

ECG-based characterization of the extent, severity and spatial location of acute ischemia in patients with and without prior myocardial infarction

Jimena Rodríguez-Carbó¹, Ana Mincholé^{1 2}, Esther Pueyo^{1 2}

¹ I3A, Universidad de Zaragoza, Zaragoza, Spain

² CIBER de Bioingeniería, Biomateriales y Nanomedicina, Instituto de Salud Carlos III, Spain

Abstract

Ischemic heart disease is the leading cause of death in the world. Its diagnosis involves monitoring of the dynamic changes in the ST segment of the ECG, although changes in other intervals and waves of the ECG have been observed. One important aspect that is usually not considered is the presence of a prior myocardial infarction (prior MI) and how this affects the response to an ischemic event. Our aim was to quantify the changes in the ECG during acute myocardial ischemia and the effects a prior MI has on them. 12-lead ECG recordings acquired during and prior the acute ischemia induced by percutaneous coronary intervention (PCI) in one of the three main coronary arteries were analyzed. Averaged heartbeats were computed and ECG depolarization and repolarization features were extracted. Our results show that, in addition to ST deviation, there is a temporal evolution in other ECG parameters during artery occlusion. Repolarization parameters show a faster and stronger change than depolarization ones. The relationship between the extent and severity of the ischemia and the ECG changes was more pronounced in patients with prior MI. A spatial lead profile was described as a function of the occluded artery and the presence of a prior MI. In conclusion, the presence of a prior MI affects the myocardial response to acute ischemia, resulting in more pronounced changes and a stronger relationship with the extension and severity of the ischemia.

1. Introduction

Cardiovascular diseases (CVDs) are the leading cause of death in both men and women. Of all CVDs, ischemic heart disease is the one associated with the highest number of deaths, which is almost three fold higher than those due to the second one, this being ischemic stroke [1].

Ischemia is defined by a reduction in the blood flow as a result of a partial or complete blockage of the heart's arteries (coronary arteries), usually produced by thrombosis or other acute alterations of coronary atherosclerotic plaques

[2]. Prolonged and sustained ischemia leads to cardiomyocyte death and myocardial infarction [3]. A timely diagnosis is crucial to achieve an appropriate treatment, including percutaneous coronary intervention (PCI). The diagnosis is based on dynamic changes in the electrocardiogram (ECG) [4]. The most common manifestation of acute coronary artery disease is ST-segment deviation, with larger deviation being associated with a greater degree of myocardial ischemia and morbidity and mortality. Other alterations in the ECG have been observed during acute ischemia, such as a reduction in the QRS downward slope [5] and an increase in the T wave amplitude [6].

Factors that affect the progression of ischemia can include the location of the occluded artery [6, 7], the fact that the occlusion is proximal or distal [3] and the extent and severity of the ischemia [8]. Another factor that, although not always considered, can also affect the temporal evolution of ECG manifestations in patients with acute ischemia is the presence of a prior myocardial infarction (MI) [9]. Our hypothesis is that a prior MI in the same zone as the current acute ischemia can enhance the ECG dynamic changes.

The main aims of this study are: to quantify the temporal evolution of ECG markers different from ST segment deviation and the effect that a prior MI has on such evolution; to quantify the relationship between the changes in the ECG during ischemia and the extent and severity of the occlusion in patients with and without prior MI; and to describe a spatial lead profile as a function of the occluded artery and accounting for the effects of a prior MI.

2. Methods

2.1. Study Population

This study analyzed the STAFF III database [10], which consists of 102 patients (mean age of 60.7 ± 11.52 years) receiving elective PCI in one of the major coronary arteries: left circumflex artery (LCx), right coronary artery (RCA) and left anterior descending artery (LAD). The distribution of the occlusion in the patients was: 21 with LCx, 47 with

RCA and 34 with LAD. Even though several patients had more than one occlusion during the procedure, we only analyzed the first balloon inflation. The duration of the balloon inflation in the PCI procedure ranged from 1.1 to 9.92 min with a mean of 4.4 ± 1.32 min. 34 of the 102 patients had a prior MI, most of them in the same zone as the occluded artery during the PCI procedure. 12-lead ECG recordings were obtained before (control recording, CR) and during (PCI recording, PCIR) the medical procedure. Standard electrode placements were used for the precordial ECG leads but the limbs leads were obtained with a Mason-Likar configuration. All the recordings were digitized at a sampling rate of 1000 Hz. 35 of the 102 patients had scintigraphic quantification using tomographic imaging and ^{99m}Tc -sestamibi as the contrast agent to evaluate the extension and severity (global reduction) of the occlusion [11].

2.2. ECG Processing

Preprocessing of the ECG signals included filtering, QRS detection [12], and ensemble (for CR) or exponential averaging (for PCIR) [5]. For both types of recordings (CR and PCIR), the filtering was performed with a band-pass filter (0.5-40 Hz). After the filtering and with the fiducial points, in each recording, the heartbeats were segmented and aligned. Beats presenting a correlation coefficient with an initially computed median heartbeat below 0.95 were removed from the analysis.

For CR, ensemble averaging was applied to non-overlapping blocks of 10 s and the obtained averaged heartbeats were used for ECG characterization. For PCIR, the beats were first separated in 10-second windows. For each window, the median heartbeat was computed and used to select the beats within the window presenting a correlation above 0.90. Considering all the selected beats in the PCIR recording, exponential averaging was used to follow the dynamic changes during the occlusion:

$$\bar{x}_i(n) = (1 - \alpha)\bar{x}_{i-1}(n) + \alpha x_i(n),$$

where $\alpha = 0.25$, i is the beat index, $x_i(n)$ is the current beat and $\bar{x}_i(n)$ is the averaged beat. The last beat within each 10-second window of the PCIR was considered for ECG characterization.

2.3. ECG markers

The averaged beats along the CR and PCIR recordings were delineated [10] to obtain the beginning, peaks and ends of the ECG waveforms. From the delineation marks, we computed the QRS width, QRS peak-to-peak amplitude, T amplitude, T area, ST deviation and the ratio T amplitude/QRS amplitude. To track the ischemia-induced

changes during PCIR and quantify the amount of change with respect to the normal variations during CR, we calculated the performance index $|R_\iota(t_j)|$ [5] for each marker in each of the 12 leads. $R_\iota(t_j)$ was defined as:

$$R_\iota(t_j) = \frac{\Delta_\iota(t_j)}{\sigma_\iota}, \quad \Delta_\iota(t_j) = \gamma_j t_j$$

Δ_ι was computed in the PCIR by fitting a linear polynomial to the values of the parameter (ι) from the onset of the occlusion ($t = 0$) until each time $t = t_j$. Here, t_j was taken every 10 seconds from the beginning of the occlusion. γ_j was computed as the slope of the fitted polynomial. The denominator σ_ι was calculated as the standard deviation of the marker ι during CR.

To evaluate the global change in each ECG marker ι throughout all the leads, the mean value of R_ι over leads was obtained at each time t_j .

To quantify the relationship between the ischemia extension and severity and the change in each ECG marker ι , we conducted a Pearson correlation analysis between the extension (severity, respectively) and $|R_\iota|$ computed 60 seconds after the start of the occlusion, as the shortest occlusion time in the study population was 69 seconds.

3. Results and Discussion

3.1. Temporal Evolution of ECG changes

Figure 1 shows the temporal evolution of the analyzed ECG markers during the first 4 min of coronary occlusion, separately for patients with and without prior MI. The depolarization parameters showed slower changes and of a lesser magnitude in both prior and non-prior MI patients. In prior MI patients, evaluation at 240 seconds of occlusion showed that the T wave amplitude increased 10.8 times its standard deviation during CR, while the QRS amplitude increased 5.9 times. The ST deviation showed the largest magnitude of change, particularly in patients with prior MI, where it changed more than 20 times its normal variations during control.

While the ST deviation showed clear differences in its magnitude of change and temporal evolution during PCIR between prior and non-prior MI patients, the other analyzed ECG markers showed changes of comparable magnitude in the two groups.

3.2. Relationship between ECG Changes and Ischemia Extension/Severity

We evaluated the correlation between the extension and severity of the occlusion with the change observed in the analyzed ECG markers during the first minute of occlusion to include all possible patients in this analysis. Figure 2

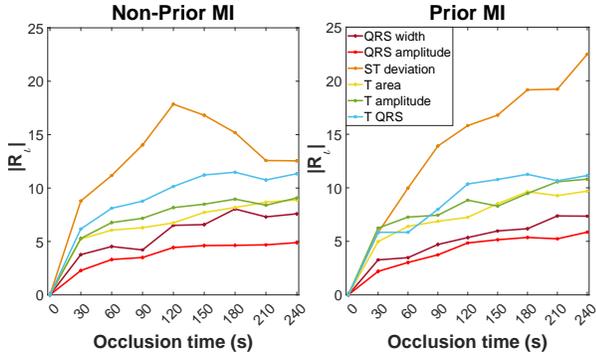


Figure 1. Median values of $|R_i|$ over patients, computed from the 12 analyzed leads, every 30 seconds from the start of the occlusion.

shows the dispersion plots between the extension percentage and the $|R_i|$ index for the T amplitude and for the T amplitude/QRS amplitude, separately in patients with and without prior MI. A higher correlation between the extension of the ischemia and the induced ECG changes, and a greater magnitude of these changes for the same level of extension, was observed in patients with prior MI. Similar results were found for the relationship between the severity of ischemia and the induced ECG changes. Also, this result was consistent when other ECG markers were investigated, even if the degree of correlation and magnitude of change varied from one to another marker.

The observed differences between patients with and without prior MI in the relationship of the ECG changes with ischemia extension / severity cannot be attributed to differences in the extension and severity between the two groups, which were found not to be statistically significant. In prior MI, the extension and severity percentages were 24.6 % [7.8,29.9] and 36.9 % [35, 43.4], respectively. In non-prior MI patients, these were 16.55 % [5.87,37.92] and 35.9 % [29.8, 44.67], respectively.

3.3. Spatial lead profile of ECG changes

Figure 3 shows the temporal evolution of the different ECG markers during ischemia for patients with each of the three occlusion sites: LAD, LCx and RCA. In all three occlusion sites, the ECG depolarization markers showed a slower and weaker change in response to the induced ischemia than the ECG repolarization markers. Besides, the ST segment deviation, the T wave amplitude and the ratio T amplitude/QRS amplitude showed a larger magnitude of change in patients with LAD occlusion compared to RCA and LCx, while ECG depolarization markers showed similar changes across the three occlusions. The differences in repolarization changes for the different occlusion sites might be attributed to the larger percentage of ischemia ex-

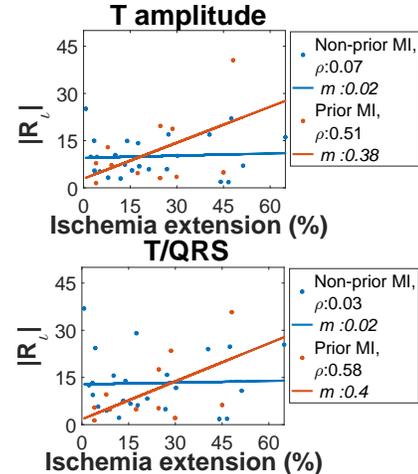


Figure 2. Top: scatter plot of ischemia extension and $|R_i|$ for the T amplitude calculated in mean over the 12 leads after 60 seconds of occlusion. Bottom: same graphic for T amplitude/QRS amplitude. ρ : Pearson correlation coefficient, m : the slope of fitted linear polynomial.

tension and severity measured in LAD patients compared to RCA and LCx.

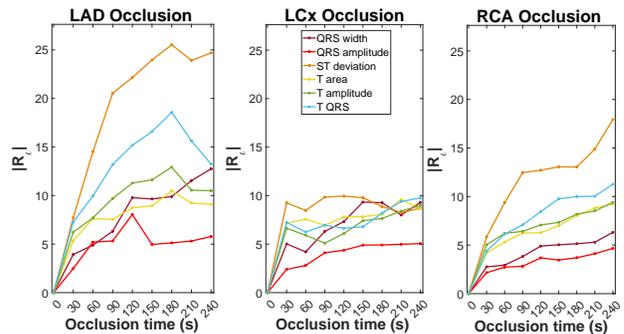


Figure 3. Median values of $|R_i|$, computed from the 12 analyzed leads every 30 seconds from the start of the occlusion, for patients with LAD, LCx and RCA occlusions.

The analysis of ECG changes during occlusion in the 12-lead ECG revealed distinct spatial lead profiles depending on the occluded artery. For both LAD and RCA occlusions, the ECG markers showed similar profiles, with V2, V3 and V4 being the leads displaying the largest magnitude of change in LAD, and II, III and aVF in RCA. LCx occlusion, however, did not display a clear spatial profile.

The presence of prior MI resulted in larger changes in the ECG repolarization markers with respect to patients without prior MI. Figure 4 shows the spatial lead profile of T amplitude during LAD occlusion in patients with and without prior MI, from which the effects of a prior MI on the magnitude of changes can be clearly appreciated. For

the ST deviation, more moderate differences between patients with and without prior MI were found.

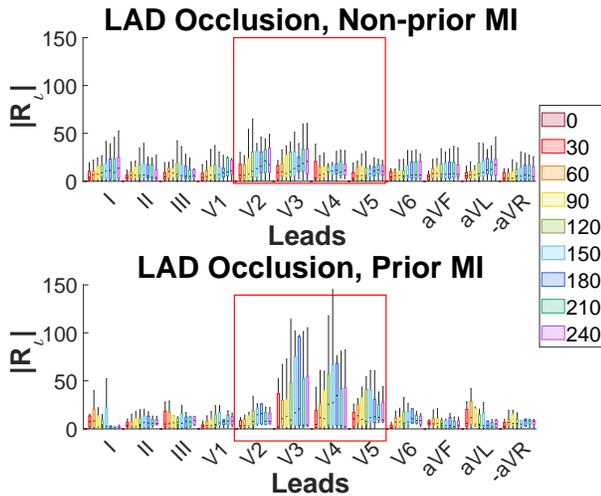


Figure 4. Spatial lead profiles for T amplitude represented as boxplots of $|R_v|$ computed in each of the analyzed leads every 30 seconds from the start of the occlusion.

4. Conclusions

By comparing ECG recordings before and during PCI-induced ischemia we have shown that, on top of ST deviation, other ECG depolarization and repolarization markers show large changes, with the magnitude and temporal evolution of those changes varying as a function of the occlusion site and the presence or not of prior MI. Importantly, ECG changes correlate significantly with the extent and severity of the induced ischemia and present a distinctive spatial lead profile, with such observations being more accentuated in patients with prior MI.

Acknowledgments

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Address for correspondence:

Jimena Rodríguez-Carbó
I3A, Universidad de Zaragoza. Address: C/Mariano Esquillor s/n, 50018, Zaragoza
jimena.rodriquez@unizar.es