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Review

Modelling of the ventricular conduction system

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Abstract

The His–Purkinje conduction system initiates the normal excitation of the ventricles and is a major component of the specialized conduction system of the heart. Abnormalities and propagation blocks in the Purkinje system result in abnormal excitation of the heart. Experimental findings suggest that the Purkinje network plays an important role in ventricular tachycardia and fibrillation, which is the major cause of sudden cardiac death.

Nowadays an important area in the study of cardiac arrhythmias is anatomically accurate modelling. The majority of current anatomical models have not included a description of the Purkinje network. As a consequence, these models cannot be used to study the important role of the Purkinje system in arrhythmia initiation and maintenance.

In this article we provide an overview of previous work on modelling of the Purkinje system and report on the development of a His–Purkinje system for our human ventricular model. We use the model to simulate the normal activation pattern as well as abnormal activation patterns resulting from bundle branch block and bundle branch reentry.

Keywords: His-Purkinje system; Ventricular arrhythmias; Mathematical model; Simulation studies

Contents

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1. Introduction

The coordinated contraction of the heart is regulated by the specialized electrical conduction system of the heart (Eckert et al., 1988; Berne and Levy, 1993; Silverman et al., 2006). The main parts of this conduction system are the sino-atrial (SA) node, Bachmann's bundle, atrio-ventricular (AV) node, the His bundle, Tawara branches and Purkinje fibres. The SA node, located in the upper right atrium, is the primary pacemaker of the heart, automatically generating electrical pulses. These electrical pulses spread as waves of electrical excitation over the atria, with the Bachmann's bundle ensuring fast propagation toward the left atrium, leading to atrial contraction. The atria and ventricles are electrically isolated from each other except for the region of the AV node via which the excitation passes to ventricles. The speed of propagation in the AV node is slow, causing a delay in the passage of the electrical wave from the atria to the ventricles, ensuring that the ventricles contract after the atria. In the ventricles the excitation spreads at a high speed from the AV node through the His-bundle, left and right Tawara branches and Purkinje fibres. The Purkinje fibres are electrically connected to the ventricular muscle at certain insertion sites, from which the wave enters the ventricular wall, leading to ventricular excitation and contraction.

The above described pattern of excitation corresponds to the normal sequence of cardiac activation. Abnormalities in this pattern may lead to cardiac arrhythmias. They may occur in any of the elements of the conduction pathway. An example of an abnormal excitation pattern involving the His–Purkinje system is left or right bundle branch block, which leads to indirect, delayed activation of the right or left ventricle, respectively (Imanishi et al., 2006; Fantoni et al., 2005; Niu et al., 2006). Another example is bundle branch reentry, where partial block in one of the bundle branches leads to occasional unidirectional block and a macro reentry through the full His–Purkinje system, giving rise to ventricular tachycardia (Caceres et al., 1989; Mazur et al., 2005). In addition, the His–Purkinje system is considered to play an important role in the development and/or maintenance of some types of ventricular tachycardia and fibrillation in which repeated subendocardial focal activity, presumably of Purkinje origin, is observed to occur (Pogwizd and Corr, 1992; Chung et al., 1997; Pogwizd et al., 1998; Arnar et al., 1997, 2001; Arnar and Martins, 2002). The source of this Purkinje focal activity has been hypothesized to be either abnormal automaticity of the Purkinje fibres, or triggered activity, i.e. early afterdepolarizations (EADs) and delayed afterdepolarizations (DADs), or (micro)reentry (Arnar et al., 1997, 2001; Pogwizd et al., 1998; Xing and Martins, 2004). However, so far, it has not been clearly established which of these mechanisms actually occur, and under what kind of circumstances.

Since the His–Purkinje system plays such an important role in both normal ventricular excitation and life threatening ventricular arrhythmias, modelling of the His–Purkinje system is essential for a realistic ventricle model of the heart. In this article we will review existing models of the His–Purkinje system and present the development of a His–Purkinje model for our human ventricles model. The model is based both on anatomical knowledge of the His–Purkinje system (Tawara, 2000; Keith and Flack, 1906; Spach et al., 1963; James and Sherf, 1971; James et al., 1974; Massing and James, 1976) and the sites of earliest activation and normal activation sequence as reported by Durrer et al. (1970). Anatomy and conductance parameters of the His–Purkinje system were iteratively adjusted to correctly reproduce the Durrer et al. (1970) activation data. The electrophysiological behaviour of individual ventricular and Purkinje cells is modelled using our detailed human ventricular cell model (Ten Tusscher et al., 2004; Ten Tusscher and Panfilov, 2006), with a few modifications to account for differential Purkinje cell behaviour.

We show that our His–Purkinje–ventricles model produces an activation sequence that agrees well with the activation data from Durrer et al. (1970). We furthermore demonstrate realistic activation sequences for left and right bundle branch blocks. Finally, we use the developed model to study the occurrence of bundle branch