INDICES OF VULNERABILITY TO ARRHYTHMIAS IN ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA

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The aim of the present study was to assess in patients with ARVD whether signs of heterogeneities of ventricular repolarization, most likely related to arrhythmias vulnerability, can be revealed by specific analysis of surface potential mapping. We studied 22 patients affected by ARVD, 9 of them with episodes of sustained ventricular tachycardia (VT), and 35 normal subjects. By applying principal component analysis of the ST-T waves we computed the similarity index (SI), which was significantly lower in ARVD patients with VT than in controls. The instantaneous variations of the surface potentials distribution during repolarization were quantified by computing the Pearson correlation coefficient between each instantaneous map and the map at the peak of the T wave. The early repolarization deviation index (ERDI) was significantly higher in ARVD patients with VT than in normals. Thus, in ARVD patients a low value of SI and a high value of ERDI were significantly associated with the occurrence of VT.

1. INTRODUCTION

Patients with arrhythmogenic right ventricular dysplasia (ARVD) have different risk for ventricular arrhythmias, which is difficult to determine because the predictive value of several variables (ECG characteristics, ventricular late potentials, QT dispersion) used in clinical practice has been found relatively low.

In a previous paper[1] we reported the characteristics of body surface potential maps in patients with ARVD and we found that principal component analysis of the ST-T waves revealed abnormalities, most likely due to repolarization heterogeneity, not detected by conventional ECG, that were associated to arrhythmias vulnerability.

The aim of the present study was to assess in patients with ARVD whether other signs of heterogeneities of ventricular repolarization can be revealed by particular analysis of surface potential mapping.

2. METHODS

Body surface potential maps were recorded from 62 chest leads in 22 patients affected by ARVD 15 males, 7 females, aged 18 to 66 (42±13 years). The diagnosis of ARVD was based on electrocardiographic, echocardiographic and cardiac magnetic resonance findings, according to the recommendations of the Task Force of the European Society of Cardiology[2]. Patients with right bundle branch block (QRS duration >120 ms) were excluded. Nine patients had experienced episodes of sustained ventricular tachycardia (VT), whereas 13 presented only frequent premature ventricular beats or short runs of nonsustained VT (4 cases) with left bundle branch block pattern of QRS. No patient had symptoms or signs of right ventricular failure. Thirty-five healthy subjects, 26 males and 9 females, aged 14 to 54 years (mean 33±10), were also studied as controls.

2.1. Principal component analysis

In each subject the 62 ST-T waves were resampled at 20 ms, resulting in N=(STT duration)/20ms samples for each ST-T interval. For each recording we performed a principal component analysis of the matrix with N variables (sets of potentials at each of the resampling points) of 62 values. The result was a set of N eigenvectors and N eigenvalues for each recording.

The amount of the body surface potential variation described by the first principal component is expressed as the first eigenvalue. The extent to which, after substracting the first principal component, the signal is described by the second principal component, is expressed by the second eigenvalue, and so on[3].

We defined the "similarity index" (SI) as the first eigenvalue divided by the sum of all eigenvalues[1,4]. The SI is inversely proportional to the extent of the variability among the morphologies of the T wave, and a low value is considered a marker of repolarization heterogeneity.

The same analysis was repeated in a subset of 12 leads from the set of 62 available. The 12 leads used roughly correspond to: V_{1-6},V_8, V_{3R}, V_{4R}, VR, VL, VF.

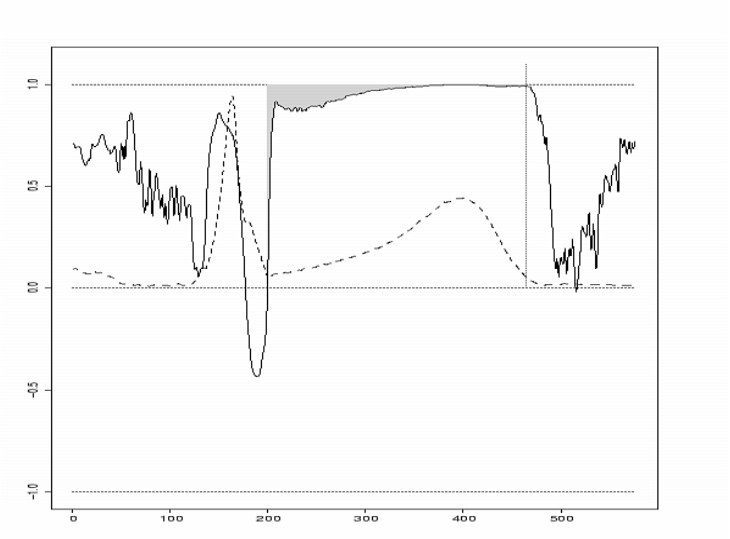

Figure 1: The ICPTM signal. The mean amplitude of the grey area between the ICPTM and 1.0 before the peak of the T wave is the ERDI. The mean area after the peak of the T wave is the LRDI. The dashed line represents the root mean sqare signal of the 62 lead ECG.

2.2. Early and Late Repolarization Deviation Index

At visual inspection the distribution of the potentials on the body surface during repolarization is generally constant in time, apart from changes in amplitude[5]. In other words, during repolarization the amplitude of the potentials on the surface is increasing and decreasing overall, but the relative amplitudes on one lead with respect to another are generally unchanged. The early repolarization deviation index (ERDI) and late repolarization deviation index (LRDI) are numerical indices which describe deviations from this behaviour during repolarization, from the J point to the T peak and from the peak to the end of T wave, respectively (Fig. 1).

The potential distribution at the peak of the T wave (peak-T map) was taken to represent the most well defined distribution of the repolarization potentials. For each instant during the cardiac cycle we computed the Pearson correlation coefficient between the potentials of each lead at that instant and the potentials of the same leads at the peak-T map. This value is 1.0 if at that instant the map has identical distribution with the peak-T map (apart

Table 1: Values of indices in ARVD patients with or without ventricular tachycardia. * p = 0.03 ** p = 0.01

	Controls	ARVD without VT	ARVD with VT
SI62	77 ± 4 %	76 ± 6 %	67 ± 12 *
SI12	81 ± 5 %	80 ± 5 %	73 ± 10 *
ERDI62	.21 ±.24	.25 ±.19	.40 ±.30 **
ERDI12	.20 ±.23	.21 ±.15	.40 ±.36 **
LRDI62	.029 ±.016	.025 ±.015	.029 ±.018
LRDI12	.023 ±.020	.026±.025	.023 ±.016

from the amplitude), -1.0 when it is exactly opposite, and 0.0 when it shows no resemblance. Subunit values measure the extent of resemblance between the instantaneous map and the peak-T map. We called this function of time quantity the "instantaneous correlation with the peak-T map" (ICPTM).

3. RESULTS

The SI was not significantly different in ARVD patients compared to normals (Table 1). Nevertheless, considering the 2 subgroups of patients with and without VT, SI was significantly lower in patients with VT than in controls (0.67±0.12 vs 0.77±0.04 , p=0.032) We performed the same analysis in a subset of 12 unipolar leads (V1-V8, V3R, VR, VL, VF) extracted from the map. In this case too, SI was significantly lower in VT patients than in normals (0.73±0.1 vs 0.81±0.05, p=0.038).

LRDI (computed from 62 and from 12 leads) was not significantly different in the ARVD patients with VT and in normals, whereas the ERDI was significantly higher in ARVD patients with VT than in control subjects (0.40±0.10 vs 0.21±0.04, p=0.011).

4. CONCLUSIONS

In ARVD patients a low value of SI and a high value of ERDI are significantly associated with the occurrence of VT. The high value of ERDI also suggests that the abnormalities (heterogeneities) of ventricular repolarization mainly occur during the first part of repolarization.

Since we found that both SI and ERDI maintained their statistical significance even when calculated on 12 leads derived from our map lead system, it is likely that the two indices could be also computed from digital recordings of the 12 conventional ECG leads. This could facilitate the validation of the proposed indices in vast population of normal subjects and patients with and without ventricular arrhythmias.

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