introduction To compute extracellular potentials from transmembrane potentials an elliptic boundary-value problem must be solved [2, 7]. To avoid artefacts, this must be done at a spatial resolution of 0.2 mm or better [1]. For macroscopic heart models this leads to very large linear systems.

purpose We attempted to reduce such artefacts by using a special downsampling method for the source data.

conclusion This method is sufficiently accurate for visualization of electrograms in a human-heart model, even in inhomogeneous tissue.

Methods

Simulations were performed using previously-described software [6]. Uniform finite-difference meshes were used. Simulations were performed on 32–128 processors of an SGI Altix 4700 supercomputer.

Propagating action potentials were simulated with a monodomain reaction-diffusion equation at 0.2-mm resolution in a model of the human ventricles. Extracellular potentials (electrograms) \( \phi_e \) were computed by solving the bidomain equation

\[
\nabla \cdot [G_e(x) + G_i(x)] \nabla \phi_e(x,t) = I(x,t)
\]

where \( x \) is position, \( t \) is time, \( G_e, G_i \) are the intracellular and extracellular conductivity tensor fields, and \( I \) is the transmembrane current given by

\[
I(x,t) = -\nabla \cdot G_i(x) \nabla V_m(x,t),
\]

where \( V_m \) is the membrane potential. We evaluated \( I(x,t) \) at the full 0.2-mm resolution of the reaction-diffusion model.

To solve equation (1) at 1-mm resolution, \( I(x,t) \) was taken from the high-resolution propagation model and summed over 1-mm³ volumes. Each fine-mesh (F) node contributed to 1–8 coarse-mesh (C) nodes. The weight of each contribution was

\[
\omega = \begin{cases} 
0, & \text{if } d_x \geq N \land d_y \geq N \land d_z \geq N \\
(N - d_x)(N - d_y)(N - d_z)/N^6, & \text{otherwise}
\end{cases}
\]

where \( N \) is the ratio of fine to coarse grid resolution (\( N = 5 \) here) and \( d_x, d_y, d_z \) is the number of fine-mesh edges between the C node and the F node along the x, y, and z axis, respectively.

\( \phi_e \) was computed at 1-mm resolution both for the isolated heart (1 million nodes) and for the in-situ heart (42 million nodes).

To test the validity of the low-resolution results, electrograms were also computed at the full 0.2-mm resolution in the isolated heart (113 million nodes).

Anatomic model

An anatomic model of a human heart and torso was created from MRI data as described earlier [5]. This model described torso surface, myocardium, intracavitary blood masses, and lungs.

For a severe test of the proposed method, we created a situation where inhomogeneous tissue caused fractionated electrograms. Fibrofatty replacement and Na-channel block were simulated as in previous work [3, 4]. Fibrosis was simulated by introducing barriers with a thickness of 0.2 mm in the outer 50% of the right ventricular wall. In these barriers, no intercellular coupling was present. In bidomain terms, \( G_e = 0 \) but \( G_i \) had the normal value for myocardium. In the barriers, gaps of 0.2 × 0.2 mm were made. The conductivity of the fast Na current was set to 30% of its normal value, in the entire heart.

Display of results

Activation times, determined automatically from the activation process of the fast Na current, are shown in a transverse section of the heart model. The plane of section and needle positions (A–E) are shown below.

Unipolar electrograms taken from 6 positions along each of 5 virtual needle electrodes (labeled A–E) are shown.

Simulated activation pattern

The figure above shows simulated activation times in a single plane of the three-dimensional heart model, with the positions of the virtual needle electrodes.

Simulated electrograms

Electrograms computed at 0.2-mm resolution:

Electrograms computed at 1-mm resolution:

Close-up (black, 1-mm resolution isolated heart; red, 0.2-mm resolution isolated heart; blue, 1-mm resolution in-situ heart):

References


Acknowledgements

Computations were performed on computers of the Résseau Québécois de calcul de haute performance (RQCHP). The anatomic model used in this study was prepared by Dr André Linnenbank and Dr Pieter Postema at the Academic Medical Center of the University of Amsterdam. The representation of fibrotic myocardium was developed in collaboration with Dr Mark Hoogendijk, from the same institution.