

Relation between Depolarization and Repolarization Phases in Body Surface QRST Integral Map

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Abstract

The aim of the study was to assess the relations between QRST and STT, QRS integral maps in three groups: healthy volunteers, patients without arrhythmia and patients with implanted cardioverter-defibrillator (ICD). The obtained results showed that the mean correlation coefficient between STT and QRST integral maps was highest in the group of healthy subjects while the mean correlation coefficient between QRS and QRST integral maps was lowest in the same group of subjects. The relation of depolarization phase with repolarization phase and the influence of depolarization sequence on repolarization sequence is noticeably disrupted in patients with impaired systolic function. In such cases the QRST integral maps seems to reflect depolarization-repolarization disorder rather than only repolarization dispersion.

1. Introduction

The area under the QRST complex at body surface ECG lead is used to define the ventricular gradient what was proposed by Wilson in 1934 [1]. It is a measure of the electrical forces produced by local variations in the recovery process and do not depend on the course of the excitatory process over the ventricular muscle in the normal heart state. As it was shown in Ramanathan work [2], in the healthy heart the recovery process is dominated by local repolarization variations due to fast spread of activation, where the Purkinje system plays key role. That is why body surface maps of QRST area are used to assess primary repolarization changes i.e. independent of the activation sequence. The QRST integral maps are also used to assess the repolarization inhomogeneity [3-7]. However ischemia, in particular myocardial infarction, can cause changes in the activation process which might affect the action potential shape in the activation phase [8] what can change relation between activation and recovery processes. In the present study the QRST, STT and QRS integral maps are analyzed in both healthy and patients groups.

2. Methods

The group of 82 subjects consisting of 25 healthy subjects and 57 patients with heart disease (coronary artery disease mostly): 23 pts without ventricular arrhythmia and 34 pts threatened by ventricular arrhythmia with implanted cardioverter – defibrillator (ICD) was studied (Table 1).

Table 1. General information about studied group.

	NUMBER	AGE
NORM	25	49±15
NON-ICD	23	52±10
ICD	34	65±12

High Resolution multi-lead ECG system Active Two (Biosemi) with 64 surface leads was used to record ECG signals. Active electrodes (with preamplifier placed beside the AgCl₂ sensor) were applied to the anterior and posterior chest. Lead location proposed by SippensGroenewegen [9] was used (Figure 1). The 64 unipolar ECGs were simultaneously recorded for 15 minutes in an unshielded environment. Next, signals were amplified and digitized with 4096 Hz sampling frequency and 24 bits amplitude resolution and sent to the computer via optical fiber.

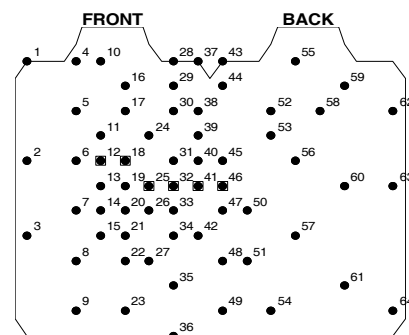


Figure 1. Lead arrangement around the torso. Standard ECG leads are marked by squares.

The raw ECG data were filtered using LP Butterworth filter limiting frequency to 300 Hz and decimate filter decreasing sampling frequency to 1024 Hz. Wilson's central terminal was subtracted as the reference point for all measured chest potentials. Next, all signals (if heart rate was varied with the standard error ± 2 bpm) were simultaneously averaged in time using cross-correlation function. The QRS complex was used as the template for cross-correlation function calculations. To receive low noise level both the number of averaged cycles and correlation coefficient were fitted. The level of noise was measured on the 20 ms isoelectric U-P interval, before the beginning of the next P wave. PQ interval was not used as isoelectric line due to atrial repolarization presence, what was confirmed in work [10]. The number of averaged cycles and the value of the correlation coefficient depended on the noise level of the averaged signal. In the whole studied group the number of averaged cycles varied from 10 to 150 cycles and the correlation coefficient from 0.96 to 0.99. The received noise level was 0.1 to 1.8 μV .

Algorithm applied to detect ECG characteristic points was based on Singular Value Decomposition (SVD) method proposed by Acar in [11]. As a result of SVD decomposition of ECG signals' matrix M into unitary matrices U and V and diagonal matrix Σ :

$$M = U \Sigma V^T \quad (1)$$

The reduced- space data matrix S was obtained by projecting matrix M down into the reduced space defined by only the first 3 left - singular vectors of unitary matrix U . From this three vectors of matrix S (S_1, S_2, S_3) the Root Mean Square signal was calculated (Figure 2). R – peak was marked as the maximum of the RMS signal, T maximum was marked in relation to R peak. The P wave onset and U wave offset were calculated by inspecting the stationarity of the time relation between vectors S_1 and S_2 in respectively defined time windows. Next the onset of Q wave and the offset of S wave were calculated on the difference signal of RMS signal using thresholding.

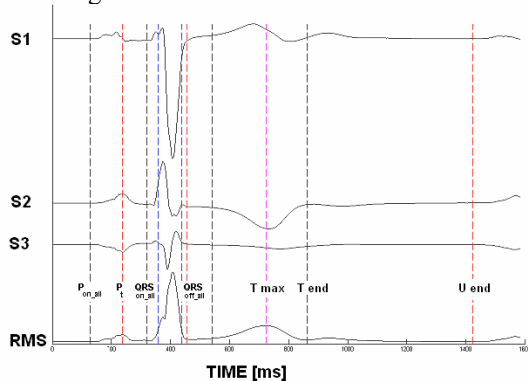


Figure 2. Waves ends detection on RMS signal.

The T wave end was established as the minimum of RMS signal in time window in relation to T-wave maximum. Then in each lead individual waves ends were established in relation to global ends of waves using thresholding (Q onset, S offset) or extrema searching procedures (P, R, T, extrema).

3. Results

The integral maps of QRS, STT and QRST complexes were obtained by calculating for each lead the algebraic sum of all instantaneous potentials from the wave onset to the wave end multiplied by the sampling interval. The values were plotted on the diagram representing the thoracic surface and iso-integral maps were drawn.

Below the QRS, STT and QRST integral maps of the healthy person and patient after myocardial infarction without implanted ICD are shown (Figure 3).

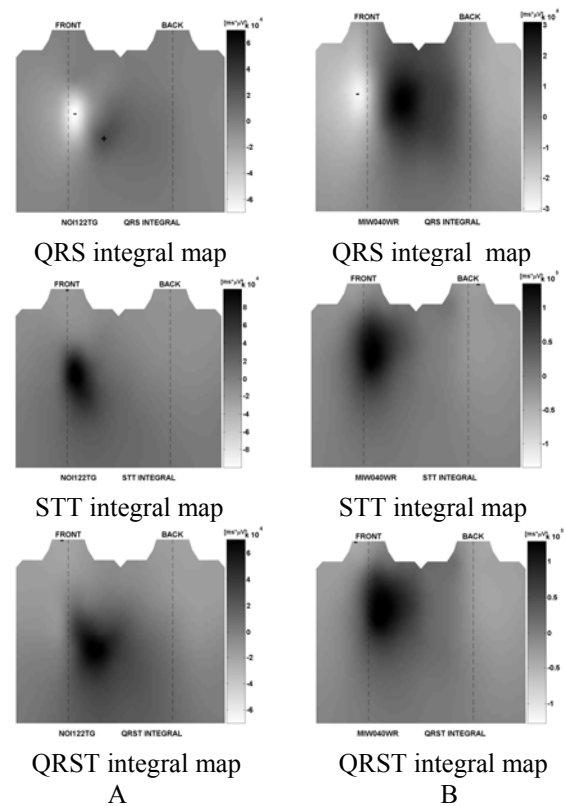


Figure 3. The QRS, STT and QRST integral maps for A – healthy person, B – MI patient without ICD.

In Figure 4 the QRS, STT and QRST integral maps for two patients after myocardial infarction with implanted ICD are presented.

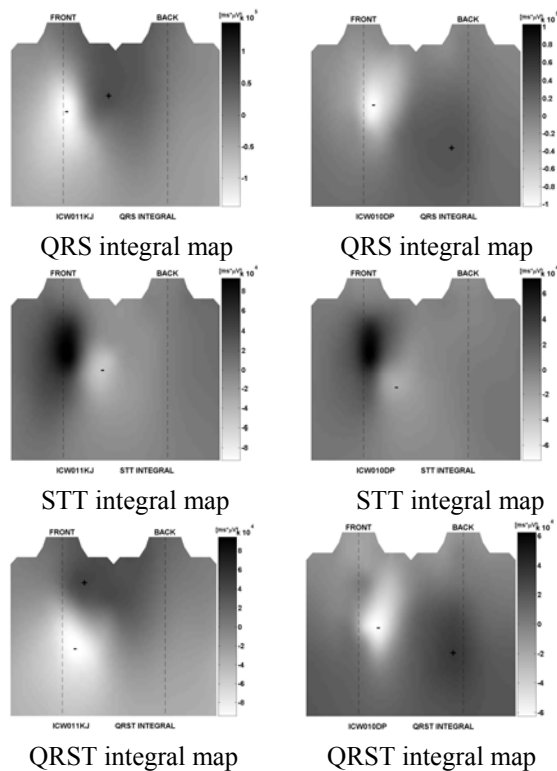


Figure 4. The QRS, STT and QRST integral maps for two MI patients with implanted cardioverter-defibrillator.

The correlations between QRST, STT and QRS integral maps were calculated in three groups: healthy volunteers, patients after myocardial infarction (MI) and patients with implanted cardioverter-defibrillator (ICD). Results are presented in Table 2.

Table 2 STT-QRST and QRS-QRST correlation coefficient values in studied groups.

	STT_QRST CORR	QRS_QRST CORR
NORM	0.94±0.06	0.48±0.51
NON-ICD	0.50±0.55	0.54±0.53
ICD	0.38±0.56	0.57±0.58

The statistical significance was studying using Mann-Whitney test. The significant differences ($p < 0.01$) were observed in values of STT_QRST between healthy volunteer group and both patients groups.

4. Discussion and conclusions

In presented study the relations between QRST and STT, QRS integral maps were examined. In healthy

subjects the correlation coefficients between spatial distribution of QRST and STT integrals were very high (mean 0.94) what reveals the dominant influence of repolarization phase on the QRST area. Similar conclusion was drawn by Ramanathan in work [2]. They studied the activation and repolarization processes on the surface of the heart applying inverse solutions to BSPM recordings. They have shown that the local repolarization is the major determinant of the repolarization sequence in the normal human heart and that activation sequence slightly influences the repolarization sequence. In patients without ICD the correlation coefficient between body surface QRST and STT integral maps was 0.50 and in group of patients with ICD the correlation coefficient was 0.38.

These findings might be explained by changes in ischemic cells' action potential shape and duration. Geselowitz in his work has shown that the spatial variations of the ventricular gradient are related to the local spatial variation of action potential area and hence to changes in action potential associated with cardiac pathologies such as ischemia or infarction [12]. In ischemic cells an increase in resting potential, a decrease in peak amplitude, an increase in rise time of the upstroke as well as change in duration cause the change in the action potential area what influences the QRST area.

In summary one may state that the distribution of QRST area on the body surface reveals the local repolarization distribution for healthy subjects but for patients with myocardial infarction the QRST area might be dominated by activation process i.e. QRS area, especially in patients with implanted cardioverter – defibrillators.

Acknowledgements

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