Cardiac homogeneity despite asynchrony?

Cardiac homogeneity despite asynchrony?

[1]Roy Kerckhoffs, [1]Peter Bovendeerd, [2]Jiska Kotte, [3]Frits Prinzen, [4]Karel Smits, and [1,2]Theo Arts
[1]Eindhoven University of Technology, Department of Biomedical Engineering
[2]Maastricht University, Department of [2]Biophysics, and [3]Physiology
[4]Medtronic, Department of lead modeling

Introduction

Contraction of cardiac muscle fibers (myofibers) is initiated by a propagating wave of electrical depolarization. The use of a 3D finite element model of the heart combining wave propagation and wall mechanics may provide new insights in the interpretation of deformation, following conduction disturbances.

The relation between the moments of depolarization and increase in active myofiber stress (onset of crossbridge formation) in myofibers is not well known. In a mathematical model of the left ventricle (LV) we tested the hypothesis that electromechanical delays (EM-delay) of all myofibers are homogeneously distributed. Computed myofiber strain was compared with reported experimental results.

Material and methods

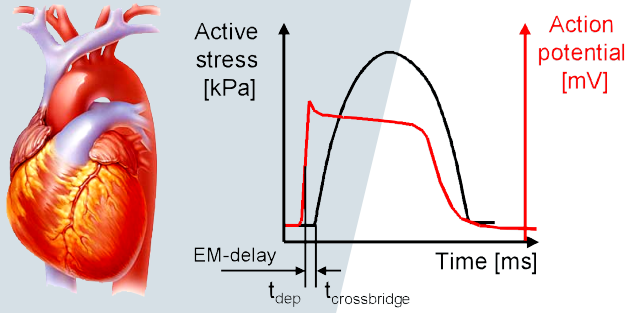

Figure 1 *Left: artist's impression of the heart. Right: the myofiber action potential [mV] and active stress [kPa] as a function of time. The moment of depolarization (t_{dep}) was solved in the eikonal-diffusion equation [1] as a function of position in the cardiac wall. Cardiac mechanics was solved from the momentum equation. Myofiber active stress increase started at the moment of crossbridge formation ($t_{crossbridge}$), and was dependent on time, sarcomere length, and sarcomere shortening velocity.*

Results

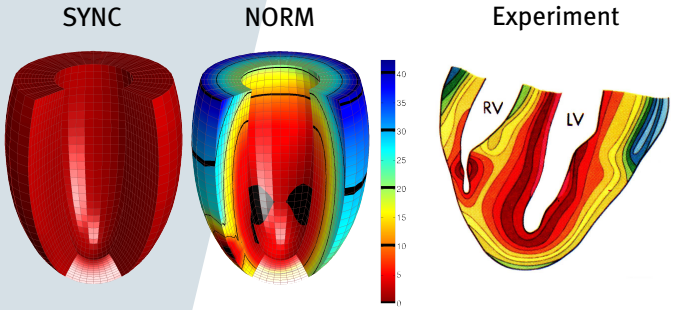

Figure 2 *Patterns of moment of crossbridge formation [ms]. SYNC simulation: unphysiological synchronous ($t_{crossbridge}$ = 0 ms). NORM simulation: crossbridge formation following a normal depolarization pattern ($t_{crossbridge}$ = t_{dep}), which is similar to measurements of depolarization [3].*

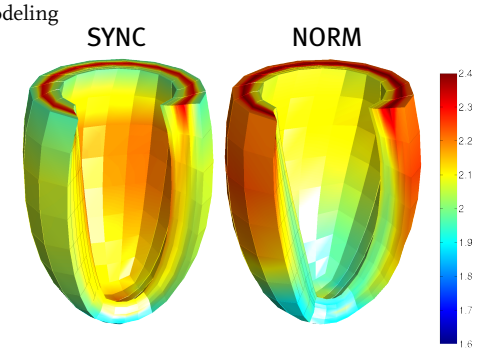

Figure 3 *Distribution of sarcomere length [μm] at beginning of ejection for the NORM simulation is less homogeneously distributed than in the SYNC simulation.*

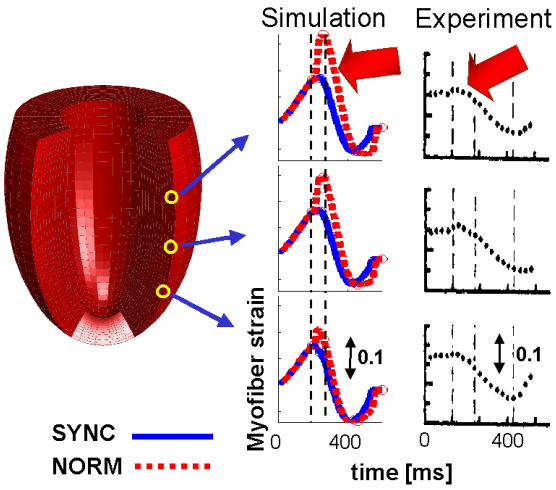

Figure 4 *Epicardial myofiber strain as a function of time from simulations (left column) and experiments (right column, [2]). In the NORM simulation large strains are present in the late depolarized regions (red arrows), whereas in the SYNC simulation strains are similar to the strains in the experiment.*

Conclusions

The simulations predicted either an unphysiological nonuniform contraction pattern during physiological depolarization, and a physiological contraction pattern during unphysiological synchronous depolarization. We reject the hypothesis of homogeneous EM-delay. The new hypothesis is that EM-delay times are heterogeneously distributed, such that a contraction in a normal heart is more synchronous than depolarization. This hypothesis is highly intriguing and urges experimental validation.

References:

[1] Colli-Franzone, P. et al.: *Mathematical modeling of the excitation process in myocardial tissue: influence of fiber rotation on wavefront propagation and potential field* (Math. Biosc. 101, 155-235, 1990)

[2] Delhaas, T. et al.: *Subepicardial fiber strain and stress as related to left ventricular pressure and volume* (Am. J. Physiol. 264, H1548-H1559, 1993)

[3] Durrer, D. et al.: *Total excitation of the isolated human heart* (Circ. 41, 899-912, 1970)

PO Box 513, 5600 MB Eindhoven, the Netherlands