DIFFERENCES IN QRST INTEGRAL MAP VARIABILITY IN HEALTHY SUBJECTS AND ARRHYTHMIA PATIENTS

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1. Introduction

Ventricular fibrillation is the most common cause of sudden cardiac death (SCD). According to experimental and theoretical studies, the biophysical substrate of ventricular fibrillation is connected to the structural and/or dynamical repolarization dispersion (RD) changes. According to a recent scientific statement of the AHA/ACCF/HRS, current non-invasive risk assessment methods are not efficient enough; further efforts are justified for their improvement. In our long term pursuit for an improved non-invasive SCD risk assessment, we used QRST body surface potential maps instead of conventional ECG leads, in order to grasp the whole RD related information accessible on the thoracic surface [1].

Due to the spatial filtering effect of the body as a volume conductor, RD, on the epicardial surface generally results in a "dipolar pattern" on the thoracic surface by visual inspection (i.e. one convex positive and one negative region) on the chest surface. However, non-dipolarity indices (NDI), computed from the coefficients of Karhunen-Loeve (KL) expansion of the QRST integral maps characterize sensitively the spatio-temporal variability of the subsequent QRST integral maps [2]. In our small-sample study, beat-to-beat NDI plots sensitively illustrated the significant changes of RD in the group of implanted cardioverter (ICD) patients with documented malignant arrhythmia vulnerability.

In this study we attempted to give a source level explanation of the observed normal and pathological NDI behaviour, with a special regard to the extremely large NDI spikes. To this end, numerical chest and heart models were used.

2. Material and methods

Details of our computer model of human cardiac ventricles were described previously [3]. Briefly, the model is defined in a 3-dimensional matrix of cubic elements, where these elements represent the functional property of an excitable cardiac tissue and ventricular transmembrane action potentials (MoAPs). To simulate the repolarization heterogeneity, ventricular walls are sliced to 5 layers, paralleling with their inner and outer surfaces. The characteristics of model elements may be defined differently in dependence on their localization in respective layers.

To compute the electrocardiological potentials on the surface of human thorax, the multiple dipole model of the cardiac generator was inserted in a realistically shaped torso model. Models of lungs with 4x lower conductivity than general conductivity of the torso and ventricular cavities with 3x higher conductivity were inserted in torso introducing (assuming) main inhomogeneities. The electric potentials on the body surface were computed in the points of the torso model using boundary element method (BEM), which was proposed already [4] and is frequently used up to this day.

3. Results and conclusion

In statistical terms NDI histograms calculated from our BSPM measurements showed significantly skewed lognormal distributions, sometimes with NDI values up to 90% in arrhythmia case (Figure 1), while in normal subjects the subsequent NDI values remained typically in the range of 10%-20% with nearly symmetric distributions.

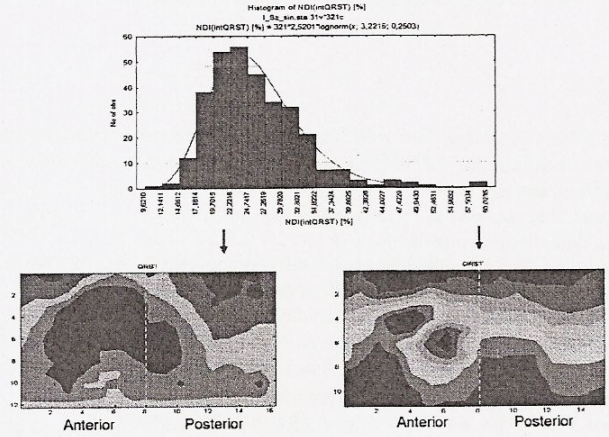

Fig. 1. NDI histogram of an arrhythmia patient with dipolar (left) and non-dipolar (right) QRST integral maps

A systematic exploration of MoAP modulation effects on the NDI values revealed that the NDI spikes frequently observed in ICD patients can be generated in our conceptual model by apical extra activation fronts spreading from the epicardium to the endocardium. According to our assumption, the MoAP duration in this case is the largest in the firstly activated epicardial layer, gradually decreasing in the subepicardial layers, i.e. the physiological MoAP duration decreasing from the endocardium to the epicardium is reversed. We should remark, that the reversed MoAP duration at the apex might be a result of a changed intra- and extracellular level regulation of the ionic transport.

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