BIOPHYSICAL FOUNDATIONS OF ELECTROCARDIOGRAPHY (ECG) SIGNALS

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Abstract

In this thesis, the biophysical foundations of electrocardiography (ECG) signals are briefly analyzed at the cellular and tissue levels. The study explains how ionic currents of sodium (Na^+), potassium (K^+), and calcium (Ca^{2+}) generate cardiac action potentials and how these electrical impulses propagate through the cardiac conduction system. The relationship between depolarization–repolarization processes and the main ECG components (P wave, QRS complex, and T wave) is also described. The work highlights the importance of biophysical mechanisms for accurate ECG interpretation and clinical application.

Keywords. Electrocardiography (ECG), cardiac electrophysiology, action potential, ion channels, depolarization, repolarization.

Electrocardiography (ECG) is one of the most fundamental and widely used diagnostic tools in modern medicine. Since its clinical introduction by Willem Einthoven in the early 20th century, ECG has become indispensable for evaluating cardiac function, diagnosing arrhythmias, myocardial ischemia, conduction abnormalities, and other cardiovascular disorders. The technique is based on recording the electrical activity generated by the heart during its rhythmic contraction and relaxation cycles. Despite its routine use in clinical practice, the ECG signal itself is the result of complex electrophysiological and biophysical processes occurring at cellular, tissue, and organ levels. The human heart functions as an electromechanical pump. Its mechanical contraction is preceded and controlled by precisely coordinated electrical events. These electrical signals originate in specialized pacemaker cells located in the sinoatrial (SA) node, propagate through the atrial myocardium, pass the atrioventricular (AV) node, and travel along the His–Purkinje system to activate the ventricular myocardium. The coordinated depolarization and repolarization of millions of cardiac myocytes generate time-varying electric fields that spread through the conductive tissues of the thorax and can be detected on the body surface using electrodes.

From a biophysical perspective, ECG signals arise due to transmembrane ionic currents across cardiac cell membranes. These currents are primarily mediated by voltage-gated ion channels that regulate the movement of sodium (Na^+), potassium (K^+), and calcium (Ca^{2+}) ions. The resulting changes in

membrane potential create action potentials, which propagate through cardiac tissue as electrical waves. The summation of these electrical activities forms the measurable surface potentials recorded as ECG waveforms. Understanding the biophysical foundations of ECG requires integration of knowledge from electrophysiology, bioelectricity, membrane biophysics, and volume conductor theory. At the cellular level, cardiac action potentials are governed by ion channel kinetics and electrochemical gradients. At the tissue level, electrical propagation depends on intercellular coupling via gap junctions and the anisotropic structure of cardiac muscle fibers. At the organ level, the heart behaves as a dynamic electrical dipole whose orientation and magnitude change throughout the cardiac cycle. Finally, at the body level, the thorax acts as a volume conductor, shaping and attenuating the electrical signals before they reach surface electrodes.

Advancements in biomedical engineering and computational modeling have significantly enhanced our understanding of ECG signal formation. Mathematical models of cardiac electrophysiology, such as those derived from Hodgkin–Huxley-type formulations, allow simulation of ionic currents and action potential dynamics. Modern signal processing techniques further enable accurate analysis of ECG morphology, frequency components, and pathological deviations. The relevance of studying the biophysical basis of ECG signals extends beyond clinical diagnostics. It plays a critical role in the development of wearable cardiac monitoring devices, telemedicine systems, artificial intelligence-based diagnostic algorithms, and implantable cardiac technologies. A thorough understanding of ECG signal generation improves interpretation accuracy and helps differentiate physiological variations from pathological abnormalities. Therefore, the purpose of this article is to comprehensively examine the electrophysiological and biophysical mechanisms underlying ECG signal generation. By analyzing processes at the cellular, tissue, and systemic levels, this work aims to provide a scientifically grounded explanation of how electrical activity in the heart is transformed into the characteristic ECG waveform observed in clinical practice.

The foundation of ECG signal formation lies in the electrophysiological behavior of individual cardiac myocytes. Cardiac cells maintain a resting membrane potential of approximately –80 to –90 mV, primarily determined by potassium ion (K^+) permeability and the activity of the Na^+/K^+ ATPase pump. The electrical excitability of these cells is governed by voltage-gated ion channels embedded in the cell membrane. When a cardiac cell is stimulated, a rapid depolarization phase (Phase 0) occurs due to the opening of fast sodium (Na^+) channels, leading to an influx of Na^+ ions. This is followed by partial repolarization

(Phase 1), caused by transient outward potassium currents. The plateau phase (Phase 2), which is characteristic of cardiac muscle cells, results from a balance between inward calcium (Ca^{2+}) currents through L-type calcium channels and outward potassium currents. This plateau ensures prolonged depolarization, allowing sufficient time for myocardial contraction and preventing tetanic contractions. Repolarization (Phase 3) occurs when potassium efflux predominates, restoring the membrane potential to its resting level (Phase 4). These coordinated ionic movements generate local electrical currents that propagate to adjacent cells via gap junctions. The summation of millions of synchronized action potentials produces the macroscopic electrical activity detectable on the body surface.

Pacemaker cells in the sinoatrial (SA) node differ from contractile myocytes in that they exhibit spontaneous depolarization due to a slow inward “funny” current (If), primarily carried by sodium and potassium ions. This automaticity establishes the intrinsic rhythm of the heart and initiates each cardiac cycle. Electrical impulses generated in the SA node spread through atrial myocardium, reach the atrioventricular (AV) node, and then travel along the His–Purkinje system. The conduction velocity varies across different cardiac tissues. For example, Purkinje fibers conduct impulses rapidly (2–4 m/s), ensuring near-simultaneous activation of ventricular myocardium. Propagation depends on: Intercellular coupling via gap junctions; Tissue anisotropy (direction-dependent conduction); Membrane resistance and capacitance; Intracellular and extracellular conductivity.

Cardiac tissue behaves as a functional syncytium, meaning that electrical activity spreads efficiently through interconnected cells. However, structural heterogeneity and fibrosis can disrupt conduction pathways, leading to arrhythmias that are reflected in abnormal ECG patterns. From a biophysical standpoint, the heart during depolarization and repolarization can be modeled as a time-varying electrical dipole. An electrical dipole consists of two equal but opposite charges separated by a small distance. As depolarization spreads, regions of activated (negative extracellular charge) and resting (positive extracellular charge) myocardium create a dipole field. The direction and magnitude of this dipole change continuously during the cardiac cycle. This concept forms the basis of vector cardiography and explains how different ECG leads record varying waveforms depending on their orientation relative to the cardiac electrical axis. For example:

The **P wave** represents atrial depolarization.

The **QRS complex** reflects ventricular depolarization.

The **T wave** corresponds to ventricular repolarization.

The amplitude and polarity of these waves depend on the alignment between the cardiac electrical vector and the axis of the recording lead. The electrical signals generated by the heart must pass through various biological tissues lungs, blood, muscle, fat, and bone before reaching surface electrodes. These tissues act as a volume conductor.

The conventional 12-lead ECG system records electrical activity from multiple spatial orientations. Limb leads (I, II, III) and augmented leads (aVR, aVL, aVF) measure frontal plane activity, while precordial leads (V1-V6) assess horizontal plane activity. Each lead detects the projection of the heart's electrical vector onto its axis. Thus, ECG interpretation relies heavily on understanding spatial orientation and vector relationships. Signal acquisition involves: Placement of surface electrodes; Amplification of microvolt-level signals; Filtering to remove noise (e.g., muscle artifacts, powerline interference); Analog-to-digital conversion for computerized analysis. Electrocardiography (ECG) represents the macroscopic expression of highly coordinated electrophysiological and biophysical processes occurring within the heart. The ECG waveform is not merely a graphical tracing but the integrated result of ionic currents across cardiac cell membranes, synchronized propagation of action potentials through myocardial tissue, and the transmission of electrical fields through the thoracic volume conductor.

At the cellular level, ECG signals originate from precisely regulated ion fluxes primarily sodium (Na^+), potassium (K^+), and calcium (Ca^{2+}) which generate cardiac action potentials. The structural and functional properties of cardiac tissue, including intercellular coupling via gap junctions and anisotropic fiber orientation, ensure effective impulse propagation. At the organ level, the heart behaves as a dynamic electrical dipole whose magnitude and orientation change throughout the cardiac cycle. Finally, the torso functions as a conductive medium that modifies and attenuates these electrical signals before they are recorded by surface electrodes.

A comprehensive understanding of the biophysical foundations of ECG signals is essential for accurate clinical interpretation. Variations in ECG morphology such as changes in wave amplitude, interval duration, and segment displacement can be explained through alterations in ionic channel function, conduction velocity, myocardial mass, or tissue conductivity. This knowledge

enables clinicians to distinguish physiological adaptations from pathological abnormalities.

Moreover, advances in computational modeling, biomedical engineering, and signal processing continue to refine our understanding of ECG signal formation. Mathematical models of cardiac electrophysiology and high-resolution imaging techniques have bridged the gap between theoretical biophysics and clinical cardiology. These developments have facilitated the creation of wearable monitoring systems, artificial intelligence–based diagnostic tools, and personalized treatment strategies.

In conclusion, ECG signals reflect the intricate interplay between membrane biophysics, tissue conduction dynamics, and electromagnetic principles. A solid grasp of these bio-physical mechanisms not only enhances diagnostic accuracy but also supports innovation in cardiovascular medicine and biomedical technology.

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