COMBAT-VT:

Computational Model of the Mechano-Electrical Interaction in Ventricular Tachycardias

E. Kruithof[1], M.J.M. Cluitmans[2], P.H.M. Bovendeerd[1]

[1]Department of Biomedical Engineering, University of Technology Eindhoven
[2]Philips Research Eindhoven

Keywords: Finite Element Analysis | Heart Arrhythmia | Myocardial Infarction | Cardiac Electrophysiology | Cardiac Mechanics

MOTIVATION AND OBJECTIVE

Myocardial infarction causes **remodeling** of mechanical and electrical properties within the infarct area, disturbing the normal cardiac activities which increases the risk of **ventricular tachycardia** (VT). To improve treatment outcome, further insight in long-term VT risk after myocardial infarction is needed to optimize the selection of **patient-specific therapy**. Here, the effect of electrical abnormalities is well studied by means of computer models [1] but lacks insight in **mechano-electrical interaction**, which will be explored in this project.

TISSUE REMODELING

Infarct **tissue remodels** in both mechanical and electrical terms and involves an increase in stiffness, a loss of contractility, altered action potential durations and lowered conduction velocity with which the electrical activation front propagates over the myocardium (Fig. 1). The increased heterogeneity in electrical tissue properties is an important **determinant of VT risk**. Our hypothesis is that **electrical heterogeneity** is affected by **mechanical heterogeneity**.

MECHANO-ELECTRICAL INTERACTION

The cardiac myocytes are organized in a **fiber structure** over the left ventricle, arranged in series to combine their contracting forces during the ejection phase of the cardiac cycle. Mechanical remodeling alters the amount of **myofiber stretch** in and around the infarct area, which affects the electrical cell-cell communication (Fig. 2). By incorporating this interaction in the computer model, the electrically remodeled tissue area will be **extended** to parts outside the infarct area.

In this computational model, the relation between myofiber strain and electrical remodeling will be explored to study its effects on the initiation of VT.

REFERENCES

[1] Arevalo, H. J. et al. (2016). *Arrhythmia risk stratification of patients after myocardial infarction using personalized heart models*.

[2] Pimentel, R. C. et al. (2002). *Autocrine regulation of myocyte Cx43 expression by VEGF*.

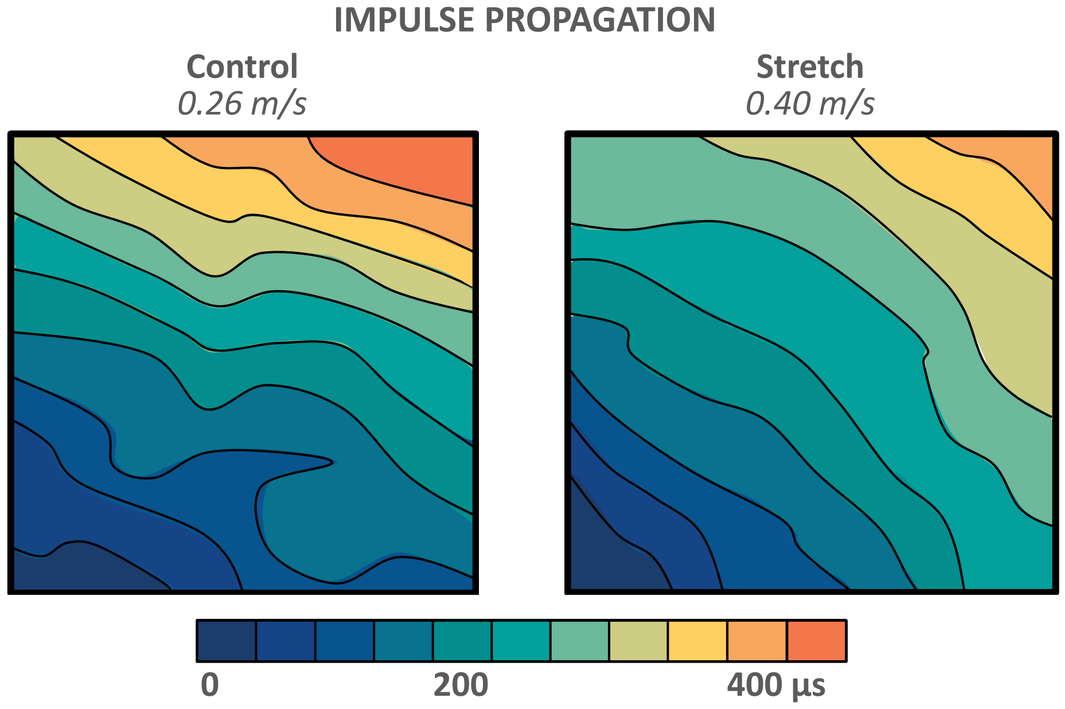

Fig. 2 Optical isochronal maps showing impulse propagation in rat myocytes. One hour of pulsatile stretch (10%) causes an increase in conduction velocity from 0.26 to 0.40 m/s. The isochrones are separated with 40 µs. Figure modified from [2].

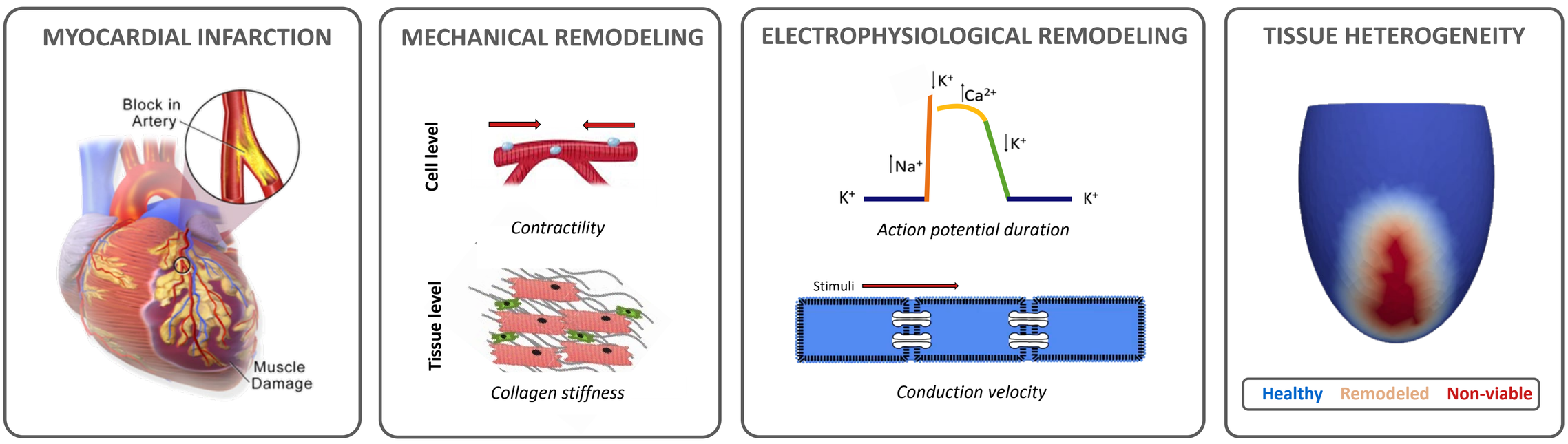

Fig. 1 Myocardial infarction leads to remodeled tissue properties in the damaged area. Mechanical remodeling involves a loss of contractility of cardiomyocytes and increased tissue stiffness due to collagen formation. Electrophysiological remodeling involves an altered action potential duration due to reduced conductivities of ion channels in the cell membrane and a lowered conduction velocity due to remodeled gap junction function. The resulting tissue heterogeneity can be incorporated in a computational model of the left ventricle to study its effects on VT risk.

Contact details: e.kruithof@tue.nl