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Computational modeling of electromechanical propagation in the helical ventricular anatomy of the heart

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ABSTRACT

The classical interpretation of myocardial activation assumes that the myocardium is homogeneous and that the electrical propagation is radial. However, anatomical studies have described a layered anatomical structure resulting from a continuous anatomical helical disposition of the myocardial fibers. To further investigate the sequence of electromechanical propagation based on the helical architecture of the heart, a simplified computational model was designed. This model was then used to test four activation patterns, which were generated by propagating the action potential along the myocardial band from different activation sites.

1. Introduction

It is well established that the sequence of electrical activation of the heart starts at the septum, propagates toward the apex and then to both ventricles, and eventually ends at the base of the heart [1,2]. This sequence accounts for the electrocardiographic signal of ventricular depolarization, which is known as the QRS wave. Based on this sequence, it has been hypothesized that the apicobasal spread of the electrical activation within the subendocardium initiates the myocardial contraction sequence [3]. Thus, the sequence of mechanical contraction is hypothesized to follow an apex-to-base propagation. However, other studies have suggested a base-to-apex sequence. For example, magnetic resonance studies have shown that the initial mechanical activation takes place at two sites at the base of the heart [4] and progresses to the apex. In contrast, isotopic studies using a Fourier analysis of the ventricular blood pool revealed that the systolic ventricular motion also follows a base-to-apex sequence [5].

The classical interpretation of myocardial activation assumes that the myocardium is homogeneous and that the electrical propagation of depolarization is radial [6]. However, it has been shown that the myocardial architecture is far from homogenous. In fact, anatomical studies have described a layered myocardial architecture. In addition, the controversy regarding the anatomical disposition of the myocardial fibers [7] has been resolved in favor of a continuous anatomical helical disposition of the myocardial fibers [3,8–10] based on the results of recent diffusion tensor magnetic resonance imaging studies [11]. Accordingly, the electromechanical wave propagation along the myocardial fibers should follow a path that follows the helical disposition of the myocardial fibers [12–14]. However, although the knowledge of cardiac mechanics has considerably improved over the last decade, the current cardiovascular textbooks do not incorporate the recent concepts on cardiac mechanics. Instead, the current textbooks emphasize the electrical aspects of the ventricular depolarization and somehow establish confusion between the initial electrification of the heart, which corresponds to the QRS wave on the surface electrocardiogram (ECG) tracing, and the electromechanical ventricular wave, which lasts almost throughout the systolic period [15,16].

Computer modeling could provide insights into these issues. However, to date, no studies have taken into account the helical myocardial anatomy. To further investigate the sequence of electromechanical propagation according to the helical configuration of the heart, a simplified computational model was designed to test how different sites of stimuli initiation can affect its propagation sequence. In spite of the currently described models, which are complexly designed to study the whole geometry of the ventricular cavity [17,18], the simple model used herein simulates the behavior of the myocardial tissue based mainly on the continuous helical fiber architecture [19,20].

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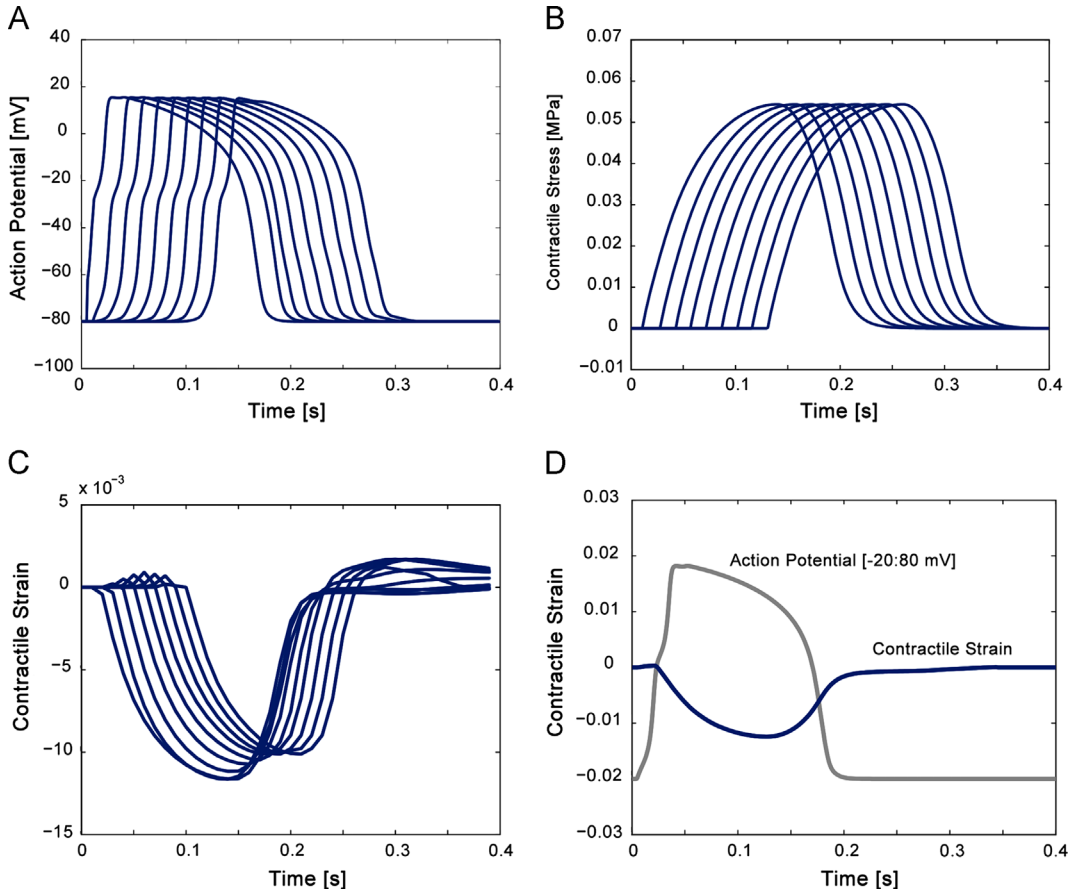

Fig. 1. Evolution of propagation of the (A) action potential, (B) contractile stress, and (C) contractile strain over time along the fibers. (D) Contractile strain of the fiber after activation of the action potential.

2. Methods

2.1. Modeling

To design a simplified computational method, several assumptions were taken into account. Due to the rod-shaped features of the myocardial fiber cells, the electromechanical propagation takes place along the longitudinal axis in what has been called anisotropic conduction, which corresponds to preferential conduction along one axis [21].

The morphological and functional features of the helical myocardium were used as a reference [8,10,22], and numerical computational methods were employed to recreate its behavior. These numerical methods approach the solutions of partial differential equations and are used in computational simulation in engineering, biomechanics, bioengineering, and especially computational mechanics of the heart [17,18].

The electromechanical behavior of the myocardium was considered a coupling of two parts, which are mathematically presented and largely discussed in a previous study [19]:

- the **active part** of the myocardium due to the fibers, which are modeled as a one-dimensional finite element projected in a three-dimensional space;
- the **passive part** of the myocardium due to the connective tissue, which controls the tissue deformation and maintains the cardiac fibers compact.

This electromechanical model for fiber contraction has been proven successfully for ventricular architectures other than the band approach [18,23].

2.2. Active part

The action potential activates fiber contraction, and both the action potential and the contraction propagate along a longitudinal path. The action potential **u(t)** uses the Aliev–Panfilov equations and the values proposed for the different parameters [24], which allows the calculation of the current **u(t)** at every time step. The fiber contraction was modeled using the rheological model developed by Hill–Maxwell based on Huxley's theory of the sliding filaments and cross bridge [25]. These equations enable the calculation of the fiber contraction in terms of the stress and strain.

The coupling of these two mathematical models describes the active part of the myocardium and generates a propagation model of the electromechanical behavior along the fibers (Fig. 1) [19].

2.3. Passive part

Biological tissues are currently modeled as hyperplastic materials [26]. However, for the purpose of simplicity, a linear elastic response was assumed for the passive part of the myocardium, which was modeled as a three-dimensional continuum element in the present study. In particular, this element was formulated using the Finite Element Method as an isoparametric hexahedrical element of eight nodes. The connective tissue of the myocardium was treated as a quasi-incompressible, elastic, and solid material governed by a lineal stress–strain relationship in the constitutive equation

$$\underline{\underline{\sigma}}_p = \frac{\partial W}{\partial \varepsilon} = \underline{\underline{D}} \cdot \varepsilon, \tag{1}$$