Electro-mechanics of the human heart

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IHPCSS 2017

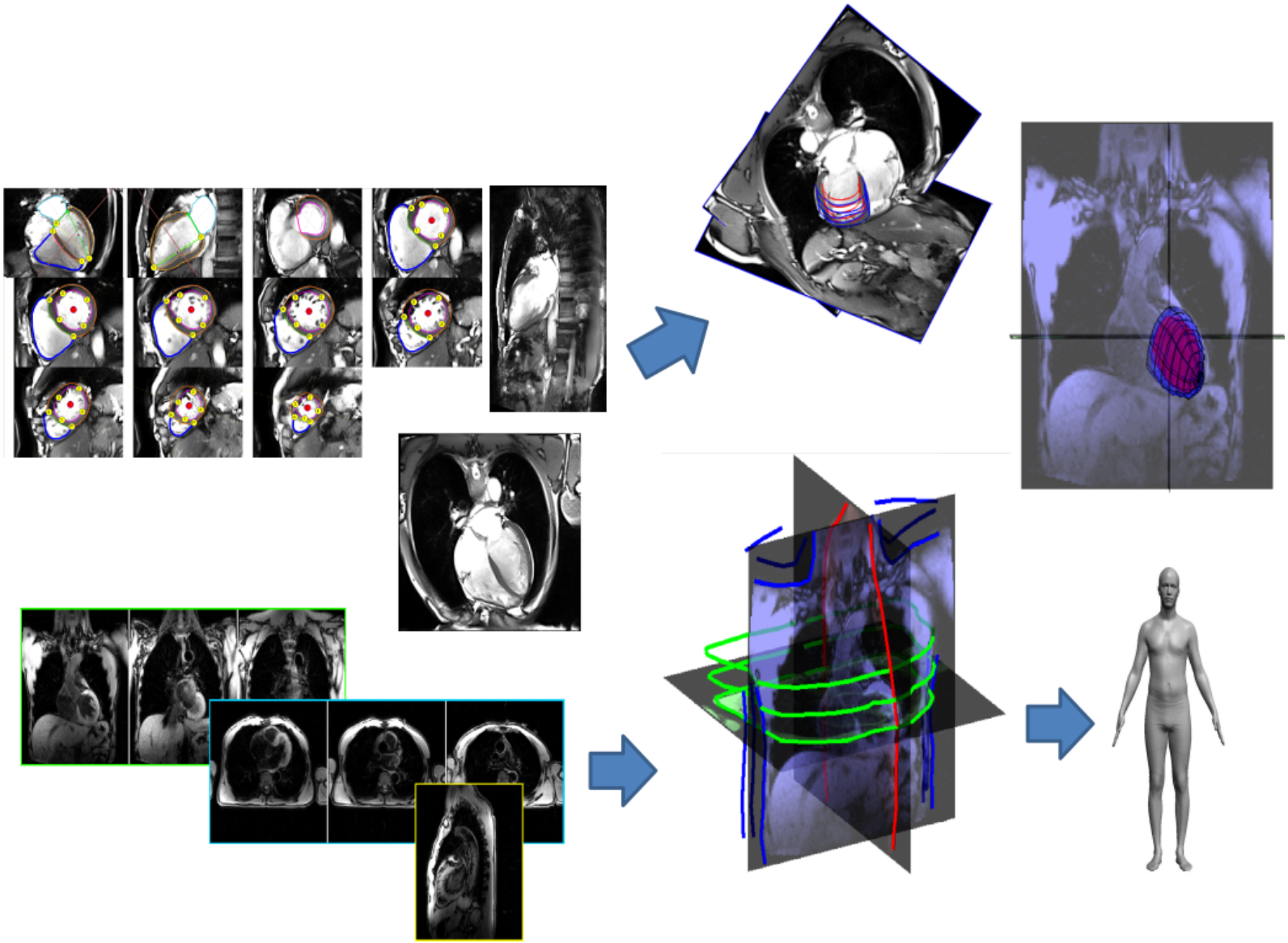
Abstract

Cardiovascular diseases are amongst the deadliest diseases worldwide according to the World Health Organization (2015), with ischaemia and strokes being the two first causes. This project aims at obtaining accurate predictive tools for cardiovascular diseases, in particular to assess arrhythmic risk. The associated simulations consist of complex electro-mechanical biventricular finite elements (FE) systems of the human heart, in which different drugs and ischaemic conditions are tested.

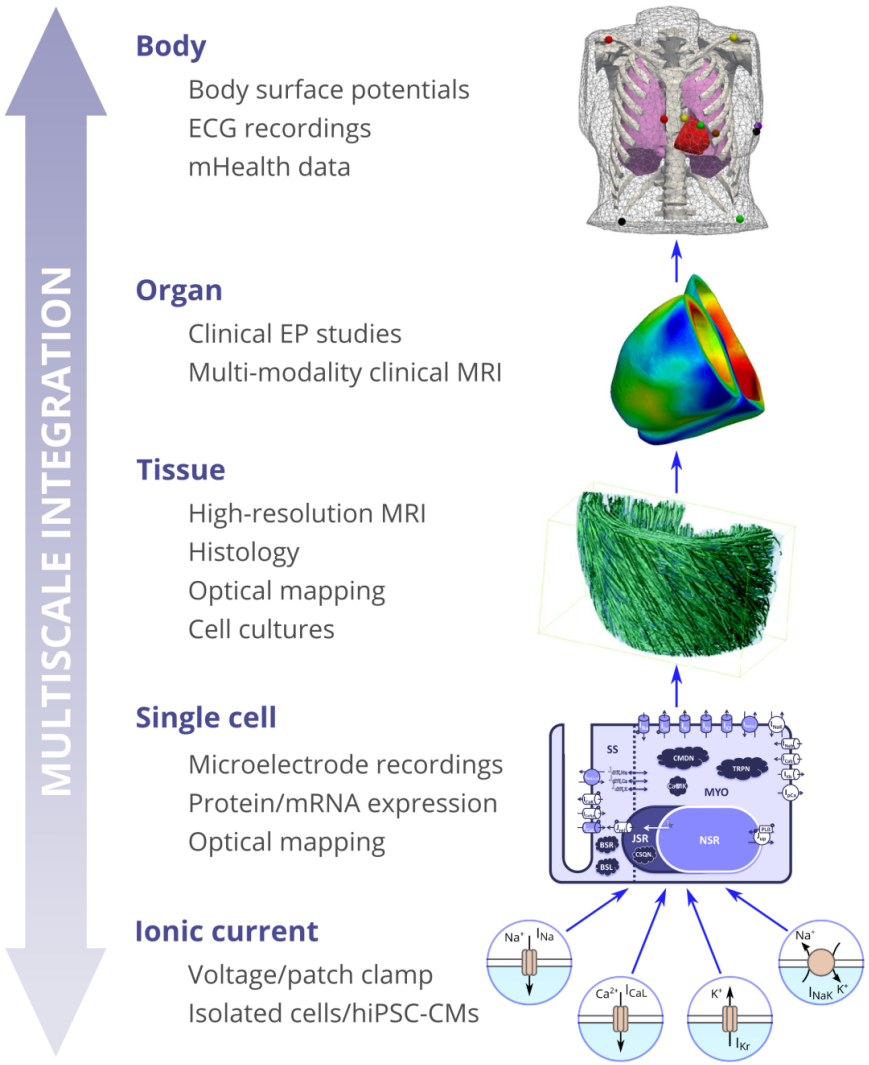
Heart models comprise several coupled physical phenomena, which means that electrical propagation, fluid flow and solid deformation interact with each other. The heart geometry is extracted from medical images provided by clinical collaborators (magnetic resonance images, MRI), through a semi-automated process. Due to certain requirements of the electrical propagation, these geometries are discretised into FE systems of tens of millions of nodes. This leads to the need of using HPC platforms to obtain a solution.

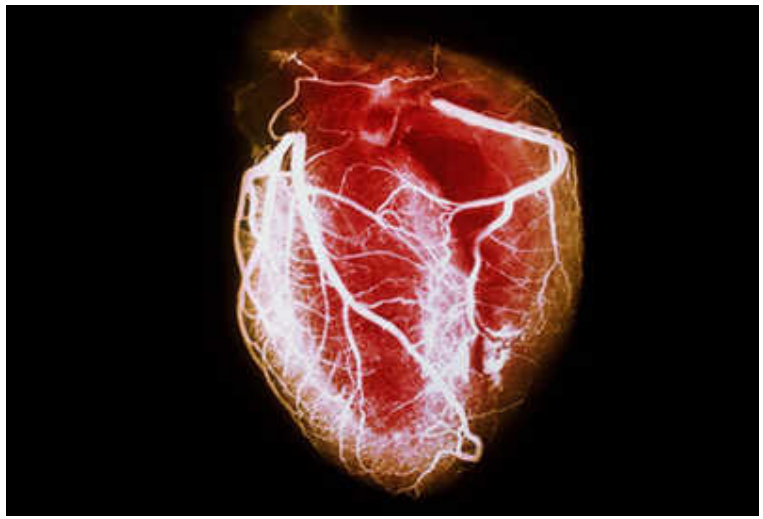
Different sets of simulations are used in this study. A first set is used to validate subsequent simulations with the respective 4-dimensional MRI data. Further sets are used in which different ischaemic locations and/or severities are tested, with and without the effect of drugs.

Image processing



Multiscale approach





In this simulation we can see the blood ejection from the ventricles: an **electrical wave** triggers the **contraction of cardiac muscle**, which, at the same time triggers the **blood pumping** out of the ventricles.

Computational approach

Electrical propagation

$$\nabla \cdot (\mathbf{D}_0 \nabla V) = \chi(C\frac{\mathrm{d}V}{\mathrm{d}t} + I_{ion} - I_{app})$$

Cellular model of action potential

$$\frac{\mathrm{d}\mathbf{w}}{\mathrm{d}t} = m_{\mathbf{w}}(V, \mathbf{w}, \mathbf{c});\ \frac{\mathrm{d}\mathbf{c}}{\mathrm{d}t} = m_{\mathbf{c}}(V, \mathbf{w}, \mathbf{c})$$

Equations of motion

$$\nabla \cdot \mathbf{P} + \rho_0 \mathbf{b} = \rho_0 \ddot{\mathbf{u}}$$

Active contraction model

$$\frac{\mathrm{d}\mathbf{s}}{\mathrm{d}t} = m_{\mathbf{s}}(\mathbf{c}, \mathbf{s}; \lambda, \dot{\lambda})$$

Lumped circulation model

$$\left(1 + \frac{R_1}{R_2}\right) I(t) + CR_1 \frac{\mathrm{d}I(t)}{\mathrm{d}t} - C\frac{\mathrm{d}P_{ar}(t)}{\mathrm{d}t} - \frac{P_{ar}(t)}{R_2} = 0$$

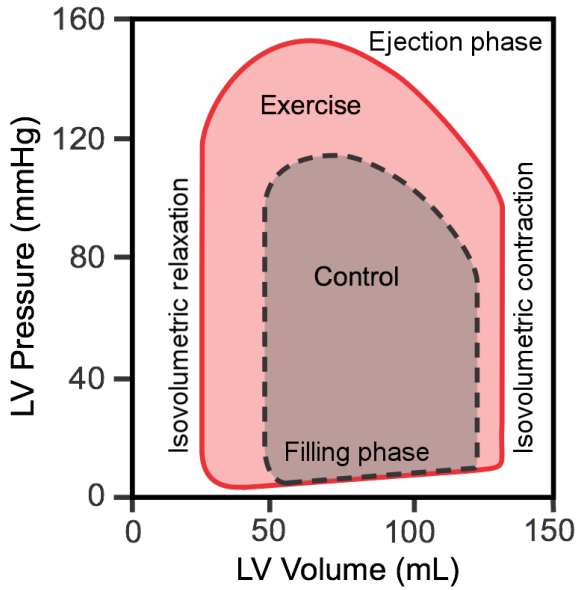


Figure: Pressure–volume diagram of the heart

Pressure in the ventricles

$$P_{ven}^{n+1} = \begin{cases} -K\left(\frac{V(t)}{V_d} - 1\right) & IC \\ P_{ar}^{n+1} & EP \\ -K\left(\frac{V(t)}{V_s} - 1\right) & IR \\ P_{ven}^{n} - \gamma \Delta V^n & FP \end{cases}$$

Future plans and acknowledgements

Future work

- Perform studies on drug cardiotoxicity for the electro-mechanical behaviour of the heart (electro-mechanical window)
- Study multiscale (in time) diseases, such as pulmonary hypertension
- Perform a massive validation effort on these electro-mechanical models to allow for their use in *in-silico* clinical trials

Acknowledgements

- **Computational Cardiovascular Science, University of Oxford**, Alfonso Bueno-Orovio, Ana Minchole, Ernesto Zacur, Blanca Rodriguez
- **Barcelona Supercomputing Center**, Jazmin Aguado-Sierra, Alfonso Santiago, Ruth Aris, Marina Lopez, Mariano Vazquez
- **ARCHER Leadership project**
- **European Centre of Excellence** in Computational Biomedicine