ECG-Based Characterization of Acute Ischemia During Percutaneous Coronary Intervention

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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. Our aim was to quantify the changes in the ECG ventricular depolarization and repolarization during acute myocardial ischemia. 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 showed that, in addition to ST deviation, there is a temporal evolution in the QRS and T wave amplitudes, durations and areas during artery occlusion. Repolarization features showed faster and stronger changes than depolarization ones. A distinctive spatial lead profile was described in patients with occlusions in the left anterior descending artery and the right coronary artery. In conclusion, PCI-induced acute ischemia results in changes in the ECG waveforms, specially in the T wave, which vary as a function of the occlusion site.

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 threefold higher than the number of deaths 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, which may involve a percutaneous coronary intervention (PCI).

The diagnosis is commonly based on dynamic changes in the electrocardiogram (ECG) [4]. The most common manifestation of acute coronary artery disease is ST-segment deviation, with larger deviations being associated with higher degrees of myocardial ischemia, morbidity and mortality. Other alterations in the ECG have been observed during acute ischemia, such as a reduction in the downward slope of the QRS complex [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] and the fact that the occlusion is proximal or distal [3]. Our hypothesis is that acute ischemia will change the characteristics of the ECG waveforms, beyond the ST segment, and the changes in the ECG depolarization and repolarization features may present different dynamics.

The main aims of this study are: to automatically quantify ECG markers, including but not limited to the ST segment deviation, before and after PCI; to characterize the temporal evolution of the ECG markers during PCI-induced ischemia; and to describe a spatial lead profile as a function of the occluded artery.

2. Methods

2.1. Study Population

This study analyzed the STAFF III database [8], which consists of 102 patients (mean age of 60.7±11.52 years) receiving elective PCI in one of the major coronary arteries: 21 in the left circumflex artery (LCx), 47 in the right coronary artery (RCA) and 34 in the left anterior descending artery (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 an average of 4.4±1.32 min.

12-lead ECG recordings were obtained before (control recording, CR) and during (PCI recording, PCIR) the clinical procedure. Standard electrode placements were used

for the precordial ECG leads. 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 99mTc-sestamibi as the contrast agent to evaluate the extent and severity (global reduction) of the occlusion [9]. Two tomographic images were obtained, one during the occlusion and one 24 h later (used as a control). The extent was defined as the region where the ratio between the occlusion and control images was below a threshold. The severity was obtained as the pixel difference between control and occlusion images in the delineated region of the extent map, expressed as a fraction of the pixel count withing the same region in the occlusion image.

2.2. ECG Processing

Preprocessing of the ECG signals included filtering, QRS detection [10], and ensemble (for CR) or exponential (for PCIR) beat averaging [5]. For both types of recordings (CR and PCIR), the filtering was performed with a band-pass filter (0.5-40 Hz). After filtering and identification of the QRS 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 whole PCIR recording, exponential averaging was used to follow the dynamic changes during the occlusion:

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

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

2.3. ECG markers and their Changes

The averaged beats along the CR and PCIR recordings were delineated [8] to obtain the beginning, peak and end 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 ι 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 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.

3. Results and Discussion

3.1. Temporal Evolution of ECG changes

Figure 1 shows the temporal evolution of changes in the analyzed ECG markers during the first 4 min of coronary occlusion. All the markers presented remarkable changes during the occlusion, with the largest ones found for the ST-segment deviation followed by markers describing repolarization characteristics such as the T-wave amplitude and area. The depolarization markers showed slower changes and of lesser magnitude during the occlusion. Evaluation at 240 seconds (4 minutes) of occlusion showed that the T wave amplitude increased 9.7 times its standard deviation during CR, while the QRS width increased 7.5 times. The ST deviation showed the largest magnitude of change, with such magnitude being 18.2 times its normal variations during control.
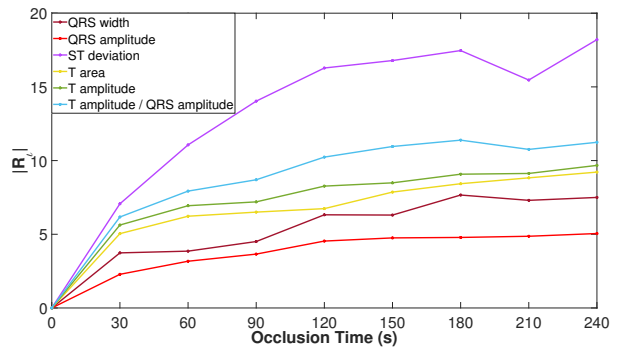

Figure 1. Median values of $|R_\iota|$ over patients, computed from the 12 analyzed leads, every 30 seconds from the start of the occlusion, for each of the analyzed ECG markers (ι).
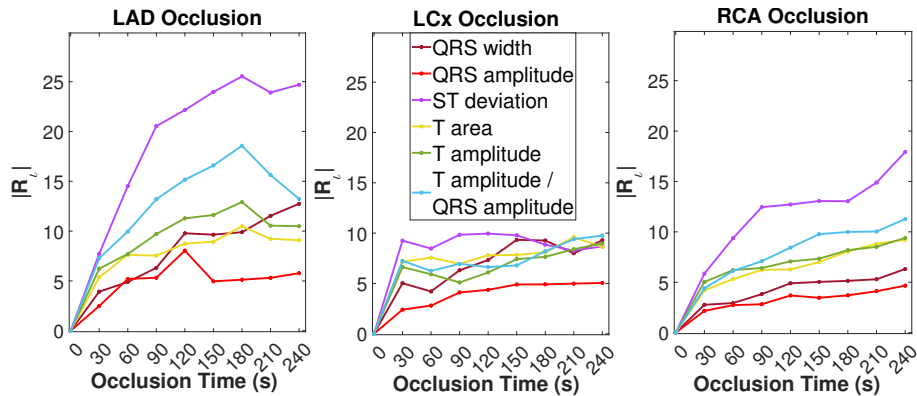
Figure 2. Median values of $|R_\iota|$, computed from the 12 analyzed leads every 30 seconds from the start of the occlusion, for patients with LAD, LCx and RCA occlusions, for each of the analyzed ECG markers (ι).
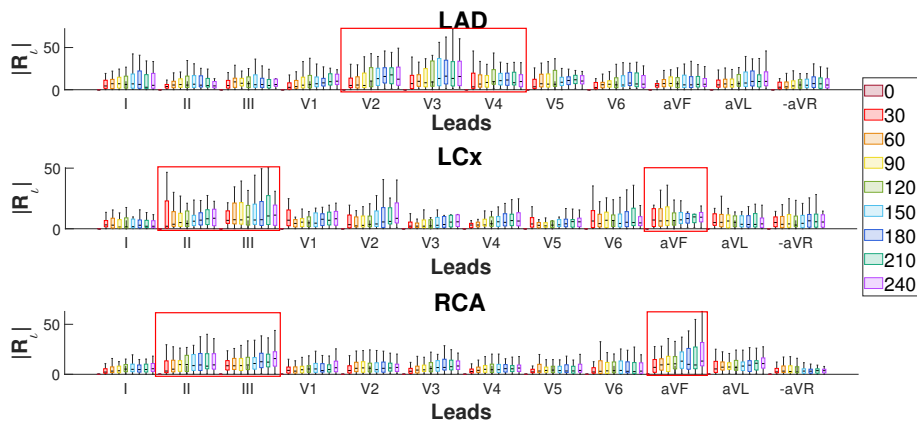

Figure 3. Spatial lead profiles for T amplitude represented as boxplots of $|R_\iota|$ computed in each of the analyzed leads every 30 seconds from the start of the occlusion.

3.2.   Spatial lead profile of ECG changes

Figure 2 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 (specially QRS amplitude) 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. Only after 4 min of occlusion, a more pronounced change in QRS width was observed in patients of the LAD group compared to patients in the LCx and RCA groups. The differences in the magnitude of repolarization changes for the different occlusion sites might be attributed to the larger percentage of ischemia extent and severity measured in LAD patients compared to RCA and LCx.

The analysis of ECG changes during occlusion in the 12-lead ECG revealed distinct spatial lead profiles depending on the occluded artery. Figure 3 shows the spatial lead profile for the T-wave amplitude and figure 4 for the QRS width. As can be observed, for both LAD and RCA occlusions, the two ECG markers showed similar profiles, with V3 and V4 being the leads displaying the largest magnitude of change in the LAD group, and leads II, III and aVF in the RCA group. The LCx group, however, did not display a clear spatial profile. This spatial lead profile seemed to be more prominent for the QRS width than for the T-wave amplitude, specially for the LAD occlusion.

4.   Conclusions

By comparing ECG recordings before and during PCI-induced ischemia we have shown that, on top of ST-
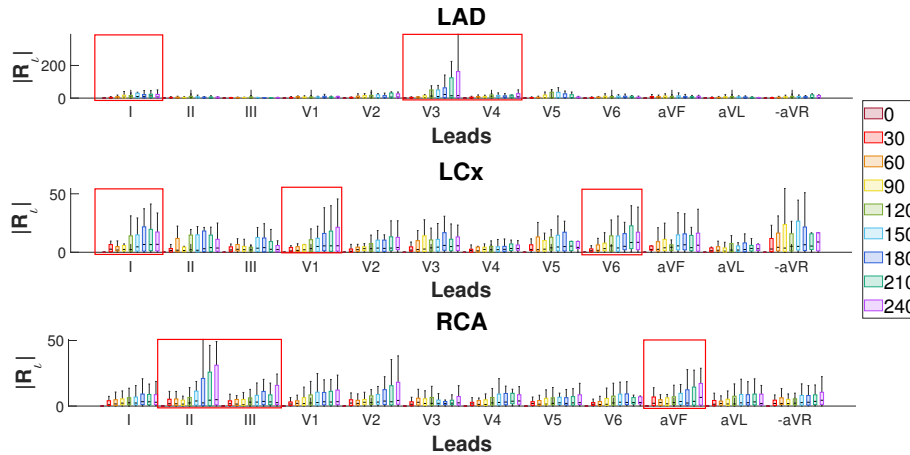
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Figure 4. Spatial lead profiles for QRS width represented as boxplots of $|R_\iota|$ computed in each of the analyzed leads every 30 seconds from the start of the occlusion.

segment deviation, other ECG depolarization and repolarization markers undergo important changes during ischemia. The magnitude and temporal evolution of those changes vary as a function of the occlusion site. Importantly, the T-wave markers show faster and stronger changes than the QRS-complex markers. All analyzed markers present a distinctive spatial lead profile, particularly for LAD and RCA occlusions.

Acknowledgments

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