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EPICARDIAL VOLTAGE MAPPING IN PATIENTS WITH POSTINFARCTION VENTRICULAR TACHYCARDIA: A PILOT STUDY

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Introduction. Radiofrequency ablation (RFA) is an established treatment of post-myocardial infarction ventricular tachycardia (VT). Endocardial VT ablation can be insufficient for VT termination when the scar is intramural/epicardial.

Purpose: to assess the extent of epicardial electrophysiological VT substrate in patients with remote myocardial infarction.

Materials and methods. Thirteen patients with sustained postinfarction VT, who signed an informed consent, were included into the study. All patients underwent full clinical evaluation. Electroanatomical voltage bi- and unipolar mapping of endocardial and epicardial surfaces was performed. Maps were evaluated for the presence of low-voltage areas and local abnormal ventricular activity (LAVA). RFA was performed at LAVA sites. The end-point of the procedure was scar LAVA abolition and VT noninducibility (procedure success). VT recurrence was detected using an implantable cardioverter-defibrillator and/or ECG monitoring.

Results. Epicardial access was successful in 12 patients. Epicardial access was performed at a first procedure in 7 patients, 4 patients had a history of previous endocardial ablation. Epicardial LAVA sites were detected in 9 patients. Endocardial and epicardial arrhythmogenic substrate localization coincided in 8 patients. One patient had only epicardial scar, 1 patient had only septal endocardial scar. In one patient LAVA sites had different localizations on epicardial and endocardial maps. Acute ablation success was noted in 12 patients.

Conclusion. In our patient group transmural scar and epicardial electrophysiological arrhythmogenic substrate was detected in 82% of cases. Isolated endocardial ablation may be unsuccessful, in such cases epicardial mapping and ablation might be useful.

Key words: postinfarction cardiosclerosis; ventricular tachycardia; radiofrequency catheter ablation; endocardial access; epicardial access; scar tissue; late potentials; mapping

Conflicts of interest: E.N.Mikhaylov and D.S.Lebedev report receiving speaker and consultation honoraria from Biosense Webster; other authors report no conflicts of interest.

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Sustained ventricular tachycardia (VT) - is one of the most important causes of sudden cardiac death in patients with structural heart disease [1]. Antiarrhythmic drug therapy is frequently limited due to VT reccurence and side effects [2], whereas radiofrequency catheter ablation (RFA) demonstrates a higher efficacy in VT treatment in patients with remote myocardial infarction [3, 4].

Different RFA approaches have been proposed for VT substrate modification: mapping and ablation of critical isthmus of each induced VT, scar dechannelling, ablation of local abnormal ventricular activity (LAVA) sites [5-8], core scar isolation, and complete scar homogenization.

It should be acknowledged that acute VT non-inducibility and long-term freedom from VT recurrence cannot be achieved in all cases. Numerous observations have shown failure of RFA in case of intramural/subepicardial arrhythmogenic substrate location when ablation is performed endocardially via vascular access. RF energy penetration depth is about 4-6 mm, and generally it doesn't reach out subepicardial myocardial layers [9, 10]. Thereby a transcutaneous access has been proposed for epicardial mapping and ablation of VTs [11].

Most frequently, epicardial ablation is required for VT treatment in non-ischemic heart disease, when substrate is characterized by intramural or subepicardial location: arrhythmogenic right ventricle cardiomyopathy, dilated cardiomyopathy, hypertrophic cardiomyopathy (HCM), and other [12].

Regarding post-myocardial infarction VT, subepicardial localization of a critical isthmus has been located in 10% of patients, which might require an epicardial access for successful ablation. There have been few studies published that advised combined endo-epicardial ablation for successful VT treatment in patients with remote myocardial infarction [3, 11].

The prediction of arrythmogenic substrate subepicardial location is frequently limited. Magnetic resonance imaging (MRI) is a useful tool in delineation of the depth and extent of a scar, but its usability is limited in patients with an implanted ICD/CRT-D. Moreover, ECG criteria for epicardial VT exit site prediction have less accuracy in the presence of a postinfarction scar.

Theoretically, a combined endo-epicardial approach can improve the long-term efficacy of VT catheter ablation in some patients.

The aim of our pilot study was to evaluate the prevalence of electrophysiologically mapped epicardial VT arrhythmogenic substrate, and the necessity of epicardial ablation in patients with remote myocardial infarction.

MATERIAL AND METHODS

Between 2015 and 2018 165 patients with structural heart disease (coronary heart disease, dilated cardiomyopathy, arrhythmogenic right ventricle cardiomyopathy, infiltrative heart disease) were referred for VT catheter ablation at the Almazov Medical Centre, and 59 had remote myocardial infarction. The study group comprised patients who signed informed consent for epicardial access (study and agreement form were established by ethical committee of the Almazov NMRC, protocol №181 14.12.2015). One patient included into study group was operated in the Sukhanov Federal Centre for Cardiosurgery.

Inclusion criteria were the following: a postinfarction scar detected by transthoracic echocardiography or MRI and ECG-criteria of myocardial scar, the presence of sustained VT registered on ECG or during ICD follow-up, ineffective antiarrhythmic drug therapy, the absence of indications for revascularisation or it's impossibility, signed informed consent for epicardial access. Exclusion criteria: myocarditis, previous cardiothoracic surgery with pericardium dissection, other acute inflammatory disease, three component antithrombotic therapy without possibility to discontinue two of them, acute myocardial infarction <3 months ago, acute coronary syndrome during screening.

Standard clinical evaluation included: 12-lead rest ECG, TTE, 24-hour Holter monitoring, coronary angiography, ICD/CRT-D check-up. Heart MRI was performed in two patients before the procedure. In three patients VT manifested as VT storm, they were operated urgently.

Endocardial and epicardial accesses, electrophysiological study, catheter ablation

Procedure was provided in an EP laboratory under general anesthesia. Vascular accesses were performed using the Seldinger technique: to the right femoral artery and right femoral vein. Percutaneous pericardial access was obtained by a subxyphoid puncture. The access technique was described in detail previously [13]. The long Preface Multipurpose sheath (Cordis,USA) was introduced into the pericardial space. Then interatrial septum punctured under fluoroscopic guidance using the Brockenbrough needle (BKR-1, St. Jude Medical, USA). The transseptal sheath (Preface Multipurpose, Cordis, USA) was positioned in the left atrium and left ventricle. Therefore, a double endocardial access to LV was used. Heparin was administered as intravenously 80-100ME/kg with further ACT monitoring, with a target value >250 sec. A quadripolar diagnostic catheter (Webster, Biosense Webster, USA) was positioned in the RV apex. The procedure was performed under the guidance of the three-dimensional navigation system (Carto 3, Biosense Webster, USA). The NaviStar Thermocool or SmartTouch (Biosense Webster, USA) ablation catheters were used for mapping and ablation. The multipolar Pentaray catheter (Biosense Webster, USA) was used for epicardial mapping in two cases. Endocardial three-dimensional LV reconstruction and electroanatomical voltage mapping was performed in all patients (cut-off values 0.5-1.5 mV for bipolar signals), then epicardial electroanatomical mapping was performed with the same cut-off values. For unipolar maps cut off values 5.0-9.0 were used. Sites with local registration of late potentials, fragmented and double potentials were tagged on maps. When a scar was localized in the interventricular septum and VT remained inducible despite endocardial ablation, then RV mapping and substrate ablation was performed. Electroanatomical voltage mapping was performed during sinus rhythm or RV pacing. A scar was defined when myocardial signal amplitude was <0.5 mV, the intact myocardium was defined when myocardial signal amplitude was >1.5 mV.

The identification of VT exit site location was performed by both, activation and pace-mapping, and according to effective ablation. Arrhythmogenic substrate surface area was evaluated on bipolar and unipolar voltage maps. Protocol of EP study for VT induction included programmed stimulation up to three extrastimuli from the RV apex, RV ouflow tract, from LV, and overdrive burst pacing was performed when VT was not induced by programmed stimulation. Pace-mapping of conduction channels in the scar, entry and exit zones VT was performed. When induced VT was haemodynamically stable, entrainment-mapping performed. Selective coronary angiography used before epicardial ablation in order to de-

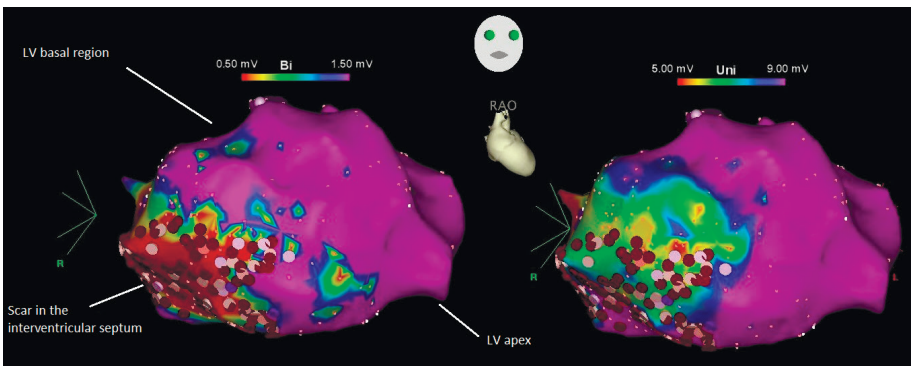

Figure 1. Patient №5. Electroanatomical voltage map with the endocardial scar only in the interventricular septum. RAO projection. The bipolar substrate area dominates over the unipolar map. Bipolar map cut off values 0.5-1.5 mV, unipolar cut off values 5.0-9.0 mV. Red color indicates the myocardial signal amplitude <0.5 mV, purple color indicates the myocardial signal amplitude >1.5 mV.

fine the proximity of coronary artery and to prevent their damage. Safety distance for ablation from the coronary artery was considered about 10 mm. RF energy was delivered at areas with late potentials, fragmented potentials until their abolishment or decreased amplitude by 85%. Entry and exit VT sites and conducting channels were ablated. When a VT cycle was mapped, the critical isthmus of tachycardia was ablated.

RF energy 40-50W was used, application duration - 10-40 sec, ablation catheter tip irrigation was 30 ml/min. Ablation was considered effective if late potentials and fragmented potentials vanished and loss of stimulation capture was achieved (amplitude of stimulation 10 V, time duration 1 ms, cycle length - 500 ms). After substrate ablation programmed stimulation for VT induction was performed. Acute procedure effectiveness was considered when VT was non-inducible.

The mean follow-up period was 20.2±16.1 months (from 2 to 46 months). VT recurrences were documented by ICD/CRT-D regular check-ups, and 24-hour Holter monitoring.

Statistical analysis

Continuous data with normal distribution were reported as mean ±s tandard deviation, compared by T-test. Categorical variables were expressed using non-parametric statistics, median with interquartile range (IQR). Mann-Whitney test and Fischer exact test were used for comparison non-parametric variables. Results were considered significant with a P-value <0.05. Statistical analysis was provided using STATISTICA 6,0 (StatSoft, Tulsa, USA).

RESULTS

Patient clinical characteristics

The study group included 13 patients, the mean age 58.1±9.8 years (12 men). Clinical characteristics are presented in table 1. Antiarrhythmic drug (AAD) therapy with a combination of amiodarone and beta-blockers was used in 9 patients, in 3 patients amiodarone was discontinued because of complications, and only beta-blockers were prescribed. In one patient without ICD AAD therapy was limited by symptomatic sinus bradycardia (nebivolol 2,5 mg per day). In nine patients the combined endo-epicardial access was used as a first-line approach. Endocardial ablation of post-infarction VT was previously performed in four patients, procedures were ineffective or with temporary effect (1-2 attempts). In one patient with two previous ineffective attempts of endocardial VT ablation three consecutive epicardial procedures were performed due to clinical VT recurrence. Final ablation was successfully performed using bipolar ablation because of intramural location of the tachycardia critical zone. Transmural postinfarction scar location was detected by cardiac MRI in two patients. Epicardial access was obtained in 12 of 13 patients, in one patient it was unsuccessful, presumably because of pericardial adhesions on diafragmatic surface of the heart. In one case comparative analysis of endocardial and epicardial electroanatomical voltage maps was not performed because of a technical failure. Thus, comparative analysis of endo - and epicardial voltage maps was performed in 11 patients.

Mapping and ablation

Electroanatomical voltage maps were created with the color threshold 5-10 mm (when an ablation catheter was used for mapping) and the color threshold 2 mm (when the multielectrode catheter was used for mapping). The mean epicardial mapped surface area prevailed above endocardial surface area because epicardial mapping included evaluation of both ventricles.

The prevalence of a median substrate surface area of unipolar voltage maps over bipolar voltage maps by 3.7 times was noted on the endocardial surface (45.8

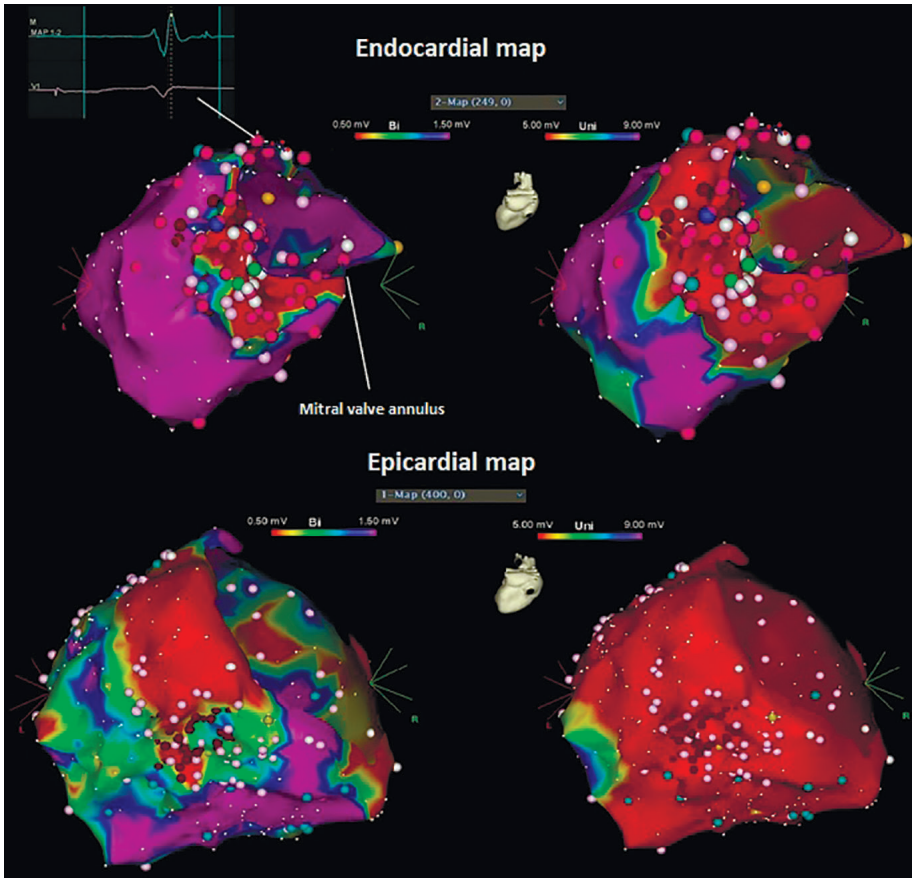

Figure 2. Patient №1. Endocardial and epicardial maps (bipolar and unipolar). Posterior projection. Bipolar map cut off values 0.5-1.5 mV, unipolar cut off values 5.0-9.0 mV. Pink tags represent sites with late potentials. The epicardial abnormal electrogram area prevails above the endocardial area. The unipolar low voltage area is more extensive than bipolar. The wide inferior myocadial involvement is seen on the unipolar map, while the bipolar map shows mainly lateral wall involvement.

(IQR:17.1;86.5) cm^2 vs 11,8 (IQR: 2.0;31.6) cm^2; p=0.035) (Table 2). Only in one patient the bipolar substrate area dominated over the endocardial unipolar map (by 2,5 times). There was no any abnormal electrical activity or low voltage signals registered on the epicardial surface. In one case there was no low-voltage substrate on the endocardial bipolar map, and was hardly represented on the unipolar map.

The median epicardial arrhythmogenic substrate area on the unipolar map prevailed over the same on the bipolar map by 2.3 times: 107.7 (IQR: 84.3; 168.9) cm^2 versus 46 (IQR: 15.9; 55.5): p=0.041 (Figure 1). Low-voltage areas were not found on the epicardial map in one patient (Figure 2); in two cases the endocardial substrate area was wider than epicardial arrhythmogenic substrate (table 2). We found no correlation between the substrate area on the endocardial and epicardial surfaces.

Table 1.

Patient clinical characteristics

Parameter	Value
Remote myocardial infarction, n (%)	13 (100)
Hypertension, n (%)	10 (76,9)
Diabetus melitus, n (%)	3 (23)
COPD, n (%)	1 (7,7)
Atrial fibrillation, n (%)	6 (46,2)
ICD, n (%)	9 (69,3)
CRT-D, n (%)	2 (15,4)
Patients with remote SCD, n (%)	10 (76,9)
ICD shock, n (%)	4 (30,8)
External shock, n (%)	7 (53,8)
Mean LV EF, %	38,8±10,6
Mean LV EDV, ml	193,8±73,7
Mean LV ESV, ml	125,3±54,9
Coronary angiography	
Without HSS, n (%)	9 (69,3)
PTCA and stenting, n (%)	5 (38,5)
Repeated PCI, n	2 of 5 patients
CABG, n (%)	0
TTE, scar localization	
Inferioir wall, n (%)	10 (76,9)
Lateral wall, n (%)	7 (53,8)
Apex, n (%)	2 (15,4)
Septum, n (%)	4 (30,8)
Anterior wall, n (%)	2 (15,4)

Description. COPD -chronic obstructive pulmonary disease, ICD - implantable cardioverter-defibrillator, CRT-D - cardiac resynchronization therapy defibrillator, LV EF - left ventricle ejection fraction, LV EDV - left ventricle end-diastolic volume, LV ESV - left ventricle end-systolic volume, HSS - hemodinamically significant stenosis, PTCA - percutaneous transluminal coronary angioplasty, PCI - percutaneous coronary intervention, SCD - sudden cardiac death, TTE - transthoracic echocardiography, CABG - coronary artery bypass grafting.

Late potentials and fragmented potentials were registered in nine patients: in seven cases - on the endocardial surface, and in six cases- on the epicardial surface. The areas of late potential registration in study patients is presented in table 2.

Two VT morphologies were induced before ablation in 4 patients, in 4 patients only one clinical VT morphology was induced, and in 4 patients VT was non-inducible.

Endocardial and epicardial scar location coincidence was noted in 8 cases. In one patient a scar was detected on the epicardial surface only. In one patient the postinfarction scar was identified endocardially only in the interventricular septum. In one case, there were different endocardial and epicardial scar localizations: an endocardial scar was identified on the inferior and septal walls, an epicardial low voltage activity was detected on the lateral RV wall.

Fragmented potentials and late potentials were detected in 9 patients, RF ablation was performed in these areas in all cases.

The mean procedure time duration was 228±62 minutes, the mean fluoroscopy time duration was 45±21 minutes. In 12 patients VT was non-inducible at the end of procedure, in two of them ventricular fibrillation was induced by an aggressive stimulation protocol. Thus, in 12 of 13 cases complete acute effect was achieved. In one patient the procedure was discontinued because of haemopericardium occurrence.

Complications

In one patient with unsuccessful epicardial access (there were adhesions on the inferior wall of LV), an attempt of transseptal puncture was performed with cardiac perforation and haemopericardium, which required surgical correction. There were no complications, associated with the epicardial access itself.

Long-term results

The mean follow-up period was 19.3±17.6 months. Two of 11 patients, who were operated using the epicardial access, were lost to follow-up. In three patients, the follow up period was less than 6 months; VT recurrence was not evident during this period of observation. In one patient with multiple ablation sessons (6 procedures in total) VT recurrence was not registered during 3-years follow-up. In one patient VT recurrence registered 1 year after the procedure: 11 VT paroxysms with ATP therapy registered and 1 shock because of acceleration VT to VF after ATP therapy at two years. Two of four patients with previous ineffective endocardial VT ablation were free from VT recurrence after combined endo-epicardial VT ablation.

DISCUSSION

VT substrate can be localized on both endocardial and epicardial surfaces in patients with remote myocardial infarction. In our pilot study among 11 patients with sustained postinfarction VT, electrophysiological VT substrate was identified epicardially in 82% of cases. Some patients with previous ineffective attempts endocardial ablation VT substrate epicardial mapping and

ablation is an advisable approach, as it was noted in two of four patients.

In 2000 Sosa E. and colleagues published results of their study of epicardial mapping VT substrate in patients with remote inferior myocardial infarction. Subepicardial myocardium involvement in re-entry loop was demonstrated in 7 of 30 registered VT (23%) for 7 of 14 patients [11]. The presence of an epicardial substrate in patients with postinfarction VT was noted in the research published by Brugada J. and colleagues [3]. It has been demonstrated that unipolar scar surface area prevailed over the same on the bipolar map, that could be evidence of epicardial localization VT substrate [14]. In our research, we obtained similar findings.

The combined endo-epicardial approach demonstrates effectiveness in long-term period as a first-line approach, and after previous endocardial ablation as well. Recently, the application of the combined endo-epicardial access in patients with VT recurrence with previous ineffective endocardial VT substrate ablation was published [3, 15]. Nowadays, a multicenter randomized EPILOGUE clinical study is going, where patients with postinfarcton VT are allocated into the group of first-line combined access or into the group of sequential endocardial approach followed by epicardial ablation one a VT recurrence is detected [16].

The scar area is a target for radiofrequency ablation, it is defined on the bipolar map as myocardial signal <0.5 mV, zones with abnormal electrical activity (late potentials), as described by Nakahara S and Tung R and co-workers [17] for epicardial VT substrate in patients with ischemic cardiomyopathy. In this study late potentials were detected on both the epicardial surface and endocardial surfaces. Late potential elimination was associated with effective epicardial ablation.

Our findings comfirm previous results by Schmidt B et al. [18]. The authors demonstrated that 30% of patients with postinfarction VT, who were ablated repeatedly with the epicardial access, had both endocardial substrate and epicardial substrate, epicardial substrate only was detected in 30% of patients at the second procedure. In our study, 7 of 11 patients who underwent the combined endo-epicardial ablation had a coincidence arrhythmogenic substrate localization on the endocardial and epicardial surfaces. In four of 11 patients previous endocardial VT ablation was carried out.

The necesserity of epicardial access can be expected during the invasive procedure when an endocardial unipolar low-voltage area map prevails over bipolar low-voltage are; when ECG criteria of epicardial localization of induced VT are present; when there is a lack of low-voltage are on the endocardial map, or excessive endocardial ablation is ineffective despite appropriate exit site or isthmus mapping [19, 20].

CONCLUSIONS

In our patient group with remote myocardial infarction and indications to VT catheter ablation, 82% of cases demonstrated transmural scar with electrophysiological signs of arrhythmogenic substrate on the epicardial ventricular surface. Subepicardial myocardium involvement should be suspected during endocardial mapping, when there is a prevalence of local abnormal electrical activity area on the unipolar map over the area on the bipolar map, and in cases of endocardial ablation failure.

We suggest that epicardial mapping and ablation is useful/advisable in some patients with postinfarction VT, who should be referred to centers experienced with complex electrophysiological procedures and epicardial ablation.

Table 2.

Voltage mapping

Patient number	Endocardial surface					Epicardial surface				
	Bipolar signals			Unipolar signals		Bipolar signals			Unipolar signals	
	<0.5 mV	>0.5-<1.5 mV	LP	<0.5 mV	>0.5-<1.5 mV	<0.5 mV	>0.5-<1.5 mV	LP	<0.5 mV	>0.5-<1.5 mV
1	8.6	6.0	25.2	45.8	14.6	54.6	315.2	0	417.1	23.7
2	2.3	0	0	2.6	25.0	46.0	2.0	49.8	77.5	64.4
3	1.7	7.5	4.1	27.8	22.9	49.9	26.1	19.8	122.1	45.0
4	11.8	12	44.1	81.6	23.7	19.7	29.7	0	574.1	45.2
5	21.9	23.8	0.7	8.6	49.6	0	37.3	0	0	17.1
6	0	0	3.5	2.0	7.6	56.4	35.0	47.2	125.0	40.1
7	66.8	63.5	18.3	88.9	55.9	124.5	650.7	29.3	212.8	569.7
8	344.5	91.2	22.4	84.1	93	166.8	439.4	30.5	91.1	487.9
9	28.6	22.3	0	109.2	195.7	19.5	39.7	0	99.6	19.4
10	47.3	172.6	52.7	152.2	159	2.4	19.5	19.5	107.5	491.8
11	0	0	0	25.5	64.4	12.3	32.1	9.3	35.4	42.9
Median (1 и 3 quartile)	11.8 (2.0; 31.6) *	12.0 (3.0; 43.7)	4.1 (0.4; 23.8)	45.8 (17.1; 86.5) *	49.6 (23.3; 78.7)	19.6 (12.3; 53.8) *	36.2 (30.3; 236.4)	14.4 (0; 29.7)	95.4 (52.2; 120.6) *	54.8 (40.8; 460.5)

Description. LP - late potentials, * - p<0.05.

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