

Simulation and Visualization of the electrical Activity of the Heart with focal ventricular tachycardia in a 3D Model

Christian Quester, Biomedical Engineering Department of University of Applied Sciences Offenburg, Badstrasse 24, D-77652 Offenburg, Germany, e-mail: cquester@stud.hs-offenburg.de

Matthias Heinke, Peter Osypka Institute of Medical Engineering, Departement of Electrical Engineering, Medical Engineering & Computer Science, University of Applied Sciences Offenburg, Badstrasse 24, D-77652 Offenburg, Germany, e-mail: matthias.heinke@hs-offenburg.de

Johannes Hörth, Peter Osypka Institute of Medical Engineering, Departement of Electrical Engineering, Medical Engineering & Computer Science, University of Applied Sciences Offenburg, Badstrasse 24, D-77652 Offenburg, Germany, e-mail: Johannes.hoerth@hs-offenburg.de

Background

Patients with focal ventricular tachycardia are at risk of hemodynamic failure and if no treatment is provided the mortality rate can exceed 30%. Therefore, medical professionals must be adequately trained in the management of these conditions. To achieve the best treatment, the origin of the abnormality should be known, as well as the course of the disease. This study provides an opportunity to visualize various focal ventricular tachycardias using the Offenburg cardiac rhythm model.

Methods

Modeling and simulation of focal ventricular tachycardias in the Offenburg heart rhythm model was performed using CST (Computer Simulation Technology) software from Dessault Systèmes. A bundle of nerve tissue in different regions in the left and right ventricle was defined as the focus in the already existing heart rhythm model. This ultimately served as the origin of the focal excitation sites. For the simulations, the heart rhythm model was divided into a mesh consisting of 5354516 tetrahedra, which is required to calculate the electric field lines.

Results

The simulations in the Offenburg heart rhythm model were able to successfully represent the progression of focal ventricular tachycardia in the heart using measured electrical field lines. The simulation results were realized as an animated sequence of images running in real time at a frame rate of 20 frames per second. By changing the frame rate, these simulations can additionally be produced at different speeds.

Conclusion

The Offenburg cardiac rhythm model allows visualization of focal ventricular arrhythmias using computer simulations. By selecting the frame rate, the speed of the simulation results can be adjusted accordingly to visualize the electric field lines of focal ventricular tachycardias in more detail. The static and dynamic simulation results could be used in the future for teaching and research, including the training of medical professionals.

3D Model and Simulation of Atrial Reentry Mechanism and Supraventricular Tachycardia

Jana Lindner, Department of Electrical Engineering, Medical Engineering & Computer Science, University of Applied Sciences Offenburg, Badstraße 24, D-77652 Offenburg, Germany, e-mail: jlindner@stud.hs-offenburg.de

Matthias Heinke, Peter Osypka Institute of Medical Engineering, Department of Electrical Engineering, Medical Engineering & Computer Science, University of Applied Sciences Offenburg, Badstraße 24, D-77652 Offenburg, Germany, e-mail: matthias.heinke@hs-offenburg.de

Johannes Hörth, Peter Osypka Institute of Medical Engineering, Department of Electrical Engineering, Medical Engineering & Computer Science, University of Applied Sciences Offenburg, Badstraße 24, D-77652 Offenburg, Germany, e-mail: johannes.hoerth@hs-offenburg.de

Background: Disturbances of the cardiac conduction system causing reentry mechanisms above the atrioventricular (AV) node are induced by at least one accessory pathway with different conducting properties and refractory periods. This work aims to further develop the already existing and continuously expanding Offenburg heart rhythm model to visualise the most common supraventricular reentry tachycardias to provide a better understanding of the cause of the respective reentry mechanism.

Methods: Modelling and electrical field simulations of the AV nodal reentrant tachycardia (AVNRT), AV reentrant tachycardia (AVRT), atrial flutter (AFL) and catheter ablation were performed using the software CST from Dassault Systèmes. The simulation of the slow-fast AVNRT is to be achieved by the modelling of a dual AV node. The modelling of an accessory pathway between the left atrium and left ventricle for the simulation of an AVRT was also implemented. The common type I counterclockwise AFL with a 2:1 heart block is achieved by integrating a macro-reentry circuit in the right atrium.

Results: The static and dynamic simulations of the impulse propagation in the heart in the individual disease patterns clearly visualise the supraventricular reentry mechanisms. The most informative results were obtained in the simulation of AVRT. The clarity of the provided results is especially remarkable in the relatively small volume of the AV-node in combination with the rapidly rotating impulse. The rotating impulse in the macro-reentry circuit of the simulated AFL provided more abstract results. The simulation of the rotating impulse in the right atrium delivered less accurate results; an exact localization of the impulse at any moment during a "rotation" was not possible. Nevertheless, the motion of a rotation is visible.

Conclusions: Electrical field simulations of AVNRT, AVRT and AFL with different supraventricular reentry mechanisms are possible with the Offenburg heart rhythm model. The static and dynamic electrical field simulation may be used to optimize the catheter ablation of supraventricular tachycardia.

Spatial Sensitivity of ECG Electrode Placement

Andra Oltmann, Fraunhofer IMTE, Lübeck, Germany, andra.oltmann@imte.fraunhofer.de
Roman Kusche, Fraunhofer IMTE, Lübeck, Germany, roman.kusche@imte.fraunhofer.de
Philipp Rostalski, Fraunhofer IMTE, Lübeck, Germany, philipp.rostalski@imte.fraunhofer.de

Introduction

ECG is a well-known technique used to diagnose cardiac diseases. To acquire the spatial signal characteristics from the thorax, multiple electrodes are commonly used. Displacements of these electrodes caused by intra physician variability affect the signal morphologies and can lead to incorrect diagnoses. Furthermore, individual patient anatomy and electrode shift relatively to the heart induced by body deformation can be challenging. A quantitative analysis of these effects over the whole thorax is still not comprehensively investigated in literature. Therefore, we propose the usage of a numerical computer simulation to obtain placement sensitivity maps for different ECG waves.

Methods

In order to create a realistic digital representation of the human thorax including the heart and lungs a three-dimensional model with a simplified geometry is developed. The electrical excitation of the heart is modelled on a cellular level via the bidomain approach. To numerically solve the differential equations, describing the signal propagation within the body, we use the finite element method in COMSOL Multiphysics. In order to determine and graphically display the regional sensitivities of the electrode placements, the spatial gradients of the resulting body surface potentials are calculated.

Results

The obtained gradient plots from various points in time show that the spatial sensitivity is different for each ECG wave. In general, the impact of electrode displacement is increased as an electrode is more closely located to the heart. However, in some specific regions typically associated with differential ECG leads the placement sensitivity distribution deviates from this simple circular pattern.

Conclusion

The results provide useful information for physicians as well as medical device manufacturers to enable more reliable ECG measurements and easier electrode placement. By additional consideration of patient-specific body characteristics in the future, this model can be enhanced to provide individual placement recommendations.

Fluid dynamics in the human heart: Altered vortex formation and wash-out in mitral regurgitation simulations

Jochen Brenneisen (Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT)), Karlsruhe, Germany, publications@ibt.kit.edu

Carlo Wentzel (Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT)), Karlsruhe, Germany

Farokh Karwan (Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT)), Karlsruhe, Germany

Olaf Dössel (Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT)), Karlsruhe, Germany

Axel Loewe (Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT)), Karlsruhe, Germany

Introduction

Computer models of the human heart can be used to investigate disease phenomena, to deduce measures and to support physicians in medical treatment. Visualizing flow patterns is a powerful simulation-based tool to reveal disease-induced flow alterations. Thus, in this work we evaluated altered flow patterns in the left ventricle (LV) caused by different severities of a regurgitant mitral valve.

Methods

A patient-specific finite element heart geometry was created from MRI scans. The boundary conditions for fluid dynamic simulations, namely the movement of endocardial surfaces and the in- and outlet pressure over time were obtained through a whole-heart mechanical simulation including a closed-loop circulatory system.

All heart valves were modelled by porous zones and controlled depending on flow. By including a hole in the mitral valve, mitral regurgitation with different severities was modelled by different effective regurgitant orifice areas according to European Society of Cardiology guidelines.

By solving the Navier-Stokes equations, the pressure distribution and the velocity field across the left part of the heart geometry was computed. Passive scalar transport was used to analyze the fraction of the initial blood volume that remains in the LV.

Results

Altered flow patterns (particular in end-diastole) and wash-out fractions were observed in the diseased geometry. For moderate mitral regurgitation, the ventricular wash-out was 6% lower than in the healthy control case.

Particle tracking confirmed the slower wash-out: The ‘direct flow’, which measures the flow component that enters and leaves the LV during one cycle, was reduced by 10%.

Conclusion

We showed how blood flow patterns and ventricular wash-out fraction in the human heart are affected by mitral valve regurgitation. This work builds a basis to characterize vortex structures in mitral valve disease in future studies.

Adaptation of the Calcium-dependent Tension Development in Ventricular Cardiomyocytes

Stephanie Appel, Institute of Biomedical Engineering, Fritz-Haber-Weg 1, Karlsruhe, Germany, publications@ibt.kit.edu

Tobias Gerach, Institute of Biomedical Engineering, Fritz-Haber-Weg 1, Karlsruhe, Germany, tobias.gerach@kit.edu

Olaf Dössel, Institute of Biomedical Engineering, Fritz-Haber-Weg 1, Karlsruhe, Germany, olaf.doessel@kit.edu

Axel Loewe, Institute of Biomedical Engineering, Fritz-Haber-Weg 1, Karlsruhe, Germany, axel.loewe@kit.edu

Introduction

Today a variety of models describe the physiological behavior of the heart on a cellular level. The intracellular calcium concentration plays an important role, since it is the main driver for the active contraction of the heart. Due to different implementations of the calcium dynamics, simulating cardiac electromechanics can lead to severely different behaviors of the active tension when coupling the same tension model with different electrophysiological models.

Methods

To handle these variations, we present an optimization tool that adapts the parameters of the most recent, human based tension model. The goal is to generate a physiologically valid tension development when coupled to an electrophysiological cellular model independent of the specifics of that model's calcium transient. In this work, we consider a ventricular cell model. In order to identify the calcium-sensitive parameters, a sensitivity analysis of the tension model was carried out. In a further step, the cell model was adapted to reproduce the sarcomere length-dependent behavior of troponin C.

Results

With a maximum relative deviation of 20.3% per defined characteristic of the tension development, satisfactory results could be obtained for isometric twitch tension. Considering the length-dependent troponin handling, physiological behavior could be reproduced.

Conclusion

In conclusion, we propose an algorithm to adapt the tension development model to any calcium transient input to achieve a physiologically valid active contraction on a cellular level. As a proof of concept, the algorithm is successfully applied to one of the most recent human ventricular cell models. This is an important step towards fully coupled electromechanical heart models, which are a valuable tool in personalized health care.