Changes in activation wavefront in dependence on propagation velocity. Simulation study.

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INTRODUCTION It has been shown in previous simulation studies that a slowing in conduction velocity of an activation wavefront during depolarization can result in signs of left ventricular hypertrophy in spite of the fact that the left ventricular mass remains unchanged.

The aim of this study was to visualize the relationship between the changes in the extent and position of the activation wavefront and the heart vector in cases of slowing in conduction velocity in the left ventricular midwall.

METHODS A simplified geometrical model of the ventricles created from parts of ellipsoids was used for simulations. The wall of each ventricle was divided in five layers. For each layer an action potential amplitude and duration could be defined separately as well as the propagation velocity. To mimic Purkynje fiber properties, the propagation velocity in the endocardial layer was defined three times higher than in other tissue. It was possible to change the properties of the model in a selected subvolume.

The action potential propagation was started in selected starting points according Durrer's conclusions and later developed by Huygens principle. The equivalent electrical heart generator was computed in the form of multiple dipole. First, a simulation of normal heart activation was performed. Then two pathological cases were simulated: 1. The propagation velocity was slowed down by 50% in the middle layers of the whole left ventricle (LV) and 2. The propagation velocity was slowed down by 50% in the middle layers only in anteroseptal (AS) region of the LV.

For each simulation a resulting heart vector (HV) was computed and the propagation wavefront was visualized during the depolarization period.

RESULTS For both cases the heart vector moved forward and its horizontal component increased (Fig1. a, b). The mean spatial vector magnitude increased approximately 2.5 times for the whole LV changes and by 30% for AS changes. The maximum spatial vector magnitude appeared considerably later than in the reference case (41/65 step) for the whole LV changes but for AS changes it was almost at the same time (55/58 step).

DISCUSSION A slowing of propagation velocity changes a balance of propagation wavefronts. For changes in the whole LV it is the balance between left and right ventricle; while the resulting vector of right ventricle decreases after a right ventricular breakthrough the vector of the left ventricle is still large because of slow movement of propagation wave (Fig1. c,d). For AS changes the balance between anterior and posterior wavefront of the left ventricle is changed in favor of forward orientation of resulting HV.

Computer modeling is a useful tool for understanding ambiguous clinical observations.

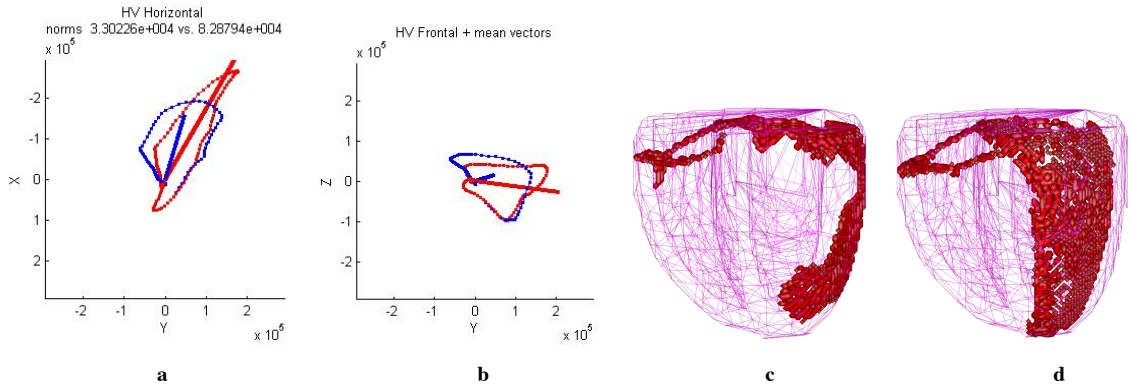

Figure 1. HV for the reference (blue) and for whole LV changes (red). **a –** Shift of the HV forward, **b** – Increasing of the HV horizontal component, **c,d** - Actual propagation wavefront in the time of maximal vector for LV changes (step 65), reference (c) and slowing activation velocity(d).