Susceptibility for ventricular tachycardia and the correlation between depolarization and orthogonal components of repolarization

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ABSTRACT

Objective: There is a continuing need of methods to identify subgroups of patients at high risk of ventricular arrhythmias, in particular after myocardial infarction (MI).

Methods: We performed a singular value decomposition of repolarization potentials in individual recordings in 134 healthy males, in 203 males with old MI and without documented sustained ventricular tachycardia (VT) and in 104 MI males with documented VT. We considered the absolute correlation coefficient between the first orthogonal component, constructed by matrix multiplication of the first left and right singular vectors and the QRS integral (RT1) and a similar index for the second component (RT2).

Results: Abnormally high (more than two standard deviations above the mean) value of the RT1 had a 89% specificity for VT in MI patients. Abnormally low RT2 had specificity of 87%. Both indices combined had a 97% specificity. However, sensitivity of the combined indices was only 13%.

Conclusion: Abnormalities in the correlation of orthogonal components of repolarization with depolarization are highly specific for a small group of patients with old myocardial infarction at high risk of ventricular tachycardia. *(Anadolu Kardiyol Derg 2007: 7 Suppl 1; 139-41)*

Key words: ventricular tachycardia, repolarization, depolarization, orthogonal component of repolarization

Introduction

The implantable cardioverter defibrillator (ICD) trials have highlighted the need for methods with better specificity for patients at the highest risk of ventricular arrhythmias, who would mostly benefit from an ICD (1).

Even specific arrhythmias, such as ventricular tachycardia (VT) can be produced through a variety of mechanisms (2), thus most arrhythmogenic markers may only select a small fraction of the patients at risk. Progress in this field may turn out to consist of numerous markers, obtained with a variety of investigational methods, each of them specific for a limited subgroup of patients.

A pathological substrate associated with arrhythmogenicity is abnormally heterogenous ventricular repolarization. As the relative distribution of potentials on the body surface during repolarization is largely constant in time, changing mostly in the general amplitude, subtle abnormalities of repolarization, as deviations from this normal feature (7), have been described using a singular value decomposition of the series of instantaneous repolarization potentials over the ST-T interval in a single cardiac cycle.

In normal subjects, the first component corresponds to the general repolarization pattern. An abnormal reduction in its contribution to repolarization potentials, measured by the magnitude of the first singular value relative to the sum of singular values, has been associated with arrhythmogenicity in a variety of pathological contexts (3-5).

However, even when a major component is present, it might reflect a different electrophysiological phenomenon in different individuals. This might lead to a reduction of specificity.

We are trying to better understand this phenomenon and perhaps find ways to avoid the loss of specificity in indices based on singular value decomposition of repolarization potentials in individual recording. To this end, we reconstructed individual components by multiplying the left, right singular vector and the singular value of each component. Thus, we obtained orthogonal components, the sum of which, lead by lead and sample by sample correspond to the original electrocardiogram (ECG). Orthogonal components have each a strictly constant relative distribution of body surface potentials, that only changes in amplitude over the STT, and the relative distributions of any two components are orthogonal.

We then examined these signals, and in particular their relationship with depolarization potentials. We found that even in normal subjects there is considerable variability, for example in some subjects the second component being almost identical to depolarization pattern, while in others the first component is more like depolarization. There is a continuous spectrum of cases between the two and there is also a substantial proportion of cases (20%) that is outside of this spectrum (6).

In this study, we attempted to further explore the relationship of depolarization to orthogonal components of repolarization in

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patients with previous MI, with and without recent history of sustained ventricular tachycardia on Holter recordings.

Methods

We used ECG body surface map recordings, obtained at the Dalhousie University, in 134 healthy males and in 307 males with MI, of whom 104 had a recent history of sustained VT and electrophysiological study of inducibility of VT. We used a single cardiac cycle, averaged from 15-second recordings.

After singular value decomposition of the leads x time matrix of ST-T potentials in each recording we multiplied the set of the first left and right vectors and singular value to obtain the first orthogonal component (OC) T1, and the second set for T2. Orthogonal components are leads x time matrices of volt values like the ECG.

We noted as RT1 and RT2 the absolute values of the correlation coefficients between the QRS integral and T1 and T2 respectively.

Results

The first orthogonal component of repolarization was in general more closely correlated with depolarization potentials in MI patients with VT compared with MI patients without VT (Table 1). The correlation of the second component with the QRS was significantly lower in VT patients compared with the non-VT group.

Recordings in MI patients were mostly concentrated in an arc in the RT1/RT2 plane, that extended at a distance from 0.8 to 1.0 from the origin (Fig. 1), a feature shared with the distribution of recordings in healthy subjects, as we have shown in a previous study (6). Despite the significant differences mentioned above, there was a substantial overlap of the distribution of recordings of MI+VT, MI-VT patients and the healthy, except for the region of abnormally high RT1 and low

RT2 (lower right corner on the Figure 1) where some MI+VT cases appear concentrated.

In order to estimate the diagnostic power of these abnormalities we chose the lowest 2.5 centile of RT2 (0.17) and highest 2.5 centile of RT1 (0.86) from the healthy males group as normal limits and used them as cutoff values. Either or both identified arrhythmia susceptibility with a high specificity (Table 2).
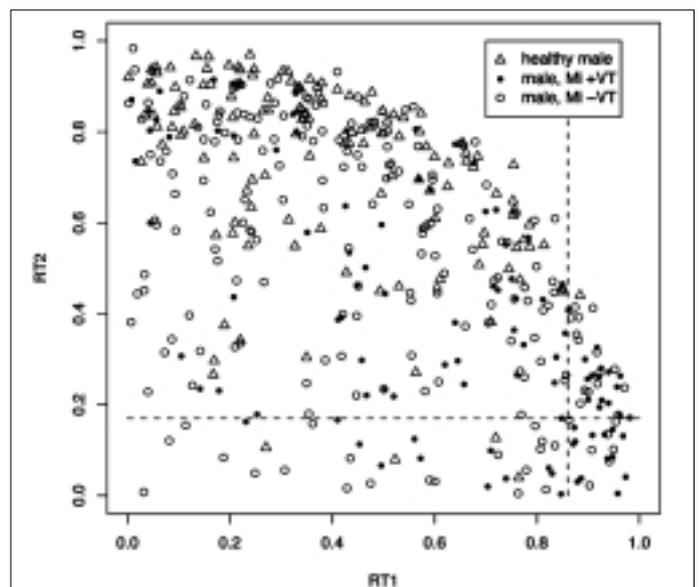
Discussion

In this study we found that there is a substantial proportion of the post-MI patients with recent VT history for whom the first orthogonal component of repolarization potentials is highly correlated with the QRS integral while the second orthogonal component is uncorrelated.

Our MI+VT and MI-VT samples have been separately drawn from their respective populations. Application of these indices in other populations will result in different predictive values, depending on the a priori prevalence of the MI+VT cases. A lower prevalence (8) would result in lower positive and higher negative predictive value. Thus, there is substantial scope for supplementary reduction of false negatives. Further elucidation of the mechanism underlying the normal and pathological variability of the orthogonal components will be necessary for this purpose.

A limitation of our study is the lack of assessment of the independence of these indices from other indices of arrhythmogenic risk (such as heart rate variability or ejection fraction of the left ventricle) which was due to lack of data.

Conclusions

Abnormalities in the correlation of orthogonal components of repolarization with depolarization are highly specific for a small group of patients with old myocardial infarction at high risk of ventricular tachycardia.

Table 1. Absolute correlation coefficients, lead by lead, between integrals of each of the first two orthogonal components of repolarization (T1, T2) and the QRS integral

	Healthy	MI+VT	MI-VT	p (MI+VT vs MI-VT)
N	134	104	203	-
RT1	0.40±0.23	0.61±0.29	0.47±0.28	<0.0001
RT2	0.72±0.20	0.39±0.27	0.53±0.27	<0.0001

Data are presented as Mean±standard deviation
MI- myocardial infarction, RT- orthogonal component of repolarization and the QRS integral, VT- ventricular tachycardia

Table 2. Specificity, sensitivity, positive and negative predictive values of RT1 and RT2 beyond normal 95% confidence interval limits, for associated sustained ventricular tachycardia in males with old myocardial infarction

	Specificity	Sensitivity	PPV	NPV
RT1>0.86 MI +VT vs VT	89%	28%	57%	71%
RT2<0.17 MI +VT vs -VT	87%	25%	49%	69%
RT1>0.86 and RT2<0.17	97%	13%	68%	65%

MI- myocardial infarction, NPV- negative predictive value, PPV- positive predictive value, RT- orthogonal component of repolarization and the QRS integral, VT- ventricular tachycardia

Figure 1. Distribution of the correlation between the first orthogonal component of repolarization and the QRS integral (RT1) and the second orthogonal component and the QRS integral (RT2) in healthy males and males with myocardial infarction with and without history of ventricular tachycardia

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