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Modulation of spatial variance of ventricular repolarization after myocardial infarction

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ABSTRACT

Background: Myocardial infarction (MI) alters spatial features of the surface electrocardiogram. Spatial variance of the T-wave (SV_T) describes the interlead dispersion about a mean T-wave morphology. SV_T was linked to arrhythmia vulnerability and sudden cardiac death.
Methods: Herein, we studied the evolution of SV_T over the healing (MI_7, up to 7 days after MI) and healed (MI_{60}, from 60 days on post-MI) stages. A control group ($n = 49$) was compared to paired MI_7 and MI_{60} groups ($n = 39$). Five representative sets of frontal and precordial leads were analyzed: $I–II–III$, $V_1 – V_2 – V_3$, $V_4 – V_5 – V_6$, $aV_F – V_2 – V_5$ and $II – aV_F – V_5$.
Results: SV_T index significantly increased at MI_7 ($p < 0.05$) in four out of five sets and returned towards control values at MI_{60} (p = NS). The *preferential combination of ECG leads* resulted $aV_F – V_2 – V_5$, since it showed the strongest modulation. In order to test whether such a modulation was maintained on the presence of ventricular tachycardia and/or ventricular fibrillation (VT/VF), recordings from VT/VF patients were also analyzed. SV_T modulation was lost in the VT/VF group, significantly increasing from controls at MI_{60} ($p < 0.05$) for all sets of leads tested.
Conclusions: SV_T modulation over MI_7 and MI_{60} would signal a good recovery from MI, whereas lack of this modulation could herald a VT/VF event.

1. Introduction

It is well described the cascade of changes in membrane properties that myocardial infarction (MI) induces across the entire heart. More specifically, it can be observed a reduction of peak L-type inward Ca^{2+} in myocites from the epicardial border zone (EBZ) [1] as well as a delayed recovery of the transient inward Na^+ current [2]. Furthermore, myocardium outside the EBZ also presents changes with infarction, such as a prolongation of action potential durations (APDs) during chronic MI [3] or a reduction of transient outward K^+ currents [2]. Besides the changes in ionic currents, proliferation of connective tissue and edema contribute to the nonuniform anisotropy. As a result, it is expected that ECG morphology is altered. More specifically, the interlead morphology differences appearing simultaneously in one beat are expected to change from health to disease. Second central moment analysis, also known as variance, helps quantitate the interlead heterogeneity (or spatial variance) of the entire T-wave morphology recorded from multiple ECG leads. In this regard, many reports focussed on the spatial variance of repolarization in experimental ischemia [4], heart failure [5], MI patients [6] or the general population [7]. Certainly, all these changes are not static, but evolve with time. Pinto and Boyden reviewed the changes in infarcted and non-infarcted cells occurring at different times following MI [8]. During the first week post-MI, so-called healing phase, there exists a shortening and shrinking of action potentials [9], while they shift to prolonged APDs with normal voltages in the healed phase, two months after MI [8]. Moreover, it is during the healing phase that increased heterogeneity of the time course of repolarization in the EBZ is origin of inducible ventricular tachycardia (VT) and ventricular fibrillation (VF) [2].

The hypothesis behind this work, is that these two distinctive stages would modulate the spatial variance of repolarization, which in turn is associated to the cardiac risk that one MI patient is exposed to. This modulation would be reflected on different electrocardiographic markers, such as the T-wave spectral variance, as recently described by Arini and Valverde [10]. Therefore, our

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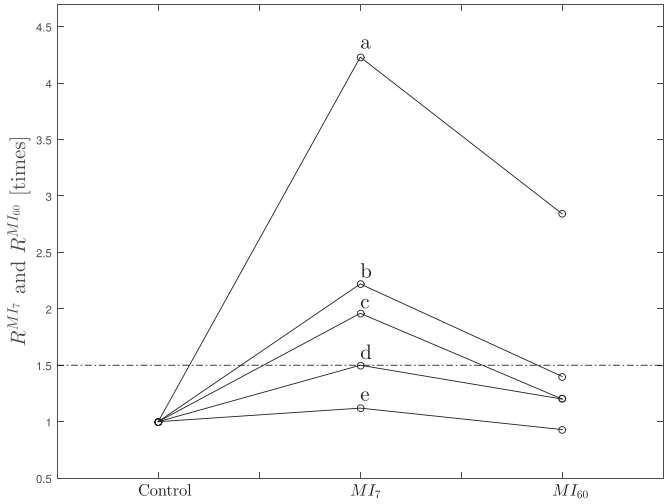

Fig. 1. Relative changes $\mathcal{R}^{MI_7}$ and $\mathcal{R}^{MI_{60}}$ in different sets of leads for control, MI_7 and MI_{60} groups. (a) $V_4 - V_5 - V_6$, (b) $II - aV_F - V_5$, (c) $aV_F - V_2 - V_5$, (d) I–II–III, (e) $V_1 - V_2 - V_3$. Dotted line signals 50% increase with respect to controls. Notice the tendency towards control values at MI_{60} for all the sets.

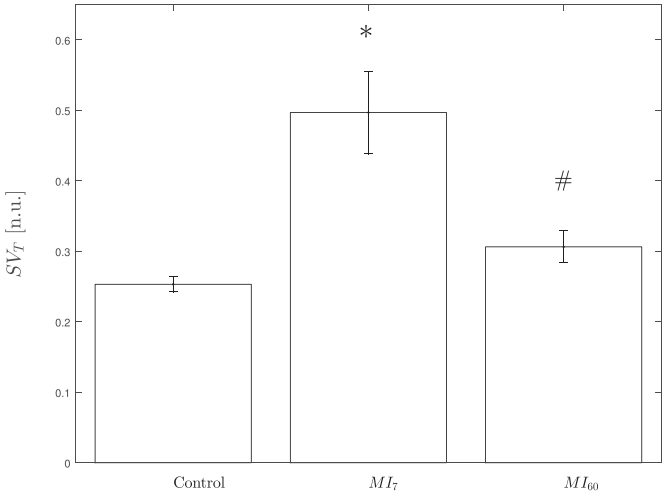

Fig. 2. Mean (SEM) values for SV_T of the *preferential combination of leads* $aV_F - V_2 - V_5$, for control, MI_7 and MI_{60} groups. Notice the tendency towards control values at MI_{60}. *MI_7 vs Control, $p < 0.05$, #MI_7 vs MI_{60}, $p < 0.05$.

main goal is to study the modulation of the spatial variance of the T-wave associated to time elapsed after MI. More specifically, during the healing and healed phases following myocardial infarction. Finally, assess if this modulation is maintained in MI patients showing VT/VF events.

2. Materials and methods

2.1. Database

We have used the Physikalisch-Technische Bundesanstalt (PTB) ECG dataset which is available free on the Physio-Bank [11]. This database comprises 52 healthly subjects and 148 MI patients. The ECGs were digitized at 1000 samples per second, with 16 bit resolution over a range of ±16.384 mV with 2000 A/D units per mV. The recordings are in average 1.35 min long and include 12 standard ECG leads.

We have chosen the following subsets of data, according to the detailed clinical summary included in the PTB dataset [12]: the ECG of healthy subjects (control), $n = 49$ (37 males and 12 females, 43 ± 14 years old), and those infarcted patients without documented ventricular tachycardia (VT) and/or ventricular fibrillation (VF), which simultaneously comprised two ECGs recordings, $n = 39$ (31 males and 8 females, 53 ± 5 years old): one record within the first seven days of MI (MI_7, healing phase), and the other 60 days after MI (MI_{60}, healed phase). In order to asses differences in MI remodeling with occurrence of VT or VF, twenty recordings (ten at MI_7 and ten at MI_{60}) from six patients (3 males and 3 females, 56 ± 2 years old) who underwent such events and presented ECG recordings at both post-MI stages formed the VT/VF counterparts: MI_7^{TF} for the healing phase and MI_{60}^{TF} for the healed phase.

None of the subjects studied had showed bundle branch block or intra-ventricular conduction defects. The QRS durations for healthy subjects were comparable with MI patients. The ECG recordings have been analyzed anonymously, using publicly available secondary data, therefore no ethics statement is required for this investigation [11].

2.2. ECG preprocessing

A set L of five representative combinations of leads were analyzed: $L = \{$I-II-III, $V_1 - V_2 - V_3$, $V_4 - V_5 - V_6$, $aV_F - V_2 - V_5$ and II-$aV_F - V_5\}$. ECG was filtered with a notch filter (Butterworth, 8th order, 50 Hz) to minimize the power-line interference and a cubic spline interpolation filter was used to attenuate ECG baseline drifts and respiratory artifacts [13]. QRS-complexes were detected by means of the wavelet-transform delineator presented in [14]. For every lead $l_i \in L$, a start-up QRS template was built up with the ten first beats and then a new jitter-corrected QRS template was obtained when the cross-correlation coefficient between every new QRS complex and the QRS template was greater than 98%, otherwise the complex was rejected.

2.3. Spatial variance of ventricular repolarization

The spatial variance of the T-wave morphology (SV_T) was calculated using several interlead combinations. These leads were selected from 12 standard ECG leads according to the following criterion: bipolar frontal leads, right and left precordial leads and combinations of frontal and precordial leads. The method to compute spatial variance was modified from Nearing et al. [4], and consists in measuring the splay of waveforms around a mean waveform in a certain set of leads. Briefly, sinusal QRS-complexes were identified and a unique R-wave fiducial point was obtained as the median of the all R-wave marks. Then, for every *jth* beat, a segmentation window W_j^T from 60 to 290 ms after the R-wave occurence was defined, so that the repolarization process was fully covered. After that, T-wave amplitude was normalized and isoelectric level was made uniform for every lead $l_i \in L$, and T-waves of all sinusal beats were superimposed along the ensamble. The T-wave interlead average was accomplished as follows,

$$\bar{T}(n) = \frac{1}{N}\sum_{i=1}^{N} T_i(n) \tag{1}$$

where $T_i(n)$ was the amplitude at sample n $\in W_j^T$, of every jth T-wave in $l_i \in L$, with $N = 3$ leads. After that, the interlead variance was computed along the ensamble as in Eq. (2):

$$\sigma_T(n) = \sqrt{\frac{1}{N}\sum_{i=1}^{N}[T_i(n) - \bar{T}(n)]^2} \tag{2}$$