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LEADING ARTICLE

Late ventricular potentials and their significance for clinical practice

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Cardiovascular diseases (CVD) keep the leading position among the mortality causes in the developed countries, and in the Russian Federation within the last 10 years around 60 % of deaths have been caused by CVD. Various arrhythmias and/or acute myocardial ischemia are one of the main causes of cardiovascular mortality. Therefore, it is particularly important to provide the early diagnostics of myocardial electric instability with its consequent correction and secondary prevention. One of the methods for determining the electrical instability of the myocardium is the registration of late ventricular potentials using signal-averaged electrocardiography. There are numerous evidences demonstrating that late ventricular potentials registration may be used as an available, non-invasive, and safe method of risk stratification in patients with cardiovascular disease in terms of aggravation of principal disease, risk of developing ventricular tachycardias and sudden cardiac death. However, the attitude to the phenomenon of late ventricular potentials is ambiguous, and information about its influence on the prognosis of cardiovascular diseases is contradictory. This article reviews methodologic aspects of late ventricular potentials registration, data on their sensitivity and specificity in different conditions (coronary artery disease, including unstable angina, acute myocardial infarction, ventricular tachycardias, etc), and the questions of prognostic importance and possibility of late ventricular potentials correction.

Keywords: *late ventricular potentials, sudden cardiac death, ventricular tachycardia, signal-averaged electrocardiography.*

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Introduction

Over the last years cardiovascular mortality has decreased in high-income countries in response to adoption of preventive measures to reduce burden of CAD and heart failure. Despite these results, cardiovascular diseases are responsible for approximately 17 million deaths every year in the world, approximately 25 % of which are sudden cardiac death (SCD) [1].

In the structure of total mortality in Russian Federation over the last 10 years cardiovascular diseases occupy about 60 %. Approximately in 70 % of cases the death from CAD is sudden [2] and in 25–30 % of cases SCD is the first and last manifestation of CAD [3]. The main causes of SCD in young patients are the pathologies of ion channels, cardiomyopathies, myocarditis, several intoxications, including narcotic intoxication, while in older patients the main causes of SCD are degenerative diseases (CAD, valvular heart diseases and heart failure). Prediction of sudden cardiac death is the insoluble and everlasting question of arrhythmology, which promoted several studies attempting to find reliable prognostic markers of sudden cardiac death over the last 10 years.

The basis for the development of arrhythmogenic death mechanisms is the electrical instability of myocardium, which can be detected by the method of registration of late ventricular potentials (LVP) by using high-resolution signal-averaged electrocardiography (SA-ECG) [4]. Signal averaged ECG has been included to diagnostic approach for family members of sudden unexplained death syndrome or sudden arrhythmic death syndrome victims due to ESC guidelines (2015) [1].

Methodological aspects

LVP are low-amplitude potentials due to fragmented electrical activity at the terminal part of QRS complex or at the beginning of ST segment during the diastole of ventricles appearing at the region of delayed excitation of myocardium also known as «delayed myocardial depolarization potentials», which creates arrhythmogenic zones and contributes to the appearance of malignant ventricular tachyarrhythmias (VTA). Back in the 70s of the XX century E. Berbary et al., L. Fontain et al. discovered delayed ventricular electrical activity that preceded ventricular tachyarrhythmias while analyzing the records from epi- and endocardial electrodes [5].

The mechanism of micro-reentry is considered to be the cause of LVP phenomenon at the regions of local delayed excitation with different origin: ischemic zone of myocardium, local electrolyte balance disturbances, sympathetic activation and other impacts that cause local conduction delay and the appearance of spontaneous electrical activity focuses [6–8].

LVP are detected during SA-ECG with the ECG signal frequency of 1000 Hz and more. Quantitative criteria for LVP can be calculated by three parameters: a) duration of the filtered QRS complex (totQRS); b) duration of low-amplitude signals (< 40 mkV) at the end of the QRS complex (LAS-40); c) root-mean-square amplitude of last 40 ms of the filtered QRS complex (RMS-40). The appearance of at least two of three of these criteria allows to diagnose LVP. It is remarkable that every criterion is considered to be specific, while its sensitivity varies depending on the operation conditions of filters, interference and other external conditions.

Processing of the data obtained during SA-ECG involves the averaging of cardiac signal or sequential analysis of complexes The Simpson method is the most common method of temporal signal averaging which includes the summation of hundreds of sequential cardiac cycles [9]. ECG registration is conducted using three orthogonal leads (X, Y, Z) with the following filtering of the signal. The important advantage of this high-resolution ECG averaging method is the ability to extract the signal from the noise (to stabilize LVP by extracting it from accidental noise).

However, this method has significant disadvantages, such as: the probability of smoothing the high-frequency signals is high because of unstable characteristics of LVP (duration, configuration, frequency of occurrence); the risk of signal distortion by filters and other external sources; deformed and wide QRS complex can obscure late potentials in patients with interventricular conductance disturbances [10].

Spatial averaging is another useful method, which involves simultaneous registration of several (4–16) ECG using a large number of closely spaced pairs of electrodes [11–13].

The most interesting and promising method is spectral analysis of SA-ECG. This technique expands the diagnostic possibilities and has a number of advantages: elimination of background and filter noise, the opportunity to investigate patients with His bundle branch blocks and, moreover, no effect of focal myocardium changes localization on the results [10].

Another method of spectral analysis is wavelet transform for time-frequency mapping of the signal. This technique allows to register and analyze LVP

with modern radiophysical methods by using a large number of cardiac cycles without signal averaging [11].

LVP can be analyzed by the next quantitative criteria (Simpson): duration of the filtered QRS complex after averaging (totQRS > 114 ms); duration of low-amplitude signals (< 40 mkV) (LAS-40) longer than 38 ms; c) root-mean-square amplitude of last 40 ms of the filtered QRS complex (RMS-40) less than 25 mkV [6].

The analysis of late ventricular potentials has been suggested to be used according to the results of Holter monitoring since 1989. Using automatic analysis of LVP during Holter monitoring M. Sosnowski et al. identified two different groups of patients: with and without late potentials [14]. The criteria to determine the presence of late potentials were: totQRS ⩾120 ms; RMS-40 ⩽25 mkV; LAS-40 ⩾39 ms. The patients with myocardial infarction had the circadian rhythm of LVP registration. Specificity of parameters detection reached 100 % from 9 to 12 o'clock in the morning and was lower at night (80 %).

However, L. Zhao identified slightly different criteria to determine the presence of LVP in patients with ventricular tachycardia during Holter monitoring: totQRS ⩾114 ms; RMS-40 ⩽12 mkV; LAS-40 ⩾38 ms. The sensitivity of positive ECG criteria in the patients with tachycardia was 95.7 % and the specificity in the patients without arrythmia was 97.8 %.

Consequently, nowadays there is no consensus regarding which analysis (time, spatial or spectral) is the most preferred to determine LVP and which parameters for totQRS, RMS-40, LAS-40 are more diagnostically accurate.

Prognostic value of late ventricular potentials

Registration of LVP is mostly used for prediction of sudden cardiac death and ventricular arrythmias development in survivors of myocardial infarction (MI). It is important to determine the frequency of LVP on different stages of the disease and the correlation between the localization of MI and the development of LVP. SA-ECG is considered to be a useful and promising non-invasive method for identification of myocardial infarction survivors with high risk of arrythmias, especially VTA.

Thus, Rubal B.J., Bulgrin, Gilman J.K. [16] concluded that the sensitivity of SA-ECG in LVP analysis is about 92–100 % and specificity is about 78–92 % in 90 MI survivors with high risk of sudden cardiac death.

The dynamics of LVP is reflected by instability, variability, electrophysiological and morphological characteristics of cardiomyocytes. Delayed ventricular activity is caused by the alteration of vital cardiomyocytes and areas of ischemia, necrosis and fibrosis. Recovery of ischemic myocardium function and ischemic zone demarcation during treatment lead to cessation of LVP. Delayed fragmented ventricular activity is more often detected on the early stages of acute myocardial infarction (AMI) and in 60 % of patients at the stage of ischemia, but the maximum is registered from 10th to 14th day. Thus, in Zhalyunas R. et al. study LVP were detected on the first day after AMI in 40.5 % of patients, on the third day in 28.5 % of patients and on the fourteenth day in 45.2 % of patients [17]. It is remarkable that in patients with Q wave AMI LVP were significantly more common on the 3d and 14th day (39.1 % and 7.7 %, 55.1 % and 23.1 % respectively).

According to some data LVP detection is more common during the first month after AMI with future decreases of frequency. Pozdnyakova N.V. et al. noted that in 31 % of patients with AMI LVP were detected during all days of study, in 25 % LVP were detected only at the acute phase of MI and disappeared completely by the time of discharge from the hospital and in 44 % of patients LVP weren't detected during the next ECG registrations [18]. The reason for the LVP disappearance can be myocardial «stunning» and «hibernation» by the time of discharge. Buziashvili Yu. I. et al. found LVP in 18.7 % of patients with irreversible myocardial dysfunction and in 61.1 % of patients with hibernation of cardiomyocytes [19]. Consequently, the authors showed that myocardial hibernation is more common reason for LVP appearance than cicatricial changes.

According to other data the viable myocardium in patients with EF < 40 % in the early post-infarction period had correlation with the absence of LVP: viable cardiomyocytes were detected in 80 % of patients without LVP and only in 35 % with LVP by the results of stress echocardiography and SA-ECG. Similar results were obtained in another study: patients with LVP had asynergy zones in 47 % of cases, whereas patients without LVP had them only in 28 % of cases [20, 21].

During the investigation of LVP in patients with acute myocardial ischemia and during the coronary angioplasty it was shown that transient, spontaneous or induced by temporal coronary artery occlusion during transluminal balloon angioplasty myocardial ischemia leads to the significant «impairment» of SA-ECG parameters and LVP appearance. Stabilization of

patient's condition had correlation with the «improvement» of SA-ECG parameters and the disappearance of LVP in one-third of patients [6, 7, 8]. At the same time exercise-induced myocardial ischemia wasn't associated with the changes of SA-ECG parameters.

Areas with reduced blood supply, which include isolated necrotic cardiomyocytes or small focuses of necrosis, are the cause of fragmented impulse conduction. Patients with unstable angina in some cases have an increase in end-diastolic pressure, that can cause changes in electrophysiological characteristics of cardiomyocytes, inhomogeneity of the refractory periods, in particular, which can be the reason for micro-reentry and LVP development.

Stabilizing patient's with unstable angina condition is connected with the «improvement» of SA-ECG parameters and the disappearance of LVP in one-third of them. Antianginal pharmacological therapy or transluminal angioplasty does not change SA-ECG parameters or the frequency of LVP registration significantly in patients with stable CAD [7, 9]. It has been shown that CAD patients with unstable angina and ST segment elevation have LVP twice as often as patients with ST segment depression [6, 7].

According to Saveleva I.V. et al. LVP were detected in 25 %, 30 % and 37 % of patients with single-, double- and triple-vessel CAD respectively [7]. In other studies, a statistically significant predominance of LVP frequency was detected in a group of patients with triple-vessel CAD without focal myocardial changes; it is remarkable that the most sensitive parameter of SA-ECG was the duration of low-amplitude signals (LAS40) [6, 7], what can be explained by the increase of myocardium mass with electrophysiological inhomogeneity. Solmon A.J. et al. also revealed significant differences in the quantitative parameters of SA-ECG in patients with CAD and coronary artery stenosis compared to healthy individuals [22].

LVP were registered in 80 % of patients with chronic CAD, focal cardiosclerosis and ventricular tachyarrhythmias [23]. Brembilla-Perrot B. et al. [24] study included 58 patients with dilated cardiomyopathy and LVP were registered in 13 of 14 patients with induced and maintained VTA. The sensitivity of this method for risk factors estimation of VTA maintenance was high (93 %). LVP were also detected in 9 patients with induced trembling or fibrillation of ventricles.

LVP were 3 times more common in patients with stable VTA compared with CAD patients without ventricular rhythm disturbances (29 %) according to a number of authors [17, 20, 25–27]. However, the presence of high grading premature ventricle contractions didn't correlate with the increase of LVP detection.

According to many researches most patients with CAD and VTA had LVP during SA-ECG, which were connected to the focuses of cardiosclerosis, myocardial ischemia and loss of contractility. At the same time, it is possible to identify a group of patients with stable spontaneous VTA who has high frequency of LVP detection permanently. Typically, it is possible to induce VTA in these patients during electrophysiological study [7, 29, 30].

The study of Akasheva D.U. et al. proves the commonality of conditions for the emergence of LVP and VTA. The positive correlation between the presence of LVP and stable VTA induction during electrophysiological study has been demonstrated (the most sensitive parameter was LAS-40) [6]. Authors suggest to expand the possibilities of LVP in clinical practice, for example as a screening test to decide the question of intracardiac EPS necessity.

It is remarkable that patients with «labile» LVP during physical activity had VTA 5.4 times more often compared to the patients with «stable» LVP (27 % and 5 % respectively). Thus, the «lability» of LVP precisely reflects the electrophysiological processes that underpins the development of VTA during exercise.

However, it has been proved that LVP correlate with sudden cardiac death frequency, but only when the mechanism of sudden cardiac death is directly connected with VTA. Authors associate dangerous LVP with electrophysiologically induced VT. Incze A., Cotol S., Carasca E, [31] determined high prognostic value of LVP for predicting the risk of sudden cardiac death. 60 victims of MI aged over 51 year were included into the 5-year study, by the results of which LVP were determined only in 30 patients. During the investigation sudden cardiac death was registered in 6 patients and the episodes of VTA in 2 patients from the group with LVP. In the group without LVP sudden cardiac death wasn't registered. In case of Roithinger F.X., Punzen-gruber C., [32], Sanjuan R., Morell S., et al. [33] study it had been showed that LVP is a great predictor for sudden cardiac death and VTA development.

The great majority of works are dedicated to the analysis of LVP significance in estimation of fatal arrythmias development. Thus, it was shown that LVP increase the probability of life-threatening arrhythmias by 5 times and exceeding instrumental criterion (QRSd > 106 ms) increase the relative risk value by 9 times. Simpson M.B. et al. showed in their study a

positive correlation between life-threatening arrhythmias and LVP development. It is remarkable that the frequency of LVP is increasing with the class of ventricular tachyarrhythmia [29, 30]. The sensitivity of this method for ventricular arrythmia prognosis is about 86–92 % and the specificity varies from 62 to 97.5 % [23].

Gottfridsson C. et al. found that LVP are more often registered in patients with the history of monomorphic ventricular tachycardia than in the ones with ventricular fibrillation. The first group of patients was characterized by shorter duration of RMS40, totQRS and ejection fraction. Thus, the electro-anatomical substrate for LVP development is more pronounced in patient with VT.

Considering the data obtained, as well as the sensitivity of the temporal and spectral SA-ECG characteristics (for patients with monomorphic VT — 90 %, with EF — 58 %) and specificity (63 %), the authors suggest to use a combination of time and spectral SA-ECG analysis to identify patients with an increased risk of VT developing [34]. In the study of D. U. Akasheva et al., devoted to the study of LVP registration frequency in patients with induced VT with the programmable pacemaker, the sensitivity of LVP was 71 %, and specificity was 89 % [6].

Thus, after analyzing the data on LVP we can't confidently link them with the mechanism of sudden death. However, when sudden death is caused by VT directly, there is a significant correlation between dangerous LVP and VTA induced by programmed stimulation.

The work of Steinbigler P. et al. represents the importance of LVP prognostic value as a criterion for early detection and prevention of sudden cardiac death on the example of 756 AMI victims. However, the authors emphasize that the most informative pet-ameters in post-myocardial infarction risk estimation are LVP and LV EF [35]. According to Boldueva S.A. et al. LVP (as well as recurrent MI, hypotension during active standing test, LV EF, ventricular rhythm disturbances according to 24-hour ECG monitoring, heart rate variability) correlate with sudden cardiac death frequency [36].

The observation of Steinbigler P. et al. is very remarkable — MI victims after VT have LVP permanently during the day (regardless on the time of the day), whereas the patients after ventricular fibrillation (VF) have LVP transiently (in the mourning), which can be detected only with 24-hour monitoring [35]. Transient occurrence of LVP in patients with VF was accompanied by acceleration of heart rate in the morning, changes in the ST segment or transient decrease in heart rate variability. The variability of the appearance of trace potentials within 24 hours may depend on patient's activity and autonomic nervous system status and explains the fact that sudden cardiac death usually occurs in the morning or early afternoon hours.

At the same time, some authors during the examination of 1.800 AMI survivors, did not reveal any significant correlation between LVP, life-threatening arrhythmias and sudden cardiac death. The authors concluded that LVP have no significant prognostic value and can be limitedly used for risk-stratification of patients [37].

LVP prognostic value have been demonstrated in the work of Pozdnyakova. et al: during 18 months after AMI 36 % of patients had recurrent AMI, 32 % of patients with LVP died, 20 % of which had sudden cardiac death [18]. 48.9 % of patients with post-infarction cardiosclerosis (PICS) and unstable angina had LVP and stabilizing of their condition led to the decrease of LVP frequency in patients with non-Q wave myocardial infarction from 46.7 % to 13.3 %, and in patients with the history of Q-wave AMI from 50 to 47 % [18].

LVP in patients with PICS can indicate the development of rhythm disturbances, possible disease complications (early post-infarction angina due to return and persistence of ischemia or peri-infarction myocardial ischemia), reduction of myocardial contractility. According to S. Boldueva. et al. LVP in most cases correlated with sustained ventricular tachycardia (74.1 %), LV aneurysm (61.0 %) and reduced EF (52.5 %) [36]. In the work of Pozdnyakova N.V. et al. patients with PICS and LVP had significantly bigger end-stystolic and end-diastolic volumes than patients without LVP. Residual potentials were detected in 65.6 % of patients with LV dilatation. The frequency of recurrent AMI in patients with PICS and LVP was 19.4 % (4.9 % in the control group) and mortality was 32.2 and 8.3 %, respectively [18]. AMI survivors with LVP had early post-infarction angina in 20 % and acute left ventricular failure in 24 % of cases, whereas patients without LVP had these complications in 10.2 % and 10.2 % of cases, respectively.

Interesting data have been published in the study of overweight patients. Increased body mass as well as arterial hypertension, hyperinsulinemia and insulin resistance compose the metabolic syndrome, which increases the cardiovascular complication risk. In addition, arterial hypertension, diabetes mellitus and dyslipidemia, which usually accompany the obesity

are independent risk factors of sudden cardiac death. Lalani A.P. et al. registered LVP in 55% of patients whose body mass index (BMI) exceeded 30 kg / m^2 and showed that the frequency of LVP directly correlated with the Quetelet's index value. Thus, LVP were registered in 35% of patients with a BMI of 31–40 kg/m^2, 86% of patients with a BMI of 41–50 kg/m^2 and 100% of patients with a BMI> 50 kg /m^2 [38].

The possibilities of late ventricular potentials correction

The works that study the effects of stress test, physiotherapeutic methods of treatment and medication treatment on LVP are particularly important. The study of SA-ECG individual dynamics in patients with CAD showed that physical activity can lead to the occurrence or cessation of LVP regardless of myocardial ischemia. The instability of late potentials was greater in the subgroup with PICS than in the subgroup with the history of MI [6, 7]. LVP during the initial SA-ECG monitoring do not affect the results of the stress test: ST-segment depression was registered in patients with chronic CAD with the same frequency as in patients with or without LVP.

There are many researches evaluating pharmacological and non-pharmacological VTA treatment effectiveness. The works on the LVP changes and effectiveness of VTA comparison of treatment are much more uncommon.

According to Boehrer J.D., Glamann D.B., et al. [39], even temporarily performed restoration of blood flow in the arteries that supply the infarction area in patients with acute myocardial infarction may decrease the frequency of future arrhythmias and the risk of sudden cardiac death. The study was conducted on 54 patients with AMI and angiographically confirmed coronary artery occlusion during the first 5 hours. 35 patients who underwent reperfusion (p = 0.038) had a significant decrease in the frequency (by 50%) of LVP occurrence. On the other hand, 19 patients after successful thrombolysis: 8 of 19 patients (42%) during the first 120 minutes of thrombolysis and 7 of 19 patients (37%) after the procedure did not show a significant decrease. Despite the successful performance of thrombolysis LVP persisted or appeared for the first time in 8 of 54 patients (15%). Thus, it can be concluded that successful thrombolysis decreases the frequency of late potentials during SA-ECG, but the sensitivity and specificity of this method is not enough to control the coronary flow during the post-occlusive period.

The frequency of LVP decreased significantly after systemic thrombolysis – from 68.6% during the first 24 hours to 31.4% (on the 10^{th} day) and to 11.4% (by the end of admission), whereas patients who did not underwent thrombolysis for some reason had LVP in 69%, 48.3% and 41.4% of cases respectively [37].

The influence of anti-anginal and antiarrhythmic therapy on the LVP development have been studied in many works [6, 7]. The patients with unstable angina after stabilization of their condition showed the «improvement» of some SA-ECG parameters and the decrease of LVP frequency, what can be connected with the decrease of myocardial electrophysiological inhomogeneity due to perfusion.

78 patients with recurrent VTA and CAD underwent intraoperative registration of LVP frequency in order to determine the scope and clinical significance of epicardial LVP. The ECG averaging was performed in 30 patients. Correlation of VT focus localization and the area of epicardial LVP development was not determined in 4 patients. On the other hand, the place of LVP development was in the immediate vicinity to the origin of VT in 5 patients (3 of them had polymorphic tachycardia). 76% of patients had low-amplitude LVP. 24 patients without post-operative VT showed the reduction of QRS complex duration (from 137±27 to 121±26 ms; p=0.003), the increase of the QRS complex voltage (from 16.5±16.1 to 39.0±29.4 mkV; p=0.003) and the decrease in LVP frequency (from 71% to 33%; p=0.03) after the angioplasty. Filtered QRS complex remained the same in 13 patients with post-operative VT. The absence of LVP after the surgery in 9 out of 10 cases correlated with the absence of VT (p<0.02) [40].

Early administration of pravastatin in patients with AMI reduces the risk of LVP and the probability of ventricular rhythm disturbances [36]. The frequency of LVP registration decreased from 52% to 16% during standard therapy in patients with unstable angina [36].

According to some data the frequency of LVP in patients with chronic CAD at the initial state and before the discharge was 31% and 25%, respectively. Comparing the quantitative criteria of LVP at the initial state and after the therapy no significant difference was found [6, 41]. This investigation confirms the inconsistency of data on the significance of LVP in patients with chronic CAD.

According to Akadysheva D. U. et al. IA, B, C classes of antiarrhythmic medications and amiodarone do not influence LVP parameters [6]. However, other works showed that amiodarone has positive influence

on SA-ECG parameters: as electro- impulse therapy, lidocaine or mexiletine administration suppressed the paroxysm of ventricular tachycardia, LVP succeeded after amiodarone administration [41]. The influence of beta-blockers (atenolol, bisoprolol, sotalol) on the electrical activity of ventricles have been studied: before the medication administration LVP have been detected in 68.6% of AMI survivors and by the 30th day only in 14.3%.

Consequently, many researches demonstrated high influence of LVP on the course of CAD and the estimation of therapy effectiveness. Coronary reperfusion and function of ischemic but viable cardiomyocytes improves and electrophysiological inhomogeneity of myocardium reduces during the medication treatment in patients with unstable angina and AMI.

The frequency of LVP registration significantly reduces after surgical revascularization and myocardium perfusion improvement (which can lead to the reduction of ischemic zone that is the main cause of LVP). In case of persistent ischemia (unsuccessful revascularization) LVP also stay permanent.

Conclusion

Late ventricular potentials can be the predictors of deterioration in the condition of the patient (CAD exacerbation), AMI development, rhythm disturbances, chronic heart failure progression or death. Dynamic LVP detection including the period of medication treatment can be used to prognose the course of CAD and to estimate the effectiveness of the therapy according to some investigations.

LVP are more effective when determined in combination with a number of structural, hemodynamic and functional parameters (systolic and diastolic LV function, heart rate variability, etc.) due to its uncertain predictive value when used separately. It is also possible to use the combination of time and spectral analysis of SA-ECG. Further research is needed in this area to clarify which analysis (time, spatial or spectral) is the most preferred to determine the LVP and which parameters for totQRS, RMS40, LAS-40 are more diagnostically accurate.

Such investigations can make the prognosis the undesirable disease development more accurate (including VTA and sudden cardiac death) and may identify the group of patients with high risk of sudden cardiac death. Pre-syncopal phase with unclear etiology, the history of SCD can be the signs to conduct SA-ECG.

SA-ECG can also be used separately as a method of early, pre-symptomatic and preclinical diagnosis of myocardial lesion due to several somatic diseases and as a method of estimation of cardiotoxic and pro-arrhythmogenic effects of some medications.

Consequently, LVP registration is an affordable, non-invasive method in diagnosing and predicting VTA in patients with acute myocardial infarction and other types of coronary artery disease, cardiomyopathies, patients with SCD risk, which can reduce the risk of sudden cardiac death if used widely. However, nowadays further investigations are needed to determine the range of diseases when it is reasonable to use this method to clarify the sensitivity, specificity and prognostic value of it, including the studies on applying LVP dynamics as the way to determine the effect of pharmacological and non-pharmacological treatment and surgical interventions.

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