The effect of midwall conduction velocity slowing on the QRS pattern

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INTRODUCTION It has been documented by histopathological studies that left ventricular hypertrophy is accompanied by an increase in fibrotic tissue. More recently, cardiac magnetic imaging (MRI) has shown midwall fibrosis in patients with cardiomyopathy, aortic stenosis, and mitral insufficiency. Fibrotic tissue represents a restrain to the normal sequence of impulse propagation and thus can modify the resultant QRS pattern.

In this study we simulated the effect of diffuse and of localized changes in depolarization sequence due to slowed conduction velocity in the midwall of the left ventricle and in the anteroseptal region of the left ventricle on the QRS morphology using computer modeling.

METHODS The used model defines the geometry of cardiac ventricles analytically as parts of ellipsoids, and allows changing the conduction velocity in individual layers of myocardium. The conduction velocity was slowed by 50% in one and two midwall layers of the left ventricle in the anteroseptal region and in the whole LV, respectively. The dimensions of the LV/ left ventricular mass were not changed. The results are presented as derived 12-lead electrocardiograms, and corresponding values of ECG criteria for left ventricular hypertrophy (ECG-LVH criteria) (Gubner criterion, Sokolow-Lyon index and Cornell voltage).

RESULTS The midwall slowing in conduction velocity resulted in QRS complex changes consistent with ECG-LVH criteria, the changes were more pronounced if two layers were involved. The slowing in the anteroseptal region resulted in shift of the electrical axis to the left, the maximum spatial vector magnitude did not increase. Consequently, the changes were more pronounced in ECG-LVH criteria that are based on the limb leads: the Gubner criterion and the Cornell voltage. The slowing in the midwall of the whole left ventricle led to the shift of the electrical axis to the left, and to the increase of the spatial QRS vector magnitude if two layers were involved, resulting in an increase in all ECG-LVH criteria mostly pronounce in Cornell voltage.

DISCUSSION Using computer modeling we showed that the midwall alteration of impulse propagation modified QRS complex. The changes are consistent with QRS patterns usually interpreted as the effect of "hypertrophy" i.e. increased left ventricular mass. Midwall fibrosis documented by CMR in patients with LVH could therefore mimic the "classical" signs of LVH. The results of this study stress the importance of considering conduction characteristics in interpreting QRS pattern in LVH patients especially in discrepant ECG findings.

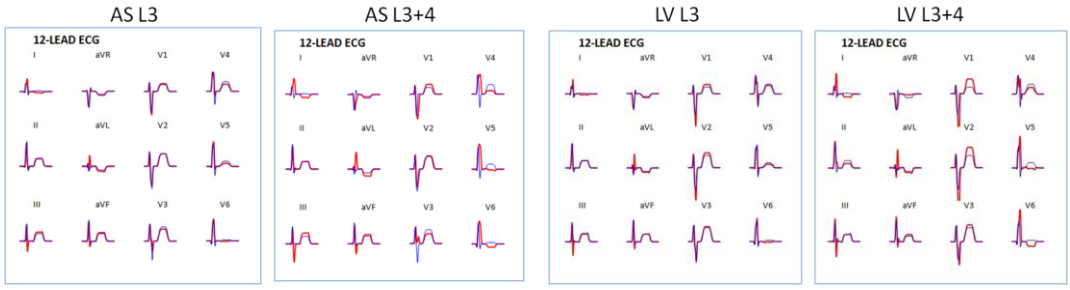

Figure 1. The effect of midwall slowing of conduction velocity on the simulated 12 lead electrocardiograms. AS L3: slowing in one layer of the anteroseptal region, AS L3+4: slowing in two layers of the anteroseptal region, LV L3: slowing in one layer of the left ventricle; LV 3+4: slowing in two layers of the left ventricle.