

## Model Interpretation of Pathological Body Surface QRST Integral Maps Related to Action Potential Heterogeneity

<sup>1</sup>G. Tuboly, <sup>1</sup>G. Kozmann, <sup>2</sup>V. Szathmáry, <sup>3</sup>J. Švehlíková, <sup>3</sup>M. Tyšler

<sup>1</sup>Department of Electrical Engineering and Information Systems, Faculty of Information Technology, University of Pannonia, Veszprém, Hungary,

<sup>2</sup>Institute of Normal and Pathological Physiology, Slovak Academy of Sciences, Bratislava, Slovakia

<sup>3</sup>Institute of Measurement Science, Slovak Academy of Sciences, Bratislava, Slovakia  
Email: tuboly.gergely@virt.uni-pannon.hu

**Abstract.** *According to theoretical and clinical considerations the risk of re-entry type malignant arrhythmia can be predicted by body surface QRST integral maps. Our aim was to understand better the intracardiac processes causing noninvasively measurable action potential (AP) heterogeneity. For this task we used a computer model of human cardiac ventricles with modifiable AP patterns to investigate the process on source level. To estimate the relevant potential maps on the body surface, we used a realistically shaped piecewise homogeneous torso model. The level of AP heterogeneity was quantified by the non-dipolarity index (NDI) of the QRST integral maps, calculated beat-to-beat. We found that the source level origin of extreme NDIs was in the apical region of the heart, due to significantly diminished or reversed transmural action potential gradients or myocardial necrosis.*

*Keywords: Malignant Arrhythmia, QRST Integral Maps, Heart Model, Torso Model, Non-dipolarity Index*

### 1. Introduction

While atrial fibrillation is considered to be a major risk factor of stroke, ventricular fibrillation can end in sudden cardiac death. According to experimental and theoretical studies, the enhanced ventricular fibrillation is related to the heart muscle cells' pathological action potential (AP) heterogeneity. Based on recent scientific statements, effective ventricular arrhythmia risk assessment methods are not available, therefore the currently used techniques have to be improved [1].

In our long term pursuit for such an improved non-invasive sudden cardiac death risk assessment method, we used body surface QRST integral maps instead of signals resulted by conventional ECG systems, in order to consider the whole AP heterogeneity related information accessible on the thoracic surface [2].

Due to the spatial smoothing effect of the body as a volume conductor, the physiological level of AP heterogeneity on the epicardial surface generally results in a dipolar QRST integral map on the thoracic surface. However, if this level of heterogeneity is pathological, QRST integral maps usually appear as non-dipolar. To quantify whether the non-dipolarity is significant, we used non-dipolarity indices (NDIs), computed from the coefficients of Karhunen-Loeve (KL) series expansion of the QRST integral maps. This way it can be characterized the spatio-temporal variability of the subsequent QRST integral maps [2]. In our small-sample study, beat-to-beat NDI plots sensitively illustrated the increased lability of AP heterogeneity in the group of implanted cardioverter defibrillator (ICD) patients with documented malignant arrhythmia vulnerability, compared to the normal subjects.

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. This investigation was performed by the help of a numerical heart and torso model.

## 2. Subject and Methods

### Heart Model

For AP property settings, we used a computer model of human cardiac ventricles. This model consists of small volume segments with modifiable AP patterns (MoAPs). Each segment includes 5 different layers from the endocardium to the epicardium and each layer is represented by a modifiable AP both in duration and amplitude. The starting points and initial time instants of the activation can also be programmed, just like the propagation velocity [3]. The default configuration of this model represents a normal heart (Fig 1), consequently cardiac cycle with low NDI value.

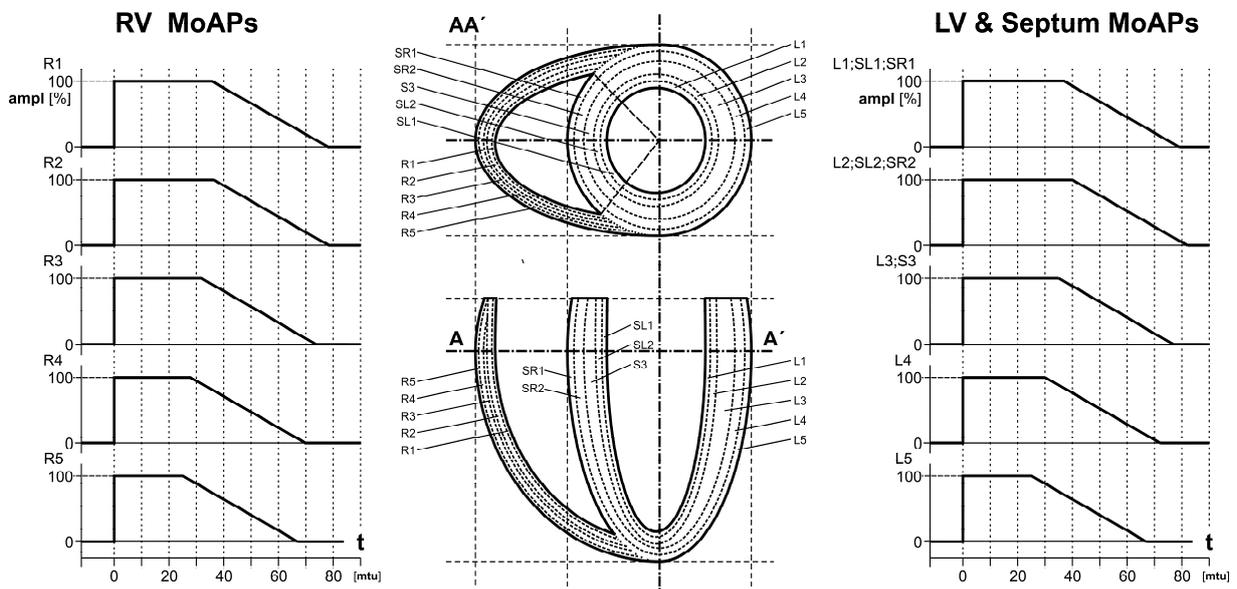


Fig. 1. The default configuration of the heart model. The left part shows the MoAP of the right ventricle and the right part represents the MoAP of the left ventricle and the septum.

### Torso Model

To obtain the QRST integral maps on the body surface, we inserted the heart model into a realistically shaped piecewise homogeneous torso model. In this model lungs are taken into consideration with 4 times lower conductivity than general conductivity of the torso, ventricular cavities with 3 times higher conductivity. The electric potentials on the body surface are computed in the surface points of the torso model using boundary element method [4].

According to Geselowitz, the amplitude of QRST integrals at an arbitrary body surface point  $P$  is a function of the AP heterogeneity, in other words, it is the function of the gradient of the  $\mu$  of MoAP areas (1) of the myocardium [5]. Consequently, beat-to-beat application of (1) provides a non-invasive tool that can be used to study the spatio-temporal variability of AP properties i.e. the AP heterogeneity.

$$\int_{QRST} \phi(P, t) dt = -k \iiint_{V_s} \mathbf{z}(P, \mathbf{r}) \nabla \mu(\mathbf{r}) dV_s \quad (1)$$

$$\mu(\mathbf{r}) = \int_{QRST} [\phi_m(\mathbf{r}, t) - \phi_{mr}(\mathbf{r})] dt \quad (2)$$

where

$\phi_m(\mathbf{r}, t)$  membrane potential at time  $t$

$\phi_{mr}(\mathbf{r})$  membrane resting potential at point  $\mathbf{r}$

$V_s$  volume of sources (myocardium)

$k$  constant

$z$  vector of transfer coefficients between point  $P$  and  $\mathbf{r}$ .

In a concise way QRST integral maps are characterized by the beat-to-beat sequence of NDI (3), based on the  $c_i$  components of the KL series expansion.

$$NDI = \frac{\sum_{i=4}^{12} c_i^2}{\sum_{i=1}^{12} c_i^2} = \frac{P_{ND}}{P_D + P_{ND}} \quad (3)$$

where

$P_D$  BSPM signal power represented by the “dipolar” KL components ( $i$ : 1-3)

$P_{ND}$  BSPM signal power represented by the “non-dipolar” KL components ( $i$ : 4-12) [6].

### 3. Results

NDI values obtained by the reference (normal) AP properties and by the 3 types of AP modulations (cases 2-84) are shown in Fig. 2.

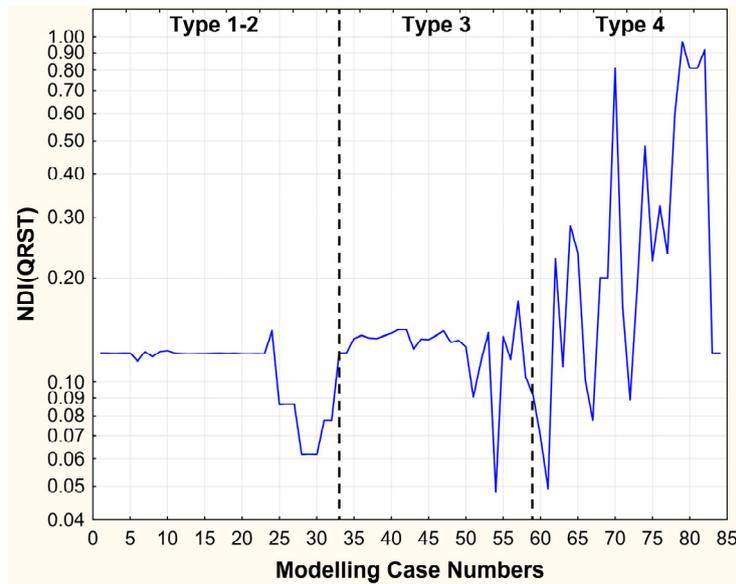


Fig. 2. NDI values of the simulated QRST integrals. Case 1 represents the normal configuration of the model (Type 1) and cases 2-84 show the NDI values of AP modulated simulations (Type 2-4).

Type 1 represents case 1, i.e. the baseline configuration of the heart model when no modifications were made. In Type 2 (cases 2-33) simulations the modulation of wavefront

starting points on endocardium and starting time instants were modulated in anterior, posterior, lateral and septal regions. According to Fig. 2 these changes did not result in high NDI values.

Type 3 (cases 34-57) modifications show cases related to mid-wall and basal AP modulations. Even the large extent changes caused minimal perturbation of the NDI values.

Type 4 (cases 58-84) simulations represent AP property changes in the apical region. Fig 2 shows that the extreme high NDI values belong to this group. Largest values were obtained by reversing the direction of the transmural gradient of AP durations and by simulating transmural, subendocardial and subepicardial myocardial infarction in the apical region.

#### 4. Discussion

The results show that extreme NDI cardiac cycles could be produced by local changing the AP properties in the apical region. Similar changes in the mid-anterior, mid-lateral and mid-posterior locations could evoke measurable, but significantly smaller changes in the NDIs.

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