REPOLARIZATION CHANGES DISPLAYED IN SURFACE ARI MAPS. A SIMULATION STUDY

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Abstract: **Body surface distribution of activation-recovery intervals (ARI) and its ability to reflect local variations of the repolarization process in different myocardium segments was analyzed. Body surface potentials were simulated for normal activation - repolarization and in cases with local shortening and/or decrease of the action potential (AP) of myocardial cells. Isotropic model of analytically shaped ventricles and cellular automata were used to simulate the spread of activation. AP was changed in defined regions in the anterior and posterior left ventricle (LV) that represented 3 to 12% of the ventricular volume. Corresponding body surface integral maps and ARI maps were analyzed. While shortening of AP in the anterior LV was clearly projected on the left antero-lateral superior torso, AP changes in the posterior LV projected mainly in the middle part of the posterior torso were influenced also by other processes in the myocardium and hardly to distinguish. Obtained results indicate that ARI maps may reflect local changes of repolarization in both subendocardial and transmural myocardial regions and could help to identify such regions namely if they are close to the anterior chest surface.**

INTRODUCTION

Vulnerability to ventricular arrhythmias is connected with inhomogeneity of myocardium repolarization caused by local changes of action potential duration and amplitude. Under simplified conditions, ARI measured as interval between the most negative derivative within the QRS complex and the most positive derivative near the T-peak of an electrographic signal can be considered as some projection of AP duration in the underlying myocardium. Similarly, ARI estimated from many surface ECG signals, though strongly influenced by the torso volume conductor, could be used as some indicator of local repolarization properties. Tank experiments showed that despite the smoothing effect of torso, there might be a high correlation between epicardially recorded AP duration and superficially measured ARI. Preliminary study of surface ARI maps from real measurements [1] confirmed their reproducibility that was much better than that of QT intervals. The aim of this study was to test the ability of surface ARI maps to reflect AP changes in the heart and their localization.

METHOD

Finite element model of heart ventricles [2] with analytically defined geometry and element size of $1mm^3$ was used to represent myocardium depolarization and repolarization. Conduction velocity and AP shape were defined for each element. Several layers of elements with up to five different AP durations decreasing from endocardium to epicardium were used to build up the ventricular walls and the septum. AP were approximated by step upstroke, constant plateau and 90 ms linear down-slope. AP duration measured in the middle of the down-slope was 126 to 162 ms in the RV and 138 to 177 ms in the LV and in the septum. A layer with 3 times increased conduction velocity on the endocardial surface simulated Purkinje fibers. Starting points of activation were in agreement with experimentally observed early-activated regions in a normal human heart. Activation spread was governed by a cellular automata supposing isotropic myocardial tissue. 168 segmental dipoles were used to represent the cardiac electric generator. Potentials on the surface of a realistic torso model with basic inhomogeneities representing lungs and heart cavities were computed using the boundary element method. ECG signals from 84 points of a 12x7 mapping grid were used to obtain potential and integral maps as well as surface isochronal ARI maps. Normal heart repolarization and repolarizations with AP locally shortened by 25% and/or decreased by 30% from the normal values were simulated. Regions of changed AP were defined in two positions in LV as shown in Fig.1: anteriorly near the apex and postero-laterally close to the heart base. In both positions, lesions of three different sizes were created and represented 3-12% of the myocardial volume. Small and medium lesions were subendocardial while the biggest one was always transmural.

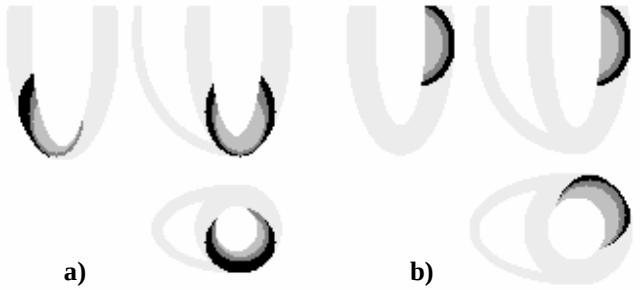

Fig. 1: Regions with changed AP in the left ventricle:
a) anterior region (3%, 6% and 10% of the volume)
b) posterior region (4%, 8% and 12% of the volume)

RESULTS

The overall patterns of simulated normal body surface potential maps as well as the patterns of ARI maps were in good agreement with those measured in real subjects [1].

Example of simulated normal ARI map and ARI map corresponding to shortened AP duration in the anterior region are shown in Fig.2. Based on the definition, ARI were evaluated only for ECG signals with positive T wave while ARI in areas with negative T waves (upper right anterior and posterior torso) were not considered and are not displayed.

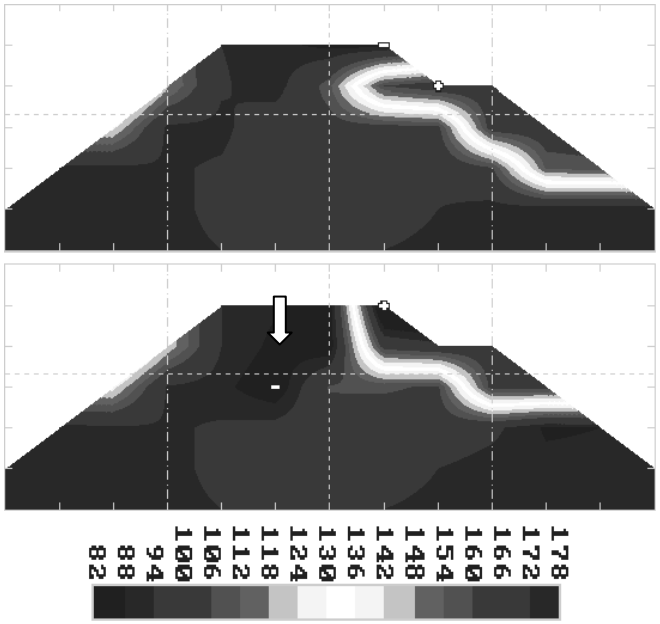

Fig.2. Example of simulated ARI maps. Left part of each maps represents anterior torso, right part the back. ARI durations in milliseconds are represented by gray levels. ARI map for normal AP (upper) and for shortened AP in transmural anterior region 10% of volume (lower map).

Changes of AP simulated in anterior regions were projected mainly to the left antero-lateral superior torso (in the middle of the map, near to the transversal level) but partially also to the left inferior posterior torso. AP changes in posterior LV regions close to the heart base were projected mainly in the middle part of the posterior torso and partially also to the left lateral superior torso.

While merely decrease of AP was difficult to recognize in the ARI maps, shortening of AP was clearly visible and changes in the map were proportional to the size of the lesion. Combined shortening and decrease of AP strengthened the changes in ARI maps. Findings in QRST integral maps were partially in contrast with results in ARI maps. AP decrease was reflected stronger than AP shortening and in most cases, subendocardial lesions of the same size as transmural lesions produced greater departures from normal QRST integral maps. Evaluation of changes in ARI maps and QRST integral maps for medium and large lesions is in Table I a), b).

DISCUSSION

As it is difficult unambiguously interpret ARI for ECG tracings with negative T wave, ARIs were not evaluated in this area (right superior part of the torso). In a transition area between negative and positive T values, T waves are small and often multi-phasic. In such region even small changes of AP caused that the maximum of T wave derivative "jumped" to other part of the T wave and possibly different processes in the myocardium were mixed in the computed ARI.

Limitation of the study is the use of isotropic myocardium model. As reported elsewhere, this can cause inaccuracy of the simulated potentials namely if the regions with changed AP were not transmural. Another limitation arises from the simulated linear AP down-slope that can influence estimation of the "recovery time instant".

Although the resolution of surface mapping is in principle limited by the smoothing effect of torso, results of our simulations suggest that AP shortening alone as well as in combination with AP decrease can be recognized in surface ARI maps, particularly in regions underlying the anterior chest. AP changes in posterior LV were also clearly visible in the middle of the inferior posterior torso but the computed long ARI values mostly did not reflect “true” AP changes but were influenced by projections from other parts of the myocardium, probably including the left septum.

TABLE I

Comparison of normal and changed ARI and integral maps for changed repolarization in anterior and posterior heart regions

a) anterior regions

AP change	size	masked ARI		QRST integrals	
		Correl.	rms [%]	Correl.	rms [%]
Shortening	6%	0.76	15	0.99	24
	10%	0.65	19	0.99	15
Decrease	6%	0.96	6	0.94	42
	10%	0.94	7	0.98	25
Shortening & Decrease	6%	0.65	19	0.85	59
	10%	0.62	20	0.98	35

b) posterior regions

AP change	size	masked ARI		QRST integrals	
		Correl.	rms [%]	Correl.	rms [%]
Shortening	8%	0.68	22	0.97	26
	12%	0.80	16	0.98	22
Decrease	8%	0.96	7	0.88	47
	12%	0.94	11	0.93	37
Shortening & Decrease	8%	0.75	20	0.78	65
	12%	0.82	20	0.85	52

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[2] V. Szathmáry, I. Ruttkay-Nedecký: "Model study of effects of different repolarization patterns in the left and right ventricle on the resultant cardiac vectors" in: *Electrocardiology 2000*, 2001, pp. 97-102.