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ELECTRIC PROPAGATION PATTERNS IN 3D ACUTE ISCHEMIC HEART USING GRAPHIC PROCESSING UNITS

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Background

Ventricular tachycardia and fibrillation are among the major cause of sudden cardiac death. Even though these arrhythmias arise from different clinical conditions, ischemic heart disease is the foremost perpetrator among them. Occlusion of the coronary artery, which causes ischemia, is followed by profound metabolic changes in the intracellular and extracellular space of the cardiac tissue. These metabolic changes are mainly hypoxia, increased concentrations of the extracellular K^+ (hyperkalemia), increased concentrations of intracellular Na^+, and Ca^{2+}, decreased concentration of extracellular Na^+, decrease of intracellular ATP, and acidosis[1]. In addition, the impact of ischemia in the myocardium is characterized with a high degree of heterogeneity comprising changes in electrophysiological properties between the healthy and ischemic regions[3]. These heterogeneities are produced not only intramurally, but also transmurally, in the depth of the ventricular wall (see Figure 1).

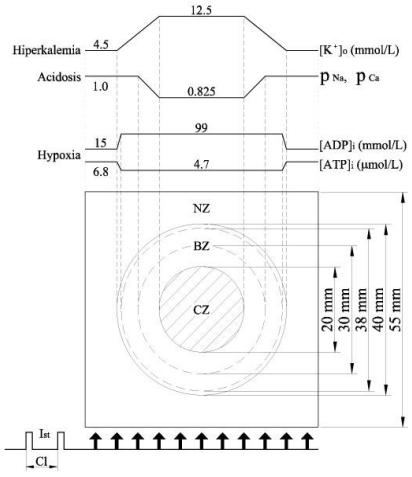

Figure 1. 2D representation of virtual ischemic zones, with the central ischemic zone (CIZ), border zone (BZ) and normal zone (NZ).

From an electrophysiological point of view, these changes imply alterations in action potential configurations, excitability, conduction velocities, refractive period among others, which enormously favour reentrant activity, and therefore arrhythmias and fibrillation.

Over the last years, mathematical modelling and computer simulations have been a useful tool in analysing electrophysiological phenomena. In this particular, one of the major contributions of computer electrophysiology has been in understanding important relations between electrophysiological parameters. For the ischemic heart, computer models have allowed to address the role of ischemic abnormalities in cardiac electrophysiological behavior[4]. However, the high computational cost of these simulations have restricted most studies to $2D^2$ simulations, with only few studies considering the full 3D ischemic heart[3,4]. This research work presents the solution of the electric activity in the acute ischemic heart using a parallel finite element code fully developed in CUDA.

Methods

A 3-D geometrically and anatomically accurate regionally ischemic human heart was simulated. The ischemic region was located in the anterior side of the left ventricle mimicking the occlusion of the circumflex artery. Realistic heterogeneity and fiber anisotropy has been considered in the model. The

electrical activity of each cell was reproduced using a modified version of the ten Tusscher and Panfilov 2006[5] action potential model. The model of regional ischemia was composed by realistically dimensioned transitional border zones (BZ) for the three main components of acute ischemia, connecting the normal zone (NZ) and the central zone (CZ) of ischemia. The effect of considering a thin layer of wash-out in the endocardium has also been studied. In the central zone of ischemia, the extracellular potassium concentration was set to 9.9 mM to mimic hyperkalemia, the inward Na+ current and Ca2+ current through L-type channels were scaled by a factor of 0.85 to imitate acidosis, and the intracellular ATP and ADP concentration were set to 5 mM and 99 uM respectively. The stimulation protocol consisted on the delivering five stimulation pulses at normal excitation position in the endocardium of the heart at a frequency of 1.25Hz, for preconditioning the tissue, followed by an extra-stimulus located in the border zone with different coupling intervals (CI).

Results and conclusions

The main results of the performed simulations can be summarized as follows: i) As a consequence of the applied extra-stimulus that originates an ectopic beat, reentrant activity is generated in all cases considered for CIs in the range 372-382 ms. This activity corresponds to an eight shape figure in some cases whereas in other cases it corresponds to a spiral like shape; ii) The reentrant activity generated as a consequence of the extra-stimulus ceases in all cases as a consequence of the interaction between wavefronts emerging from the from the wash-out zone into the ischemic zone. On the contrary, propagation patterns are highly modified when the wash-out zone is not present in the model. In conclusion, the model predicts the generation of figure-of-eight re-entries which cross the central ischemic zone formed in the epicardial surface due to the longer refractory period of the midmyocardial layers. Also, focal activity experimentally observed in the epicardium could be caused by re-entrant wavefronts propagating in the mid-myocardium that reemerge in the heart surface.

Regarding computational performance, the computing horse power of GPU technology have allowed for a reduction in total computational time when solving this problem, with accelerations up to 60x with a NVIDIA M2090 against a single Intel-Xeon Quad-core CPU.

Reference

[1] E. Carmeliet, Cardiac ionic currents and acute ischemia: from channels to arrhythmias. *Physiol Rev*. Vol. **79**, pp. 917-1017, 1999.

[2] J.M. Ferrero, B. Trenor, B. Rodriguez and J. Saiz, Electrical activity and reentry during acute regional myocardial ischemia: insights from simulations. *Int J Bif Chaos*. Vol. **13**, pp. 3703-3715, 2003.

[3] B. Tice, B. Rodríguez and Trayanova N, Arrthythmogenicity of transmural heterogeneities in a realistic model of regional ischemia. *Heart Rhythm*. Vol. **2**, pp. S261, 2005.

[4] E.A. Heidenriech, J.M. Ferrero and J.F. Rodriguez. *Patient-specific computational modeling*, pp. 81-104. ISBN 9789400745513, 2012.

[5] K.H.W.J. ten Tusscher, A.V. Panfilov, Alternants and spiral breakup in a human ventricular tissue model. *Am J Physiol Heart Circ Physiol*. Vol. **291**, pp. H1088-H1100, 2006.