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# A Simplified 2D Heart Model of the Wolff-Parkinson-White Syndrome

**Clemens Zeile** * **Eberhard Scholz** ** **Sebastian Sager** *

* *Mathematics Department, University of Magdeburg, 39106 Germany (e-mail: clemens.zeile@ovgu.de; sager@ovgu.de ).*
** *Cardiology Department, University Hospital Heidelberg, 69120 Germany, (e-mail: eberhard.scholz@med.uni-heidelberg.de)*

**Abstract:** The enormous progress made in computational cardiac electrophysiology during the past decades has resulted in a diverse range of models and numerical methods. In general, researchers have elaborated highly complex and detailed simulators on the cell and tissue level. In contrast, there has been a lack of simplified whole-heart models that study specific heart arrhythmias. In this study, we approximate the electrophysiology of Wolff-Parkinson-White Syndrome with such a model. In order to reproduce the cardiac anomaly, we apply the so-called bidomain approach involving partial differential equations. Results show that the simulations are realistic, both in ECG generation and electric activation sequence. Our assessment of the model implementation takes into account parameter and geometry variation, which supports a realistic view of medical aspects. Our in silico analysis thus helps provide clear insights into the mechanisms of arrhythmias and associated ECG changes.



*Keywords:* Medical systems, heart, electrical signal propagation, cardiac arrhythmias, electrocardiogram, Wolff-Parkinson-White syndrome

## 1. INTRODUCTION

Cardiovascular disease (CVD) accounted for 17.3 million deaths in 2013, making it apparently the leading cause of death globally (Naghavi et al. (2014)).With a growing number of sufferer, it is estimated that 90% of CVD is preventable (McGill et al. (2008)). Cardiac arrhythmias constitute a major factor for sudden cardiac death (SCD). One of these arrhythmias, atrio-ventricular reciprocating tachycardia (AVRT), is associated with the Wolff-Parkinson-White (WPW) syndrome. Here, syndrome involves an accessory electrical conduction pathway (AP) between atrium and ventricle leading to prematurely stimulated, and thus contracted, ventricles.
Symptoms of WPW include palpitations and syncope. Only in rare cases (less than 0.6%, Naghavi et al. (2014)) is WPW fatal, for example, when accompanied by atrial fibrillation. A common means of detecting the disease is a slurred upstroke in the electrocardiogram, referred to as a "delta wave". For further details on WPW, we refer the reader to Page et al. (2015).

The open source framework Chaste (Gary R Mirams (2013)) is an excellent tool for multiscale and complex simulations, and one that can handle very detailed cardiac electrophysiology computations. It takes into account a variety of anatomical and physiological features such as fiber orientation, cell heterogeneities, anisotropy, as well as restitution properties. The simulators acCELLerate (Seemann et al. (2010)), CARP (Vigmond (2003)), and OpenCMISS (Christie (2009)) are similar, highly detailed packages.
However, these very realistic approaches require extensive computation power, and occasionally even the use of supercomputers. For instance, CARP needs 6.4 h in a 64-processor machine to simulate just 200 ms of cardiac activity (Mitchell (2010)).
To reduce computational effort, Balakrishnan et al. recently introduced a 2D whole-heart model in Matlab that focuses on the simulation of cardiac arrhythmias (Balakrishnan et al. (2015)). The model uses a phenomenological approach to compute electrical signal propagation, i.e., cells in discrete form linearly exchange current with eight neighboring cells. Sovilj et al. (2014) describes a 2D simplified (in terms of anatomy) approach that also covers the surrounding torso to compute realistic ECG generation, albeit applying the numerically demanding bidomain equations. We adopted this model for a comprehensive WPW study implementation, since it represents a good compromise between realistic simulation, computational load and flexibility. We refer to Trayanova (2011), Clayton et al. (2011), and Henriquez (2014) for reviews on whole-heart modeling.

Due to WPW's innocuousness compared to other arrhythmias (e.g., ventricular fibrillation), there are only few WPW simulation approaches, and these use mainly cellular automata (Zhu et al. (2007), Fleischmann et al. (1996)). To fill this research gap, we examine the WPW syndrome using an accurate bidomain model, simplifying it only enough to achieve reasonable computational times. We extend the Sovilj-Model by means of:

- geometric adoption with an accessory pathway (so called bundle of Kent),


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- variation in position, size and conduction velocity parameters of the bundle of Kent,
- computation of intracardiac electrograms,
- induction of extrasystole to simulate AVRT.

From simulations, we can detect realistic ECG behavior, specifically, the characteristic slurred upstroke of the QRS complex called delta wave. To our knowledge this is the first WPW evaluation with both extensive and simplified bidomain approaches including torso modeling. The simulations support a holistic view on the connections between the medical anomaly and its ECG morphology.

## 2. METHODS

In this section, we describe the main features of the Sovilj model and refer the reader to Sovilj et al. (2014) for further details. We also describe our model extensions.

### *2.1 Anatomical modeling*

The model comprises torso, lungs, heart and blood in the heart chamber. The heart itself is represented by individual areas for the sinoatrial node, atria, atrio-ventricular (AV) node, ventricles, bundle of His, bundle branches and Purkinje fibers, compare Figure 1. Our cardiac geometry has a range of 11.5 cm in length and 9cm in width. We modified the model with an AP between atria and ventricles, which is varied in size and position.

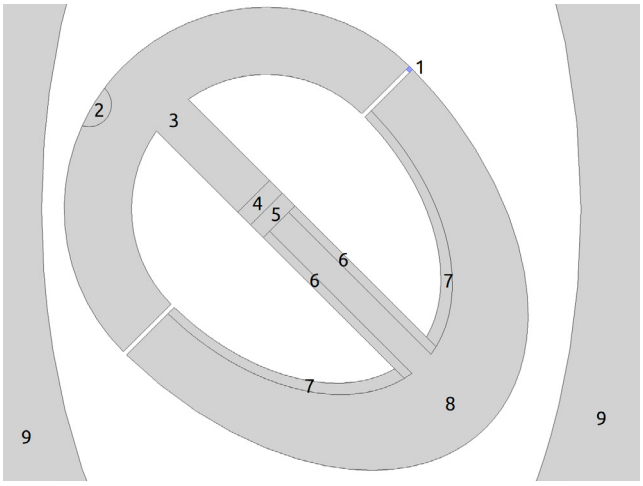


Fig. 1. Anatomical modeling: left lateral accessory pathway in blue (1), sinoatrial node (2), atria (3), AV node (4), bundle of His (5), bundle branches (6), Purkinje fibers (7), ventricles (8), and parts of lungs (9).

### *2.2 Governing equations*

The bidomain model is crucial for the electrical propagation modeling of tissue. It distinguishes intracellular from extracellular space regarding conductivites properties and anisotropy. In its parabolic-elliptic form transmembrane voltage $V$ and extracellular voltage $\phi_e$ are given by

$$\nabla \cdot ((\sigma_i + \sigma_e)\nabla\phi_e + \sigma_i \nabla V) = I_{\text{total}}^{(\text{vol})}, \tag{1}$$

$$\chi \left( C_m \frac{\partial V}{\partial t} + I_{\text{ion}}(u, V) - \nabla \cdot (\sigma_i \nabla (V + \phi_e)) \right) = I_{\text{i}}^{(\text{vol})}, \tag{2}$$

where $V = \phi_i - \phi_e$ and $\phi_i$ denotes the intracellular voltage (compare Pathmanathan et al. (2010)). The parameters in this system are the intracellular conductivity tensor $\sigma_i$, the extracellular conductivity tensor $\sigma_e$, the membrane capacitance per unit area $C_m$, and the the surface-area-to-volume ratio $\chi$. $u$ denotes a set of cell-level variables, which influence $I_{\text{ion}}(u, V)$, the ionic current per unit surface area. The source term $I_{\text{i}}^{(\text{vol})}$ reflects the intracellular stimulus per unit volume and $I_{\text{total}}^{(\text{vol})} = I_{\text{i}}^{(\text{vol})} + I_{\text{e}}^{(\text{vol})}$ is the sum of both intracellular and extracellular stimuli. Let $\mathbb{H}$ denote the region occupied by the cardiac tissue. The variables $I_{\text{total}}^{(\text{vol})}, I_{\text{i}}^{(\text{vol})}, I_{\text{e}}^{(\text{vol})}$, and $I_{\text{ion}}$ are all defined on $\mathbb{H}$ and depend on time.

We stress that the existence of a stimuli term in the model is an extension compared to the Sovijl model. It is necessary to model extrasystoles and therefore to simulate AVRT. These equations are applied only on the myocardium area. On the cell level, the modified FitzHughNagumo (FHN) equations are used

$$\frac{\partial u}{\partial t} = ke\left(\frac{V - B}{A} - du - b\right), \tag{3}$$

$$I_{\text{ion}} = kc_1(V - B)\left[a - \frac{V - B}{A}\right]\left[1 - \frac{V - B}{A}\right] + kc_2 u, \tag{4}$$

where $a, b, c_1, c_2, d, e, k, A, B$, are parameters. For the AP, we applied the physiological parameters from Baars et al. (2011) (see Table 1). We vary the conduction parameters

Table 1. AP parameters

| $a$ | $b$ | $c_1$ | $c_2$ | $d$ | $e$ | $k$ | $A$ | $B$ | $\sigma_i$ | $\sigma_e$ |
|---|---|---|---|---|---|---|---|---|---|---|
| 0.13 | 0 | 2.6 | 1 | 1 | 0.01 | 140 | -85 | $10^3$ | 2-80 | 2-80 |

to represent higher and lower velocities than in the normal ventricular conduction system, which is set to 8 mS/m. As indicated in Table 1, $\sigma_i$ and $\sigma_e$ ranges then between 2 and 80 mS/m. The extracellular voltage $\phi_e$ in the torso, lungs, and blood, i.e., the passive volume conductor domains, is given by

$$\nabla \cdot (-\sigma_p \nabla \phi_e) = 0, \tag{5}$$

where $\sigma_p$ denotes the domain specific electrical conductivity. For the boundary conditions of the torso and heart, we assume zero-flux for $\phi_i$ and that the outward flux equals the inward flux for $\phi_e$.

### *2.3 Intracardiac Electrograms*

In order to match simulations with clinical practice, we calculate the usual intracardiac electrograms resulting from catheters. For this, we locate electrodes in the model at the high right atrium (HRA), right ventricular apex (RVA), bundle of His (HIS), and coronary sinus (CS). The electrodes are bipolar, i.e., $HRA = HRA_1 - HRA_2$ , and so on. Hence, in each region, there is a pair of electrodes (see Fig. 3) that is grounded by the reference electrode at the right leg. Since the coronary sinus is not covered anatomically in the model, we placed the corresponding electrodes between the blood chambers of the left atrium and ventricle.