EXPERIMENTAL STUDY

Body surface integral maps in patients with arrhythmogenic right ventricular cardiomyopathy

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Abstract

Objectives: **The aim of this study was to evaluate changes in QRST integral maps in patients with ARVC.**
Background: **Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a progressive disorder of predominantly right ventricle characterized with arrhythmic events possibly leading to sudden cardiac death. QRST integral maps reflect local disparities of ventricular repolarization and resulting vulnerability to arrhythmias.**
Methods: **A group of 8 patients with ARVC and a control group of 8 patients with a concealed accessory pathway were studied. Body surface mapping was performed using a 63-lead Savard's system.**
Results: **Mean QRST integral map of patients with ARVC showed abnormal characteristics. The area of negativity was larger than normal and extended to lower border of thorax. Departure map of the mean QRST integral map of patients with ARVC showed areas with departure index <2 and >2 in lower part of chest and upper part of back. When statistically analyzed, areas with p<0,05 covered nearly lower half of chest and upper half of back.**
Conclusions: **We consider body surface QRST integral mapping to be an adequate method for evaluation of dispersion of ventricular repolarization in ARVC patients *(Tab. 1, Fig. 5, Ref. 17)*.**
Key words: arrhythmogenic right ventricular cardiomyopathy, body surface mapping, electrocardiography, repolarization.

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a progressive disorder of predominantly right ventricle characterized with arrhythmic events possibly leading to sudden cardiac death. It is characterized by fibrofatty replacement of right ventricular myocardium, initially with typical regional and later global right and some left ventricular involvement, with relative sparing of the septum (1). The fibrofatty infiltration is viewed as a healing phenomenon (2) in the setting of a programmed cell death – apoptosis (3).

Body surface mapping (BSM) provides a noninvasive measure to detect substrate for ventricular arrhythmias (4). QRST deflection area is largely independent of activation sequence and may permit evaluation of intrinsic ventricular recovery properties (5). It has been shown, that changes of recovery properties can be recognized on the basis of QRST deflection areas (6).

The aim of this study was to evaluate changes in QRST integral maps in patients with ARVC, reflecting local disparities of ventricular repolarization and resulting vulnerability to arrhythmias.

Methods

Study population

A group of 8 patients with ARVC and a control group of 8 patients with a concealed accessory pathway were studied. Evaluation in all patients included history, physical examination, laboratory tests, chest radiograph, 12-lead ECG recording, 24-hour Holter monitoring and echocardiography. Electrophysiologic study was performed in all patients except one. Right and left contrast ventriculography and signal averaging ECG were performed in addition in all patients with ARVC.

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Tab. 1. Characteristics of patients with ARVC.

	Programmed ventricular stimulation	Echocardiography	Late potentials
1 F	study not performed	RV 34mm, wall hypertrophy, kinetic disorders	positive
2 F	VT not induced	RV 34mm, regional muscle reduction	negative
3 M	VT monomorphic	RV 41mm, ARVC structure	positive
4 M	VT sust monomorphic	RV 31mm, ARVC structure	positive
5 M	VT sust polymorphic	RV 28mm, without kinetic disorders	positive
6 F	VT sust polymorphic	RV 26mm, LV hypokinesis	negative
7 M	VT sust monomorphic	RV 38mm, different thickness	positive
8 M	VT sust monomorphic	RV 32mm	positive

F – female, M – male, VT – ventricular tachycardia, RV – right ventricle, LV – left ventricle, sust – sustained

Patients with ARVC

There were 5 men and 3 women with ARVC (mean age at the time of body surface mapping 35.6 years, range 18–49 years). All patients fulfilled the criteria for diagnosis of ARVC (1), modified by Corrado (7). During programmed ventricular stimulation a sustained monomorphic ventricular trachycardia was induced in 3 patients and sustained polymorphic ventricular tachycardia was induced in two patients. Ventricular tachycardia was not induced in one patient. Detailed echocardiography and ventriculography revealed various abnormalities characteristic for ARVC. Late potentials were positive in 6 of 8 patients. Three patients were taking sotalol, one patient bisoprolol and another one amiodaron at the time of body surface mapping. Patients with right bundle branch block were excluded from the study.

Characteristics of patients with ARVC are described in Table 1.

Control group

There were 5 men and 3 women (mean age at the time of body surface mapping 33 years, range 14–48 years) presented with episodes of orthodromic atrioventricular reentry tachycardia. In the time between episodes there is no electrocardiographic evidence suggesting a presence of a concealed AV accessory pathway. Patients were mapping before radiofrequent catheter ablation of the underlying accessory pathway. One patient from the group had a small amount of pericardial fluid as a consequence of pericarditis. Patients were receiving no therapy during the time of body surface mapping.

Body surface mapping

Body surface mapping was performed during sinus rhythm with a portable computerized mapping system. A 63-lead Savard's system was used. Electrodes were placed on torso separately with upper border crossing jugulum and lower border matching diaphragma, thus representing a lower border of thorax. Position of electrodes is shown in Figure 1.

Wilson's central terminal was used as a reference for measuring chest potentials. Digitalized data were transmitted to the microcomputer for processing and analysis. Baseline correction was performed automatically with possible manual correction. QRS onset, QRS offset and T-wave offset were determined by manual selection.

QRST integral maps were obtained by calculating the sum of all potentials from QRS onset to T-wave offset.

Individual maps of ARVC patients and control group were visually analyzed, focusing on location and numbers of the extremes and the morphology of the zero line. The mean QRST map of patients with ARVC and control group was calculated.

The QRST integral map of each ARVC patient and the mean QRST integral map of patients with ARVC were subtracted from the mean QRST integral map of the control group. To construct departure maps, mean (m) and standard deviation (SD) was calculated for each lead to a time-integral value. For a measured value (x) of each lead, departure index was calculated as (x-m)/SD. The departure maps demonstrated body surface distribution of the departure index. Areas of departure index >2 or <2 were considered as abnormal.

Signal averaging ECG

For the signal averaging ECG recordings the portable bedside measuring unit ART 1200 EPX (Arrhythmia Research Technology) was used. The recordings were performed using three orthogonal leads X, Y and Z. The signal was filtered, averaged and combined into vector sum called the "vector magnitude", or the filtered QRS complex $/X^2+Y^2+Z^2/^{1/2}$. At least 300 cycles were averaged in order to achieve as much noise reduction as possible (<0.7 mV). Acquired data were analyzed by time-domain analysis in two filtration bands: 25–250 Hz and 40–250 Hz. The results were evaluated using Simson's criteria (8) for the detection of ventricular late potentials.

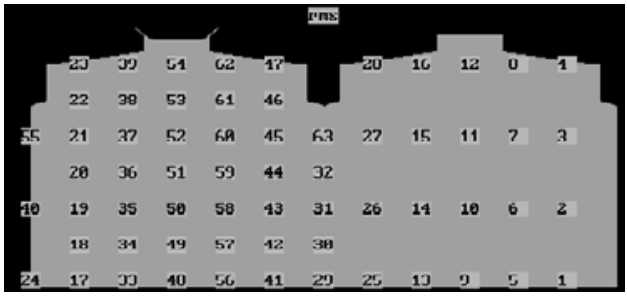

Fig. 1. 63-lead Savard's system (position of electrodes). Left side represents chest and right side represents back.

Fig. 2. Mean QRST integral map of the control group. Positions of extremes are indicated by minus and plus signs.

Fig. 3. Mean QRST integral map of patients with ARVC. Positions of extremes are indicated by minus and plus signs.

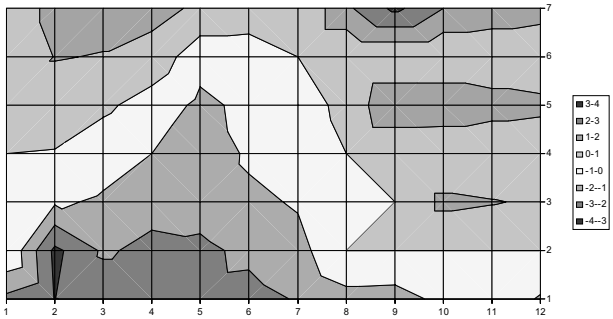

Fig. 4. Departure map of a mean QRST integral map of patients with ARVC. Areas with departure index <2 and >2 are considered abnormal.

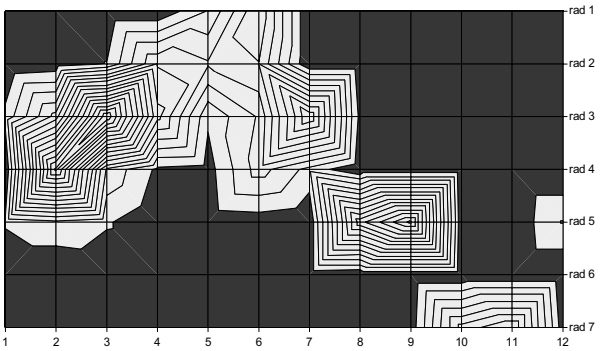

Fig. 5. Mann—Whitney U test for non-parametric variables. Areas with p<0.05 are marked dark.

Statistical analysis

The mean QRST integral map of patients with ARVC was compared to the mean QRST integral map of control group using Mann–Whitney U test for non-parametric variables. p-values less than 0.05 were considered significant.

Results

Analysis of body surface QRST integral maps

Control group

In all control subjects a smooth dipolar map pattern was found with the positive values located over the precordium and negative values over the right chest and back (Fig. 2), as described by Montague (9). In two patients with a characteristic smooth dipolar pattern was the minimum localized over the right back instead over the right chest.

Patients with ARVD

Mean QRST integral map of patients with ARVC showed abnormal characteristics. The area of negativity was larger than normal and extended to lower border of thorax (Fig. 3).

In all patients except one, individual departure maps showed areas with departure index <2 and >2, considered as abnormal. Departure map of the mean QRST integral map of patients with ARVC showed areas with departure index <2 and >2 in lower part of chest and upper part of back (Fig. 4).

Signal averaging ECG

Late potentials were considered present if at least two of the three Simson's criteria were positive. Positive late potentials were seen in 6 of 8 (75 %) patients with ARVC.

Statistical analysis

p-value <0.05 was calculated in 38 leads and p<0,01 was calculated in 27 leads. Areas with p<0.05 covered nearly lower half of chest and upper half of back (Fig. 5).

Discussion

Both etiology and pathogenesis of ARVC are still unknown. As proposed by Fontaine (10), ventricular arrhythmias in ARVC eventually leading to sudden cardiac death might be caused by two different mechanisms: depolarization abnormalities mediated by sympathetic nervous system during sports activities or repolarization abnormalities facilitated by parasympathetic drive during rest or sleep.

We used BSM to examine primary ventricular recovery properties. QRST integral maps reflect local disparities of ventricular repolarization and resulting vulnerability to arrhythmias (4). In our study, a characteristic pattern in QRST integral maps was found in all ARVC patients. QRST integral maps of our patients were similar to maps previously published by Peeters (11) and DeAmbroggi (12) with respect to the differences in lower border of electrodes' placement.

Departure maps represent the location and extent of abnormality in given map compared to pattern (13). Departure map of the mean QRST integral map of patients with ARVC showed areas with departure index <2 and >2 in lower part of chest and upper part of back. When statistically analyzed, areas with p <0,05 covered nearly lower half of chest and upper half of back, indicating a clear difference in patterns.

It should be stressed, that repolarization abnormalities resulting from structural changes in regions facing anterior side of the thorax might be more visible in QRST integral mapping. We consider body surface QRST integral mapping to be an adequate method for evaluation of dispersion of myocardial repolarization in ARVC patients.

The possible influence of antiarrhytmics on QRST integral maps needs to be elucidated. Couderc (14) states, sotalol (known

to produce QT prolongation) induced changes in repolarization interval identified by morphological changes of the T wave. The results of signal averaging ECG (positive late potentials in 75% of patients) were similar to those published by Yoshioka (15) 85 % and De Ambroggi (16) 60 %.

More information concerning pathophysiology and clinical course of ARVC is needed, therefore an international ARVC registry has been established (7) and a multidisciplinary study of ARVC was initiated (17) with the aim to establish a North American ARVC Registry.

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