QRS Width and QT Time Alteration Due to Geometry Change in Modelled Human Cardiac Magnetograms

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Abstract

The influence of changing mesh geometries due to muscle contraction on computated biomagnetic fields of the human heart is investigated. To generate a detailed computer model of the ventricle and compare it to magnetocardiographic data, MCG and MRI were taken consecutively from the same proband. Two computer models were built from the MRI data at diastole and systole in order to assess the maximum deviation induced by geometry on the MCG. We could reproduce the structure of the measured MCG. The mesh geometry had a significant effect on the simulated magnetogram. QRS width differed by 15 ms and QT_{max} times differed by 20 ms between the two computer-generated MCG. Consequently, models for biomagnetic heart activity should take detailed geometry change during the heart cycle into account.

1. Introduction

Due to the technological advances of high-sensitive superconducting quantum interference devices (SQUID) in recent times, magnetocardiography (MCG) has become a promising application in diagnostic cardiology. MCG has several advantages compared to electrocardiography (ECG), it is a contact-free method which allows rapid and easy measurement.

With the ever-growing power of modern computers, the possibility of numerical models which describe whole body organs has come into reach. These models usually describe only specific aspects of physiological systems. Our focus is the development of a realistic model of the human heart with emphasis on simulating magneto-cardiograms. Such a model allows to perform physiological experiments 'in silico' and can give valuable information on various topics of basic medical research, for example to estimate the diagnostic value of MCG on different pathological conditions.

Magnetic resonance imaging (MRI) has become one of the most important tools for imvestigating the interior of the human body. It is suggestive to use MRI to gain geometrical information on the heart muscle and use it in a computer model. It is straightforward to use MRI to provide the basic information needed to generate a patient-specific computer model.

As the heart contracts and changes its geometry during the cardiac cycle, one expects that this geometry alteration has a sizeable effect on the resulting simulated MCG. Furthermore, as sophisticated computer models of the human heart still require enormous computing power, research often is restricted to two-dimensional geometrical setups.

In this work, we deal with two questions: First, whether a two-dimensional model gained from a MRI slice can reproduce the MCG, and second, whether the geometry change during the heart cycle has a significant effect on the simulated MCG.

2. Methods

2.1. Data acquisition

Subject was a voluntary 35-years old, healthy male proband. To avoid possible disturbances in the MCG due to any tissue magnetizations, the MCG was taken before the MRI. The MCG was measured using a 83-SQUID system at a sampling rate of 1 kHz. MRI was taken directly afterwards on a 1.5 T machine with a delay of around 45 minutes for preparations. A total of 25 short axis views were taken from the heart cycle at different R-trigger times, with a time step of 32 ms, therefore covering a total of 800 ms after the R-wave. The raw MRI data has a pixel size of 1480µm and consists of 256x256 pixels.

2.2. Building the computer model

From the set of MRI images, short axis views in the middle of the ventricles were chosen as the basis for the

model. To investigate the effect of geometry change, two images were chosen at R-trigger time t=32 ms (during diastole) and t=320 ms (during systole), which differed most in geometry. The left ventricle was identified and segmented in both images (see Fig. 1). A regular finite element grid consisting of hexahedral elements at a spatial resolution of 250 µm was built out of this data (see Fig. 2).

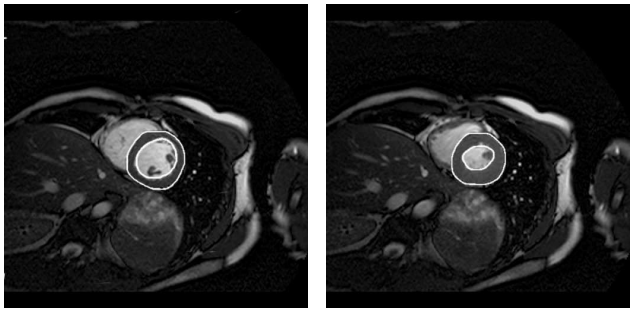

Fig. 1: Segmented MRI short axis views during diastole (left) and systole (right). The left ventricle contours are marked. Image sizes are 378x378 mm.

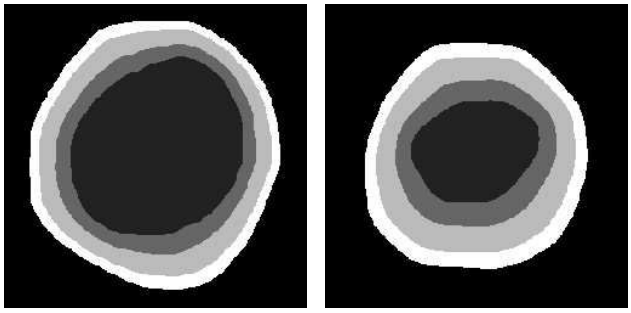

Fig. 2: The generated geometries for the model of the left ventricle incl. epicardial (white), M-cell (light gray) and endocardial (dark gray) regions. Image sizes are 88x88 mm.

The electrophysiology was described by the Ten Tusscher human ventricular model [1]. This model differentiates between three different myocardial regions. The grid was therefore split into non-myocardial tissue, blood cavity, and three myocardial regions (epicardial, endocardial and M-cells). We choose ratios of 26% for epicardial, 40% for M-cells and 34% for endocardial over the ventricular wall.

The anisotropy was assumed to rotate linearly from an angle of –45° at the epicardial wall to +75° at the endocardial wall [2]. The direction of the fiber orientation vector was taken as orthogonal to the line through the nearest points at the epi- and endocardial wall. (See Fig. 3). The intra- and extracellular conductivity values were chosen to be s_{IL}=0.174 S/m and s_{EL}=0.625 S/m along the fibers, s_{IT}=0.019 S/m and s_{ET}=0.236 S/m tangential to the fibers [3]. Isotropic conductivity values were set to s_B=0.2 S/m for passive non-myocardial tissue and s_C=1.0 S/m for the blood cavity.

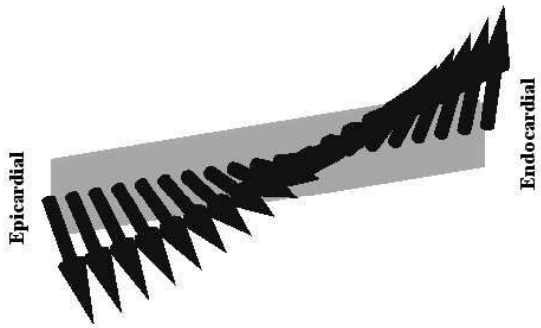

Fig. 3: Fiber orientations ranging from –45° at the epicardial side to +75° at the endocardial side.

2.3. Simulating ventricular activity / MCG

The spread of transmembrane and extracellular potentials was calculated by a modified bidomain approach [6]. We used a semi-implicit operator splitting method to separate the system of equations into a system of ODEs, a parabolic PDE and an elliptic PDE which can then be solved sequentially [4]. Instead of solving the full bidomain, we applied the monodomain conductivity tensor to the parabolic equation and solved the elliptic part with a lower frequency. The parabolic part was solved with a timestep of 10 µs while the elliptic part was solved in a 1000 µs interval. We applied a semi-implicit Crank-Nicholson scheme to the parabolic part and used a conjugate gradient method with a multigrid preconditioner to solve the linear equation associated with the elliptic part [5]. One run did take around 2 hours on a 16-processor parallel cluster.

To reconstruct the initial wavefront at the start of depolarization, one region of the size of 1mm x 1mm in the septum towards the sternum was chosen for the initial excitation by the purkinje system. Corresponding areas were identified in both diastolic and systolic geometry. A transmembrane current of 20 µA/µm² was applied to this region as initial condition for a duration of 2 ms.

The MCG was calculated by forward method. We assume the sources of the magnetic field to originate in the superposed currents through both intra- and extracellular domains which can be obtained by calculating the gradients of the potentials. The resulting magnetic field vectors can be computed by using the Biot-Savart integral over the currents in the whole volume.

3. Results

The obtained transmembrane potentials for both geometry bases can be seen in Fig. 4.

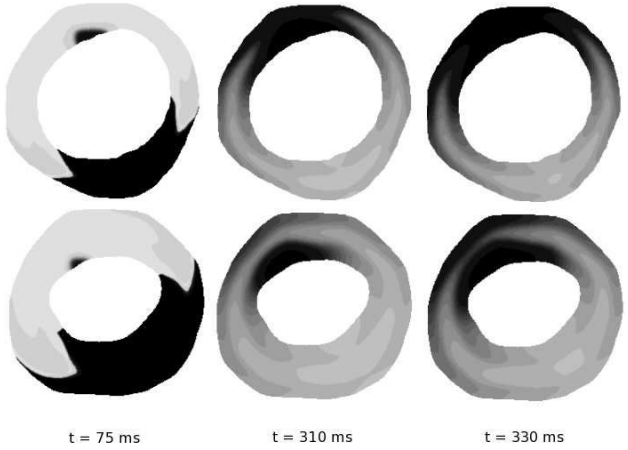

Fig. 4: Transmembrane potentials for diastolic (above) geometry base and systolic (below) geometry base during depolarization and at two timepoints during repolarization.

The proband's measured MCG is shown in Fig. 5. We found a QRS width of 95 ms and a QT_{max} time of 295 ms.

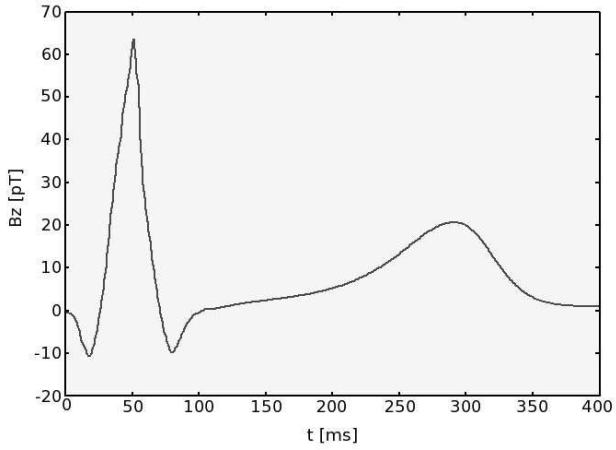

Fig. 5: Averaged measured MCG in central channel.

The simulated MCG is shown in Fig. 6 for both diastolic and systolic geometrical base. The B-field was calculated at a distance of 65 mm to the active tissue. The diastolic case shows a QRS width of 89 ms and a QT_{max} time of 308 ms; the systolic case has a QRS width of 104 ms and a QT_{max} time of 328 ms.

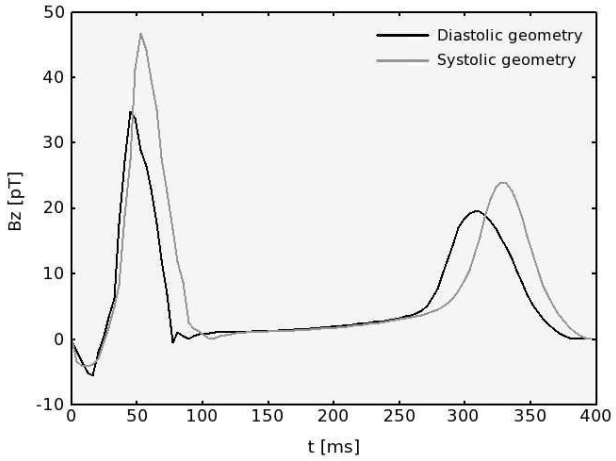

Fig. 6: Modelled MCG for both geometrical bases at 65 mm distance to the outer surface of the active region.

4. Discussion and conclusions

A two-dimensional computer model of the human left ventricle could reproduce the basic features of the measured magnetocardiogram. Modeling results for the two setups which differed only in the geometrical base show differences in the MCG. These results suggest that detailed models of the biomagnetic field of the human heart should include the effect of mechanical contraction; this is in particular important for reconstruction of the T-wave, as cardiac contraction occurs during repolarization.

In [7], a two-dimensional model of the left-ventricle was developed in order to study the mechano-electrical coupling effects on ECG waveforms. It was verified that during contraction the coupling significantly influenced the T-wave waveforms of the numerical simulations. Currently, our static model formulation does not account for cardiac movement and mechano-electrical coupling effects. In the near future, we hope to address these relevant issues, and contribute to the development of more realistic cardiac models.

Two-dimensional models of the left ventricle are simplified approaches for modeling the human MCG. Future research will be based on a 3D reconstruction of the left and right ventricle. With an appropriately chosen MRI acquisiton protocol, whole heart slices should be obtained for at least two trigger times to investigate the effect on the modelled MCG for the complete 3-dimensional geometry. Furthermore, as back-currents through surrounding tissue are thought to have a noticeable effect on the resulting MCG [8], a more complete patient-specific computer model of the human heart should also include information on the torso geometry around the heart.

Acknowledgements

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References

[1] Ten Tusscher K, Noble D, Noble PJ, Panfilov AV. A Model for human ventricular tissue. American Journal of Physiology 2004, 286: H1573-H1589

[2] Streeter D. Gross Morphology and fibre geometry of the heart. In: R. Berne, N. Sperelakis, editors. Handbook of Physiology sect. 2: The Cardiovascular System, American Physiology Society. Baltimore (MD): Williams & Wilkins 1979: 61-112

[3] Clerc L. Directional differences of impulse spread in trabecular muscle from mammalian heart. J. Physiol. 1976, 255: 335-346

[4] Strang G. On the construction and comparison of difference scheme. SIAM J. Numerical Anal. 1968, 5: 506-517

[5] Weber dos Santos R, Plank G, Bauer S, Vigmond EJ. Parallel Multigrid Preconditioner for the Cardiac Bidomain Model. IEEE Trans. on Biomedical Engineering 2004, 51: 1960-1968

[6] Vigmond EJ, Aguel F, Trayanova N. Computational techniques for solving the bidomain equations in three dimensions. IEEE Trans. Biomed. Eng. 2002, 49: 1260-1269

[7] Smith NP, Buist ML, Pullan AJ. Altered T wave dynamics in a contracting cardiac model. J. Cardiovasc. Electrophysiol. 2003, 14: S203-S209

[8] Weber dos Santos R, Dickstein F. On the influence of a volume conductor on the orientation of currents in a thin cardiac tissue. In: Magnin IE, Montagnat J, Clarysse P, Nenonen J, Katila T, editors. Lecture Notes in Computer Science Vol. 2674. Berlin Germany: Springer-Verlag, 2003: 111-121

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