.
7. Northrop FSC (1959) The Two Kinds of Deductively Formulated Theory. The Logic of the Sciences and the Humanities. Meridian Books, New Haven, CT. p. 102.
8. Oschman JL (2008) Perspective: Assume a spherical cow: The role of free or mobile electrons in bodywork, energetic and movement therapies. Journal of Bodywork and Movement Therapies 12: 40-57.
9. Oschman JL (2008) Charge transfer in the living matrix. J Bodyw Mov Ther 13: 215-228.
10. Collin RE, Plonsey R (1978) A note on dipole sources in conducting biological tissues. Bulletin of Mathematical Biology 40: 201-209.
11. Eyster JAE, Maresh F, Krasno MR (1933) The nature of the electric field around the heart. American Journal of Physiology 106: 574-598.
12. Katz LN, Korey H (1935) The manner in which the electric currents generated by the heart are conducted away. American Journal of Physiology 111: 83.
13. Harvey W (1628) Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus (Latin for "An Anatomical Exercise on the Motion of the Heart and Blood in Living Beings"). Springfield Ill, Thomas, USA.
14. Pettigrew JB (1864) On the arrangement of the muscular fibres in the ventricles of the vertebrate heart, with physiological remarks. Philosophical Transactions of the Royal Society of London 154: 445-500.
15. Torrent-Guasp F, Buckberg GD, Clemente C, Cox JL, Coghlan HC, et al. (2001) The structure and function of the helical heart and its buttress wrapping. I. The normal macroscopic structure of the heart. Semin Thorac Cardiovasc Surg 13: 301-319.
16. Waller BF, Gering LE, Branyas NA, Slack JD (1993) Anatomy, histology, and pathology of the cardiac conduction system-part II. Clincal Cardiology 16: 347-352.
17. Ekušja O (2006) Purkinje fibers of the heart conduction system. The history and present relevance of the Purkinje discoveries. Cas Lek Cesk 145: 333.
18. Sánchez-Quintana D, Siew YH (2003) Anatomy of Cardiac Nodes and Atrioventricular Specialized Conduction System. Rev Esp Cardiol 56: 1085-1092.
19. Sodi-Pallares D (1956) New Bases of Electrocardiography. The Mosby Co. St. Louis. p. 389.
20. Durrer D, van Dam RT, Freud GE, Janse MJ, Meijler FL, et al. (1970) Total excitation of the isolated human heart. Circulation 41: 899-912.
21. Torrent-Guasp F (1998) Structure and function of the heart. Rev Esp Cardiol 51: 91-102.
22. Torrent-Guasp F, Ballester M, Buckberg GD, Carreras F, Flotats A, et al. (2001) Spatial orientation of the ventricular muscle band: physiologic contribution and surgical implications. J Thorac Cardiovasc Surg 122: 389-392.
23. Ballester M, Ferreira A, Carreras F (2008) The myocardial band. Heart Failure Clinics 4: 261-272.
24. Poveda F, Martí E, Gil D, Carreras F, Ballester M (2012) Evidence of global helical structure of the heart by multiresolution tractography study of DT-MRI. Journal of the American College of Cardiology Imaging 5: 754-64.
25. Poveda F (2013) Computer Graphics and Vision Techniques for the Study of the Muscular Fiber Architecture of the Myocardium. PhD Thesis. Universitat Autònoma de Barcelona.
26. Poveda F, Gil D, Martí E, Andaluz A, Ballester M, et al. (2013) Helical structure of the cardiac ventricular anatomy assessed by diffusion tensor magnetic resonance imaging with multiresolution tractography. Rev Esp Cardiol 66: 782-790.
27. Lower R (1669) Tractatus de Corde. Item de motu & colore sanguinis et chyli in eum transitu., Londini: Typis Jo. Redmayne impensis Jacobi Allestry.
28. Mall FP (1911) On the muscular architecture of the ventricles of the human heart. American Journal of Anatomy 11: 211-278.
29. Coghlan HC, Coghlan AR, Buckberg GD, Gharib M, Cox JL (2001) The structure and function of the helical heart and its buttress wrapping. III. The electric spiral of the heart: The hypothesis of the anisotropic conducting matrix. Semin Thorac Cardiovasc Surg 13: 333-341.

30. Davis F, Francis ETB (1946) The conducting system of the vertebrate heart. Biological Reviews 21: 173-188.

31. Rubart M, Zipes DP (2001) Genesis of Cardiac Arrhthmias: electrophysiological considerations. In: Braunwald E Zipes DP Libby P (eds) Heart Disease. Philadelphia, WB Saunders Company. pp: 659-699.

32. Buckberg GD, Clemente C, Cox JL, Coghlan HC, Castella M, et al. (2001) The structure and function of the helical heart and its buttress wrapping. IV. Concepts of dynamic function from the normal macroscopic helical structure. Semin Thorac Cardiovasc Surg 13: 342-357.

33. Nasiraei-Moghaddam A, Gharib M (2009) Evidence for the existence of a functional helical myocardial band. Am J Physiol Heart Circ Physiol 296: 127-131.

34. Marcè-Nogué J, Fortuny G, Ballester-Rodés M, Carreras F, Roure F (2013) Computational modeling of electromechanical propagation in the helical ventricular anatomy of the heart. Computers in Biology and Medicine 43: 1698-1703.

35. Selvester RH, Velazquez DW, Elko PP (1990) Intraventricular conduction defect (IVCD), real or fancied: QRS duration in 1254 normal adult white males by a multilead automated algorithm. J Electrocardiol 23: 118-122.

36. Durrer D, van Dam RT, Freud GE, Janse MJ, Meijler FL, et al. (1970) Total excitation of the isolated human heart. Circulation 41: 899-912.

37. Anderson RH, Becker AE (1980) The conduction system. In Anderson RH Becker AE (eds) Cardiac Anatomy: An Integrated Text and Color Atlas. London, Gower Medical Publishing. pp: 615-629.

38. Coghlan HC, Coghlan AR, Buckberg GD, Gharib M, Cox JL (2001) The structure and function of the helical heart and its buttress wrapping. III. The electric spiral of the heart: The hypothesis of the anisotropic conducting matrix. Seminars in Thoracic and Cardiovascular Surgery 13: 333-341.

39. Cox JL (1983) Surgery of cardiac arrhyhtmias. Current Problems in Cardiology 8: 1-60.

40. Ballester-Rodés M, Flotats A, Torrent-Guasp F, Ballester-Alomar M, Carreras F (2005) Base-to-apex ventricular activation: Fourier studies in 29 normal individuals. European Journal of Nuclear Medicine and Molecular Imaging 32: 1481-1483.

41. Wyman BT, Hunter WC, Prinzen FW, McVeigh ER (1999) Mapping propagation of mechanical activation in the paced heart with MRI tagging. Am J Physiol 276: 881-891.

42. Ingels NB, Daughters GT, Stinson EB, Alderman EL (1975) Measurement of midwall myocardial dynamics in intact man by radiography of surgically implanted markers. Circulation 52: 859-867.

43. Lorenz CH, Pastorek JS, Bundy JM (2000) Delineation of normal human left ventricular twist throughout systole by tagged cine magnetic resonance imaging. J Cardiovasc Magn Reson 2: 97-108.

44. Carreras F, Garcia-Barnes J, Gil D, Pujadas S, Li C, et al. (2011) Left ventricular torsion and longitudinal shortening: two fundamental components of myocardial mechanics assessed by tagged cine-MRI in normal subjects. J Cardiovasc Imaging 28: 1-12.

45. Ballester-Rodés M, Flotats A, Torrent-Guasp F, Carrió-Gasset I, Ballester-Alomar M (2006) The sequence of regional ventricular motion. European Journal of Cardiothorac Surgery 291: 139-144.

46. Ashikaga H, Coppola BA, Hopenfeld B, Leifer ES, McVeigh ER, et al. (2007) Transmural dispersion of myofiber mechanics: implications for electrical heterogeneity *in vivo*. J Am Coll Cardiol 49: 909-916.

47. Sengupta PP, Krishnamoorthy VK, Korinek J, Narula J, Vannan VA, et al. (2007) Left Ventricular Form and Function Revisited: Applied Translational Science to Cardiovascular Ultrasound Imaging. Journal of the American Society of Echocardiography 20: 539-551.

48. Sengupta PP, Khanderia BK, Narula J (2008) Twist and untwist mechanics of the left ventricle. Heart Fail Clin 4: 315-324.

49. Frank AB, Penney DG, Marinelli WA, Marinelli R (1991) Rotary motion in the heart and blood vessels: a review. Journal of Applied Cardiology 6: 421-431.

50. Lowell BL, Adamson SL (1980) Relationship between blood flow direction and endothelial cell orientation at arterial branch sites in rabbits and mice. Circ Res 48: 481-488.

51. Stonebridge PA, Brophy CM (1991) Spiral flow in arteries? The Lancet 338: 1360-1361.

52. Kilner PJ, Yang GZ, Mohiaddin RH, Firmin DN, Longmore DB (1993) Helical and retrograde secondary flow patterns in the aortic arch studied by three-directional magnetic resonance velocity mapping. Circulation 88: 2235-2247.

53. Cohen D (1967) Magnetic fields around the torso: production by the electrical activity of the human heart. Science 156: 652-654.

54. Cohen D, Edelsack EA, Zimmerman JE (1970) Magnetocardiograms taken inside a shielded room with a superconducting point-contact magnetometer. Applied Physics Letters 16: 278-280.

55. Oschman JL (2000) Energy Medicine. The Scientific Basis. Churchill Livingstone/Elsevier, Edinburgh UK.

56. Wangsness RK (1986) Electromagnetic Fields (2nd edn). Wiley, New York, USA.

57. Tenforde TS, Gaffey CT, Moyer BR, Budinger TF (1983) Cardiovascular alterations in Macaca monkeys exposed to stationary magnetic fields: experimental observations and theoretical analysis. Bioelectromagnetics 4: 1-9.

58. Tenforde TS (2005) Magnetically induced electric fields and currents in the circulatory system. Prog Biophys Mol Biol 87: 279-288.

59. Abi-Abdallah D, Robin V, Drochon A, Fokapu O (2007) Alterations in human ECG due to the MagnetoHydroDynamic effect: A Method for Accurate R Peak Detection in the Presence of High MHD Artifacts. Conf Proc IEEE Eng Med Biol Soc. pp: 1842-1845.

60. Buckberg GD, Weisfeldt ML, Ballester M, Beyar R, Burkhoff D, et al. (2004) Left ventricular form and function: scientific priorities and strategic planning for development of new views of disease. Circulation 110: 333-336.