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NUMERICAL SIMULATION OF CARDIAC MUSCLES IN A RAT BIVENTRICULAR MODEL

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Developments in computational modelling of the human heart have been remarkable in the recent years and play a crucial role in research on heart pathology. In comparison to human hearts, rat hearts have a similar structure and cause less ethical problems. Therefore, rat hearts are often preferably utilized for investigations with a larger number of specimen associated with higher levels of confidence. The cardiac electromechanics in a rat heart takes place several times faster with the same magnitudes in blood pressure and electrical potential. However, electromechanics associated with heart diseases are not yet well understood for rats. In the electrical depolarization, a potential wave propagates through the whole ventricles as an electrical activation, leading to the mechanical contraction. This means that any change in electrical excitation can result in a change in muscle contraction. When looking into left-sided heart failure, the left ventricle loses its ability to contract properly or it becomes too stiff and unable to relax normally. These phenomena can be considered as heart diseases. Tissues remodelling may primarily cause a region in the ventricle to become too stiff with reoriented fibres and significant change in its electrical conduction properties as well. This can lead to electromechanical dyssynchrony and then the blood is not pumped sufficiently into its circulation. Hence, in this study, the influence of left-sided heart failure is investigated by making use of modelling and simulation of cardiac muscles in the rat biventricular model. To this end, we model the cardiac electromechanics of the biventricular model of a rat heart, whose 3D geometries (with and without an assumed diseased region) are constructed from MRI images provided by the Pediatric Cardiology at the University of Erlangen Nuremberg. The model is then developed with an approximated fibre orientation map and an adequate orthotropic material law. The characteristics of the action potential are based on the Aliev-Panfilov model with adjusted parameters for a general rat heartbeat [1]. The electromechanically coupled problem of cardiac muscle contraction is subsequently solved using a fully implicit finite element-based monodomain framework, in which the excitation-contraction process is completely treated using the active stress formulation [1]. Finally, our computational model and simulation results for the rat biventricular model can provide valuable insights into the healthy and diseased cases which allow to improve clinical therapy designs and medical devices significantly.

REFERENCES

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