

# THE RELATION BETWEEN LATE VENTRICULAR POTENTIALS AND ELECTROCARDIOGRAPHIC DISPERSION OF VENTRICULAR ACTIVITY IN MYOCARDIAL INFARCTION PATIENTS

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## REZUMAT

**Obiectiv:** Stabilirea relației existente între parametrii de dispersie a activității ventriculare și potențialele ventriculare tardive (PVT), ca markeri neinvazivi ai riscului aritmogen. **Material și metodă:** Au fost investigați 16 pacienți cu infarct miocardic cronic (vârsta:  $63 \pm 16$  ani, 13 bărbați și 3 femei) utilizând SAECG (PVT) și ECG în 12 derivații (indicii de dispersie a activității ventriculare). **Rezultate:** Valori medii mai mari au fost obținute pentru toți parametrii de dispersie, la toți pacienții care au prezentat PVT, comparativ cu pacienții fără PVT. Diferențe semnificative statistice au fost obținute doar pentru: QTd, QTm, QTcmax, JTcmax, QRSd, QRSdc, QRSmax. Indicii de variabilitate QT s-au corelat cu durata QRS și amplitudinea semnalului electric din ultimele 40 ms ale complexului QRS.

**Concluzii:** Infarctul miocardic are potențialul de a întârzia activarea ventriculară, fapt demonstrat de prezența PVT, ceea ce determină și creșterea parametrilor de dispersie a activității ventriculare. Totuși cele două metode utilizate au valoare predictivă diferită pentru riscul aritmogen, PVT nereflectând atât de fidel heterogenitatea structurală a miocardului postinfarct, iar dispersia activității ventriculare nefiind o expresie a blocurilor funcționale postinfarct.

**Cuvinte cheie:** infarct miocardic, dispersia QT, dispersia JT, dispersia QRS, potențiale ventriculare tardive

## ABSTRACT

**Aim:** Evaluation of the relationship between electrocardiographical dispersion of ventricular activity and late ventricular potentials (LVP), as noninvasive markers for arrhythmia. **Material and methods:** 16 patients with chronic myocardial infarction (MI) were investigated (age:  $63 \pm 16$  years, 13 men and 3 women) using SAECG (LVP) and 12-lead ECG (ventricular activity dispersion indices). **Results:** Higher average values, for all dispersion parameters, were found in all patients who had LVP, compared to the patients without LVP. There were statistical significant differences only for QTd, QTm, QTcmax, JTcmax, QRSd, QRSdc, QRSmax. The QT interval variability indices correlate with the QRS duration and the electrical signal amplitude from the last 40 ms of the QRS complex. **Conclusions:** The results of the study suggest that myocardial infarction is able to delay ventricular activation, demonstrated by the existence of LVP, and also to increase dispersion of ventricular activity. Nevertheless, the two used methods have different predictive value for arrhythmia, because LVP do not reflect with high accuracy the structural heterogeneity of myocardium after MI, and dispersion of ventricular activity is not an expression of the functional postinfarction block.

**Key Words:** myocardial infarction, QT dispersion, JT dispersion, QRS dispersion, late ventricular potentials.

## INTRODUCTION

The estimation of QT dispersion (QTd) using 12-lead electrocardiogram established a quantitative and noninvasive method to determine myocardial repolarisation inhomogeneities.<sup>1,2</sup> An increased index was proposed as a marker of arrhythmia risk after myocardial infarction.<sup>2</sup> This is also valid for the other dispersion parameters of the ventricular activity.

Signal-averaged ECG averages multiple QRS complexes, minimizing the level of noise that contaminates the periodic ECG signal, thereby exposing signals at the microvolt level that are normally hidden within noise.<sup>3</sup> These low amplitude and high frequency ventricular signals at the end of the QRS complex are termed late potentials and represent delayed conduction through diseased myocardium, as a consequence of decremental conduction of the impulse, and are a potential physical substrate for reentry ventricular tachycardias.<sup>2,4</sup> Late potentials usually arise from the border zone surrounding the scar of a previous myocardial infarction and can be also defined as the fragmented electrical activity of the inhomogenous myocardial tissue. The use of time-domain analysis to identify late potentials and to predict risk for ventricular arrhythmias has been validated in numerous studies and is the method used in commercial equipment.<sup>3</sup>

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LVP represent the ECG recording of some ventricular macroreentry circuits. The structural heterogeneity contributes to the increase of the ventricular depolarisation and repolarisation dispersion, and to fragmentation of the local electric activity and generation of LVP.<sup>1</sup> Arrhythmia risk persists after myocardial infarction even after 15 years from the coronary event.

The aim of this study was to determine the correlation between LVP and QTd, as noninvasive markers of arrhythmia risk, to establish a common mechanism for the occurrence of QTd and LVP, in other words examination of the relation between QTd and intraventricular conduction anomalies in patients with an old myocardial infarction.

## **MATERIAL AND METHODS**

The study enrolled 16 patients from the ASCAR Cardiology Clinic in Timișoara, diagnosed with an old myocardial infarction (at least 1 year), with or without pathological Q wave, considering the criteria of the Joint European Society of Cardiology and of the American College of Cardiology Committee for the Redefinition of Myocardial Infarction.<sup>5</sup> The characteristics of the investigated patients are synthesized in Table 1.

**Table 1.** The characteristics of the investigated MI patients.

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**Age:** 63±16 years

**Sex:** 13 men and 3 women

**Time from onset** of MI: 1-9 years

**MI location:** 9-anterior MI, 5-inferior MI, 2-anterior and inferior MI

**Cardiovascular risk factors:** smokers: 5, diabetes mellitus: 5, HTA: 8, obesity: 7, BMI: 24-37, dyslipidemia: 2

**NYHA functional class:** 6 in III NYHA class

**LV aneurism:** 1

**Therapy:** angiotensin-converting enzyme inhibitors (8), calcium-blockers (2), nitrates (14), beta-blockers (2), cardiotonic agents (2), III class antiarrhythmics (4), lipid lowering drugs (2), anticoagulant (2)

**History of arrhythmia:** atrial fibrillation and flutter, not in the moment of the recording

**Associated pathology:** mitroaortic disease, cor pulmonale

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Patients with atrial fibrillation or atrial flutter in the moment of the recordings were excluded. Three of the patients previously presented atrial fibrillation or flutter, but not in the moment when SAECG was performed. Patients with hypokalemia, hypocalcemia or hypomagnesiemia, were also excluded because

hydroelectrolytic disturbances may increase QT dispersion.

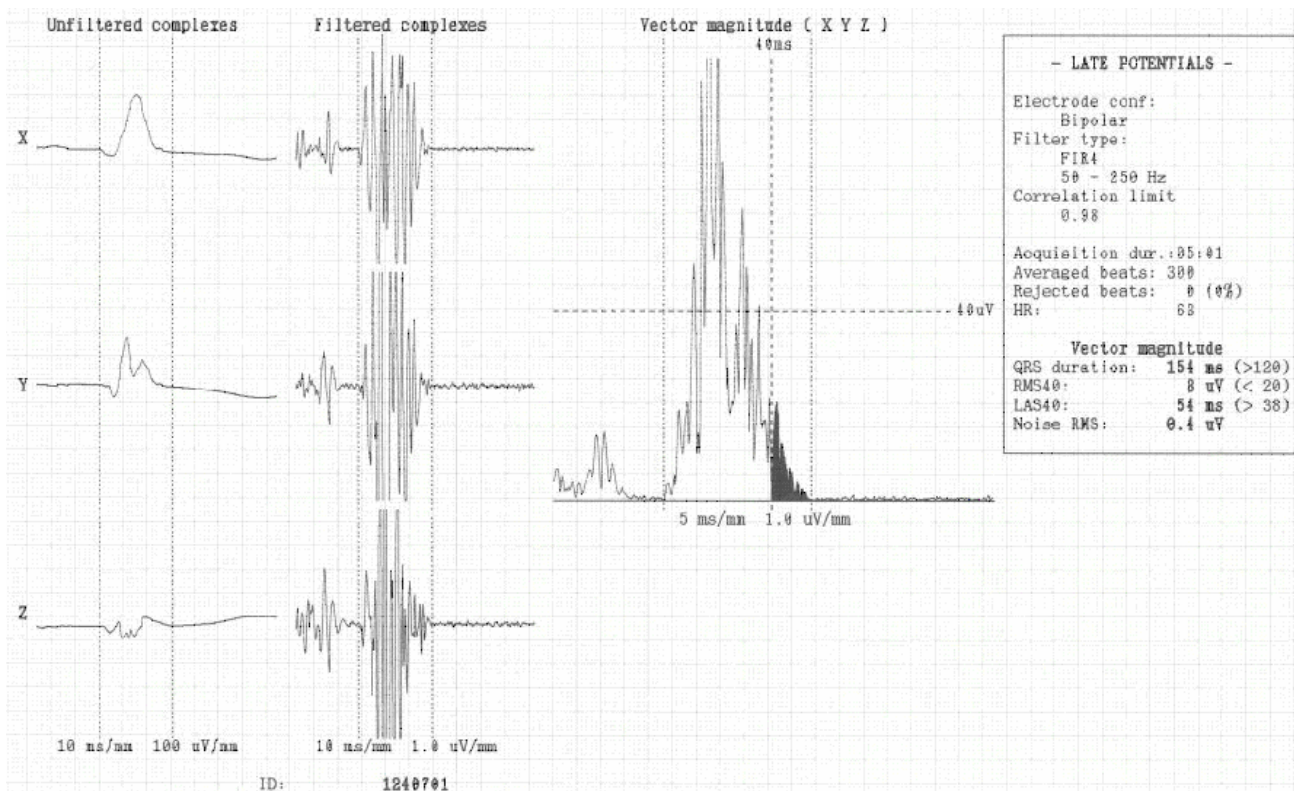
The following investigations were performed between December 2002 - July 2003: 12-lead ECG and signal averaged electrocardiography (SAECG), in the functional exploration laboratory of the Patho-Physiology Department. For all investigated patients there was access to their history, the results of their clinical examination, therapy and the results of the paraclinical investigations.

SAECG was performed using a Siemens Megacart device, from the endowment of our laboratory. The single use electrodes were applied as follows: R – on the right arm, L – on the left arm, F – on the left leg, N- on the right leg, C<sub>1</sub>- on the right midaxillary line, in the V intercostal space, C<sub>2</sub> – on the left midaxillary line, in the V intercostal space, C<sub>3</sub> – at the bottom of the sternum, C<sub>4</sub> – on the left iliac crest, C<sub>5</sub> – posterior, in the IV intercostal space, on the right side of the spinal column, C<sub>6</sub> – in the IV intercostal space, on the left side of the sternum (the normal position for V<sub>2</sub>). Three orthogonal bipolar leads resulted: x (connecting the leads C<sub>1</sub> and C<sub>2</sub>), y (connecting leads C<sub>3</sub> and C<sub>4</sub>) and z (connecting leads C<sub>5</sub> and C<sub>6</sub>).

A correlation coefficient between 0.90 and 0.98 was selected. The signals which during recording were under this value, were rejected. The signal derived from the 300 beats was amplified, processed and mediated, and filtrated with a bidirectional filter. A 50-250 Hz filter was selected. The recording lasted for 2.02 - 10.30 minutes, and the noise level between 0.3 and 0.6 μV.

The following parameters are essential for the existence of LVP: QRS duration, LAS40 (the duration of the signal at the end of the QRS complex with an amplitude below 40 μV - „Low Amplitude Signal”) and RMS40 („Root Mean Square”- the square root of the last 40 ms of the signal). (Figure 1) We considered a patient having LVP, if two of the following criteria are positive: QRS duration >120 ms, LAS40 > 38 ms and RMS40 < 20 μV.

12-lead ECG was also performed, using the same device (SAECG), at a paper speed of 25 mm/s. The ECGs were examined by two independent observers, who weren't informed about the clinical data. The QT interval was manually measured from the start of the QRS complex to the end of the T wave. QT dispersion (QTd) was calculated as the difference between the maximal and minimal value of the QT interval in the 12 leads, QTdc: the heart rate corrected QTd using the Bazett formula:  $QTc = QT/\sqrt{RR}$ , and the mean QT duration (QTm). The JT interval was measured from the beginning of the J point to the



**Figure 1.** LVP in a patient with both anterior and inferior MI.

end of the T wave and, in a similar way, the values for: JT dispersion (JTd), JTdc (heart rate corrected JTd) and the mean duration of the JT interval in the 12 leads (JTm) were calculated.

The values for QRS dispersion (QRSd), QRSdc (heart rate corrected QRS dispersion), QRSmax (heart rate corrected maximal value of QRS) and QRSmin (heart rate corrected minimal QRS value) were also calculated.

QTd, JTd and QRSd were calculated, considering at least 8 leads for each patient, eliminating the leads in which the end of the T wave couldn't be determined exactly, or in which the T wave had a too low amplitude or was isoelectric. The end of the T wave was defined as the return to the isoelectric line P-T-P. For each lead, two QT intervals were measured, and the mean value of the two measured QT intervals was considered. The obtained values were comparable with those mentioned by other authors. QTm and JTm were calculated for each patient as an average of the QT and JT duration in every lead.

## RESULTS

For the investigated patients the values from Table 2 were recorded. 62.5% (10) of the 16 investigated patients had LVP. The values from Table 3 were obtained for the ventricular activity variables.

**Table 2.** The values of the LVP parameters in the investigated chronic myocardial infarction patients.

	QRS duration	LAS40	RMS40
	(Patol > 120 ms)	(Patol > 38 ms)	(Patol < 20 $\mu$ V)
Recorded values	128 $\pm$ 28 ms	61 $\pm$ 40 ms	20 $\pm$ 16 $\mu$ V

Statistical analysis was performed using the t Student test to prove the existence of statistical significant differences between the patients groups ( $p < 0.05$  was considered significant), and the Bravais-Pearson correlation coefficient, to evaluate the correlation type between the different determined parameters.

The patients were divided in 2 groups: patients with LVP (10 patients) and without LVP (6 patients). The values of the variables which reflect dispersion of ventricular depolarisation and repolarisation were compared in the two groups of patients with chronic myocardial infarction: with and without LVP. The values obtained for  $p$  with the t Student test, the statistical significance of the differences, and the mean values of the variables in the two groups of patients may be found in Table 4.

Higher average values for all dispersion parameters in all patients who had LVP were found, compared to the patients without LVP. There were statistical significant differences only for QTd, QTm, QTcmax, JTcmax, QRSd, QRSdc, QRSmax.

**Table 3.** The values of the main ventricular activity dispersion parameters.

Dispersion parameter	Obtained values (ms)
QT <sub>d</sub>	159 ± 111
QT <sub>dc</sub>	34 ± 24
QT <sub>m</sub>	413 ± 87
JT <sub>d</sub>	122 ± 63
JT <sub>dc</sub>	21 ± 17
JT <sub>m</sub>	302 ± 59
QT <sub>cmax</sub>	112 ± 33
QT <sub>cmin</sub>	79 ± 18
JT <sub>cmax</sub>	78 ± 19
JT <sub>cmin</sub>	54 ± 14
QRS <sub>d</sub>	120 ± 90
QRS <sub>dc</sub>	32 ± 27
QRS <sub>max</sub>	50 ± 30
QRS <sub>min</sub>	17 ± 11

**Table 4.** The significance of the differences of the ventricular activity dispersion variables and the average values obtained in patients with and without LVP.

Analyzed parameter	p	Mean values in patients with LVP (ms)	Mean values in patients without LVP (ms)
QT <sub>d</sub>	0.043	150.8	80.5
QT <sub>dc</sub>	0.088	33.5	18.16
QT <sub>m</sub>	0.016	440	382.66
QT <sub>cmax</sub>	0.033	117.4	95.16
QT <sub>cmin</sub>	0.28	84.5	79
JT <sub>d</sub>	0.109	119.2	78.33
JT <sub>dc</sub>	0.36	22.6	17.5
JT <sub>m</sub>	0.11	307.7	279.16
JT <sub>cmax</sub>	0.047	80.7	69.83
JT <sub>cmin</sub>	0.25	56.9	52.33
QRS <sub>d</sub>	0.05	100.4	56.83
QRS <sub>dc</sub>	0.05	23.8	12.16
QRS <sub>max</sub>	0.048	43.8	30.83
QRS <sub>min</sub>	0.93	18.9	18.66

In summary, patients with increased non-homogeneity of ventricular activity also have a fragmentation of the QRS complex in its terminal part, responsible for the LVP. Or, viceversa, considering the higher negative predictive value of the LVP, patients with a higher homogeneity of the ventricular activity do not have arrhythmia risk.

A direct moderate correlation between QTd and QRS duration, and a moderate inverse correlation

between QTd and RMS40 were also found. Heart rate correction of QTd maintains the direct correlation to QRS duration ( $r = 0.47$ ) and the weak correlation to RMS40 ( $r = 0.29$ ). The third parameter used to define LVP (LAS40) didn't correlate either with QTd or with QTdc, but presented a moderate correlation with QTm ( $r = 0.39$ ). (Table 5)

**Table 5.** The value of the linear correlation coefficient between LVP components and QT dispersion in chronic myocardial infarction patients with LVP.

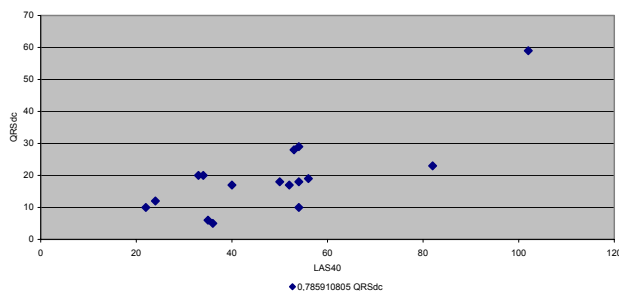
	QRS duration	LAS40	RMS40
QT <sub>d</sub>	$r = 0.46$	$r = 0.14$	$r = -0.37$
QT <sub>dc</sub>	$r = 0.47$	$r = 0.13$	$r = -0.29$
QT <sub>m</sub>	$r = 0.78$	$r = 0.39$	$r = -0.16$
QT <sub>cmax</sub>	$r = 0.102$	$r = 0.38$	$r = -0.35$
QT <sub>cmin</sub>	$r = 0.26$	$r = 0.066$	$r = 0.39$
JT <sub>d</sub>	$r = 0.27$	$r = 0.098$	$r = -0.28$
JT <sub>dc</sub>	$r = 0.24$	$r = 0.083$	$r = -0.17$
JT <sub>m</sub>	$r = -0.87$	$r = -0.39$	$r = -0.22$
JT <sub>cmax</sub>	$r = 0.113$	$r = 0.103$	$r = 0.19$
JT <sub>cmin</sub>	$r = 0.34$	$r = 0.096$	$r = 0.44$
QRS <sub>d</sub>	$r = 0.59$	$r = 0.79$	$r = 0.38$
QRS <sub>dc</sub>	$r = 0.27$	$r = 0.802$	$r = 0.47$
QRS <sub>max</sub>	$r = 0.55$	$r = 0.73$	$r = 0.52$
QRS <sub>min</sub>	$r = 0.58$	$r = 0.201$	$r = 0.43$

As a conclusion, the QT interval variability indices correlate with the QRS duration and the electrical signal amplitude from the last 40 ms of the QRS complex with an amplitude of the electrical signal lower than 40  $\mu$ V.

Further, in the same study, the relation, in patients with chronic myocardial infarction (with and without LVP), between the SAECG-LVP components and the ventricular dispersion parameters (QTd, QTdc and QTm), and the ventricular depolarisation dispersion parameters (QRSd, QRSdc, QRSmax and QRSmin) was analysed (see also Figure 2). The values obtained for the linear correlation coefficient, may be seen in Table 6. One can notice that the values for  $r$  do not differ to much if we consider all the investigated patients or just those with LVP. We found higher values for  $r$  (better correlations) between the SAECG parameters and those for ventricular depolarisation dispersion (QRSd, QRSdc, QRSmax and QRSmin). We expected a higher correlation of the QRS duration on the SAECG with QRSd ( $r = 0.61$ ) compared to QTd ( $r = 0.44$ ), considering that the LVP represent

**Table 6.** The correlation coefficient between the LVP components (QRS duration, LAS40 and RMS40) and the dispersion parameters of ventricular activity in chronic myocardial infarction patients.

	QRS duration	LAS40	RMS40
QT <sub>d</sub>	0.44	0.09	-0.37
QT <sub>dc</sub>	0.45	0.05	-0.21
QT <sub>m</sub>	0.21	0.14	-0.48
QT <sub>cmax</sub>	0.518	0.12	-0.14
QT <sub>cmin</sub>	0.22	0.1006	0.179
JT <sub>d</sub>	0.46	0.25	-0.45
JT <sub>dc</sub>	0.30	0.138	-0.31
JT <sub>m</sub>	-0.04	-0.024	-0.39
JT <sub>cmax</sub>	0.47	0.208	0.11
JT <sub>cmin</sub>	0.24	0.105	0.25
QRS <sub>d</sub>	0.61	0.78	-0.34
QRS <sub>dc</sub>	0.48	0.78	-0.22
QRS <sub>max</sub>	0.61	0.72	-0.13
QRS <sub>min</sub>	0.34	0.09	0.28



**Figure 2.** The correlation between LAS40 (determined using SAECG) and QRSdc (determined using 12 lead ECG) is positive and strong ( $r = 0.78$ ) in chronic myocardial infarction patients.

fragmented electrical activity from the terminal part of the QRS complex, and QT dispersion comprises also ventricular repolarisation. Heart rate correction of the dispersion parameters becomes questionable, considering the higher values for  $r$  when we correlate the uncorrected dispersion values with the LVP parameters.

## DISCUSSIONS

A moderate correlation between QT<sub>dc</sub> and SAECG-QRS duration ( $r = 0.459$ ) was also mentioned in an article published by Dineva and Koichev, suggesting that the existence of some slow conducting myocardial areas, related to positive LVP, is associated with a higher inhomogeneity of ventricular repolarisation, expressed as a higher QT<sub>dc</sub>.<sup>6</sup>

A similar study, which tried to find some correlations

between QT<sub>d</sub> and slow intraventricular conduction in anterior myocardial infarction patients, didn't find any correlation between electrocardiographic parameters and those obtained using SAECG, concluding the independent predictive value for QT<sub>d</sub> and LVP for arrhythmia risk.<sup>7</sup> The difference to this study consists in the use of the precordial leads, with the motivation of a better representation of the myocardial activity from the adjacent region of the electrode.

This study preferred to calculate QT<sub>d</sub> using all the 12 ECG leads, undergoing the risk of recording the different projections of the global cardiac vector, considering that most of the studies from the literature which found a correlation between QT<sub>d</sub> and appearance of ventricular arrhythmias, used all 12 standard leads.<sup>8-11</sup>

Kirchhof et al indicates that the existence of LVP in patients with myocardial infarction, doesn't affect QT<sub>d</sub> and QT<sub>dc</sub>, but, JT<sub>d</sub> and JT<sub>dc</sub>, were significantly higher in patients with LVP.<sup>12</sup> Among the analyzed LVP variables, only the QRS complex duration could be associated with significantly higher JT<sub>d</sub> and shorter JT<sub>min</sub> intervals. The mean values of the minimal and maximal QT<sub>c</sub> were higher in patients with LVP, the same thing being valid for QT<sub>c</sub>, but not for QT<sub>d</sub>. The difference to this study consists in using subacute myocardial infarction patients (at 2-3 weeks after infarction), whereas in this study, all patients had chronic myocardial infarction.

Puljevic et al finds the same insignificant difference between QT<sub>d</sub> in patients with and without LVP, and shows that the lack of correlation between LVP and QT<sub>d</sub>, although risk factors for arrhythmia, may be caused by different electrophysiologic abnormalities.<sup>13</sup>

Beta-blockers have the ability to reduce QT<sub>d</sub> and QT<sub>dc</sub>, and digoxin increases the dispersion indices, having the ability to influence the results. Nitrates and calcium-blockers do not influence QT<sub>d</sub>.<sup>14</sup> There are some drugs which prolong the QT interval associated with a high risk for ventricular arrhythmias.<sup>15-17</sup> Class III antiarrhythmics (sotalol, amiodarone) prolong repolarization by blocking the potassium channel.<sup>15,18-20</sup> Several medical articles mention decrease of QT<sub>d</sub> after angiotensin converting enzyme inhibitor therapy.<sup>21</sup> Antiarrhythmic drug therapy, on the other hand, decreases the amplitude of the late potentials without abolishing them.<sup>2</sup>

Considering the small number of investigated patients, an additional study would be necessary before the clinical use of the results of this study. A correlation with postinfarction arrhythmias could allow to select the best predictive method.

## CONCLUSIONS

The results suggest that the existence of LVP is associated with a higher inhomogeneity of ventricular depolarisation and repolarisation. QT dispersion is more affected by the QRS duration, than by RMS40, which means less by the terminal part of the QRS and more by the total QRS duration. QTd correlates also with the amplitude of the signal in the last 40 ms of the QRS complex (more than with the duration of this signal), suggesting that QT dispersion, can be ascribed to the presence of slowly conducting areas of myocardium, related to LVP, considering also the correlation between LAS40 and QTm. It is unlikely for QTd to be only an expression of postinfarction functional blocks; LVP do not accurately reflect the structural inhomogeneity of postinfarction myocardium, and the absence of a more important correlation emphasizes the different predictive value of the two exploring methods.

In other words, myocardial infarction has the potential to delay ventricular activation, demonstrated by the presence of LVP, which significantly increases QTd. On the other hand, QTd indicates differences in the action potential duration. Although the action potential duration also comprises the depolarisation time, it especially depends on the repolarisation time. The LVP, as markers of a delayed conduction through an inhomogeneous scar, explores only ventricular depolarisation.

Heart rate correction of the dispersion parameters is questionable, considering the higher *r* values when we correlate the uncorrected dispersions with the LVP parameters.

In patients with chronic myocardial infarction, the depolarisation and repolarisation variability indices correlate with the parameters of the LVP, even if the patients don't have enough criteria for LVP (at least two). This findings should redefine the diagnostic criteria for LVP.

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