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## ECG Holter monitoring and heart rate variability in patients with post-infarction left ventricle aneurysm

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### Abstract

Sudden cardiac death (SCD) is a death from cardiac causes, which occurred within hours of onset of symptoms [6]. The most common immediate cause of SCD is ventricular tachycardia, and patients with post-infarction ventricular aneurysm (PLVA) could be a higher risk group of it. That's why the aim of our research was to study peculiarities of ECG monitoring and heart rate variability in patients with PLVA. All patients underwent ECG monitoring, which was performed by the standard method [4, 7] using the Holter-system "Cardiosens K". We analyzed standard parameters along with heart rate variability (HRV) data.

In our research in summary we stated that such patients are characterized by increased sympathetic activity and higher humoral influences, prevalence of peripheral effects over the central ones, as well as significantly longer duration of QT interval. The use of CABG increases the rate of ectopic complexes of supraventricular and ventricular origin. The patients with PLVA and previous PCI have, in this context, the most favorable course of the disease.

**Keywords:** Heart rate variability, ECG Holter monitoring, left ventricle aneurysm.

### Introduction

Sudden cardiac death (SCD) is a death from cardiac causes, which occurred within hours of onset of symptoms [6]. To estimate the true prevalence of this phenomenon is difficult, but it is believed that in the US alone about 250-400 thousand people die each year from SCD [1, 4, 6]. For example, in a study performed in 1998, stated 456 076 deaths during the year; all persons were over age 35 [8]. Well known that SCD risk increases with age, and it is two to three times higher in men. Causes of SCD are extremely diverse, but the structural abnormality of the heart is thought to be the leading one. However, often it is a functional impairment that leads to formation of malignant ectopic beats or to conduction disturbances and development of lethal bradyarrhythmias or asystole [4, 6].

The most common immediate cause of SCD is ventricular tachycardia. Thus, in a recent study with the inclusion of 157 patients who wore ECG Holter register system, 62% of people died due to of VT, which transformed into VF. Only 8% had firstly developed VF, and 13% – "torsades de pointes" ventricular tachycardia [2]. Bradyarrhythmias and ventricular asystole had been ascertained in 17% of individuals. SCD is often quite difficult to distinguish from several other clinical conditions such as aortic dissection, pulmonary embolism, pericardial tamponade and others. In such a case a history and eyewitness accounts come first. Also so called predictors play an important role in the differentiation of SCD, some of which we investigated in our research.

The aim of our research was to study peculiarities of ECG monitoring and heart rate variability in patients with PLVA.

### 2. Methods

We analyzed results of 24-hour ECG monitoring of 238 patients with post-infarction left ventricular aneurysm (PLVA) who met the inclusion criteria and exclusion. The main group was divided into 3 subgroups. The first subgroup included 134 persons with PLVA who received only optimal basic therapy (OBT). The second subgroup included 56 patients with PLVA, who along with OBT underwent percutaneous coronary intervention (PCI). The third subgroup included 48 patients with PLVA after coronary artery bypass grafting (CABG). The control group consisted of 36 patients after myocardial infarction (MI) without PLVA. All patients of the research and control groups were relevant for the age and gender distribution.

All patients underwent 24-hours standard ECG monitoring [3, 5]. The ECG monitoring was performed by the standard method [4, 7] using the Holter-system "Cardiosens K". Also we analyzed standard parameters along with heart rate variability (HRV) data.

### 3. Results

Data of 24-hours ECG monitoring are presented in table 1.1. First it should be noted that patients with PLVA had confirmed tendency to tachycardia. Thus, for example, in patients of the first research subgroup during monitoring about 173.7±34.8 episodes of tachycardia were stated. In the second and the third subgroups there were 112.9±26.4 and 144.6±20.9 episodes, respectively. In comparison, patients of the control group had only 55.1±6.2 episodes,  $p < 0.05$ . Interestingly, the frequency of bradycardia episodes differed significantly only in comparison of control group to the second research subgroup (the patients with PLVA and PCI) and to the third subgroup (persons with PLVA and CABG).

Table 1.1. Patients distribution by results of ECG 24-hours monitoring (M±m).

EKG phenomena	Control group (n=36)	Research groups (n=238)		
		subgroup 1 (n=134)	subgroup 2 (n=56)	subgroup 3 (n=48)
average HR	68.6±5.4	92.7±6.3	82.3±4.7	73.9±5.1
bradycardia episodes	12.5±3.4	23.7±5.5	36.3±8.5*	32.7±7.2*
tachycardia episodes	55.1±6.2	173.7±34.8*	112.9±26.4*	144.6±20.9*
bradycardia, min/d	78.2±9.3	63.7±7.9	77.6±13.2	89.1±11.1
tachycardia, min./d	117.7±21.1	328.4±36.2*	187.3±28.2*	212.3±22.1*
sinus rhythm, %	93%	78%	84%	83%
non-sinus rhythm, %	7%	22%	16%	17%
total beats	123 284±2496	212 348±4592*	178 367±4395**	165 823±4623**
SVE, number (%)	4592±329 (3.7%)	18123±1726* (8.5%)	11328±947** (6.4%)	22385±2674** (13.5%)
VE, number (%)	5298±628 (4.3%)	27236±4263* (12.8%)	19347±3852* (10.9%)	29368±5347* (17.7%)
Abberant complexes (%)	6752±358 (5.5%)	34012±6235* (16.0%)	21745±4368* (12.2%)	33375±3298* (20.1%)

Notes: 1. Average data shown; 2. \* - standard deviation between control and research subgroups,  $p < 0.05$ ; 3. \*\* - standard deviation to the first research subgroup,  $p < 0.05$ .

Thus, in the control group averagely 12.5±3.4 bradycardia episodes were recorded, in the second and the third research subgroups – 36.3±8.5 and 32.7±7.2 episodes, respectively,  $p < 0.05$ . According to these parameters, total duration of bradycardia as well as tachycardia episodes changed. Thus, for example, the longest duration of daily tachycardia was characteristic of the first research subgroup. In this subgroup it lasted 328.4±36.2 minutes/day, which was almost three times longer compared to controls, 117.7±21.1 min./d.,  $p < 0.05$ . Interestingly, that tachycardia duration in the second research subgroup was the lowest,  $p < 0.05$  (compared to the first one). It should also be noted that the duration of bradycardia episodes per day was almost the same as in patients with PLVA (research subgroups) and without aneurysm (control group). Also the reduction of sinus rhythm time in patients with PLVA was stated, and it was the lowest in patients of the first research subgroup, 78%. In comparison, it was 93% in patients of the control group, and only 7% complexes were of non-sinus origin. Patients of the second and the third subgroups had sinus rhythm 84% and 83%, respectively. Analysis of complexes of non-sinus origin revealed a high occurrence of ectopic complexes in patients of the first and the third research subgroups. For example, there were 4592±329 SVE (supraventricular extrasystoles) per day in the control group, in the third subgroup were several times more (22.385±2.674/ d), and in the first one – 18 123±1726/ d,  $p < 0.05$ .

The occurrence of ventricular extrasystoles (VE) was the greatest in the third research subgroup (individuals with PLVA and after CABG procedure) and stated 17.7%. For comparison, in was only 4.3% in the control group. Individuals of the second research subgroup (individuals with PLVA and after PCI) had the lowest percentage of them among all research subgroups, 10.9%. It should also be noted the high percentage of aberrant complexes in patients of the first and the third research subgroups.

Heart rate variability (HRV) data analysis showed that data distribution depends on treating tactic (table 1.2). Firstly, the QT interval is significantly longer in patients with PLVA. For example, in the control group QT interval was 418.9±18.3 ms, in the first research subgroup (and it was the longest among all research subgroups) it was 523.3±24.5 ms,  $p < 0.05$ .

Table 1.2. Distribution of heart rate variability data of control and research subgroups during the active period (M±m).

Parameters	Control group (n=36)	Research groups (n=238)		
		subgroup 1 (n=134)	subgroup 2 (n=56)	subgroup 3 (n=48)
QTc, ms	389.1±16.5	492.3±14.7*	437.1±12.8*	456.7±21.1*
QT, ms	418.9±18.3	523.3±24.5*	444.8±17.3**	492.7±24.3*
TP, ms <sup>2</sup>	3592±274	5132±665*	3828±278	4919±125*
HF, mc <sup>2</sup>	1073±51.4	713±43.1*	436±21.7**	592±17.4**
HF norm, % of TP	29.9±2.2	13.9±1.9*	11.4±1.2*	12.1±1.5*
LF, ms <sup>2</sup>	1753±38.9	3103±93.6*	2215±59.8**	3295±45.4*
LF norm, % of TP	48.8±2.4	60.1±5.7	57.9±4.3	66.9±3.2*
VLF, ms <sup>2</sup>	767±27.1	1318±39.2*	1176±24.6**	1056±23.3**
VLF norm, % of TP	21.2±1.9	26.0±1.5	30.7±1.7**	21.2±1.2**
LF/HF	1.6±0.12	4.4±0.13*	5.1±0.17**	5.7±0.12**
SI, units	92.1±3.27	33.8±1.12*	55.4±2.19**	68.4±3.38**
IC	3.9±0.2	2.9±0.3*	2.3±0.1*	3.7±0.4

Notes: 1. absolute numbers shown; 2. \* - standard deviation between control and research subgroups,  $p < 0.05$ ; 3. \*\* - standard deviation to the first research subgroup,  $p < 0.05$ .

The total effects (TP) of the autonomic nervous system prevailed, mostly in patients of the first and third research subgroups. For example, in patients with PLVA and drug tactic TP was 5132±665 ms<sup>2</sup>, and in the third (persons with PLVA and CABG) – 4919±125 ms<sup>2</sup>, which was significantly greater compared with patients without PLVA (control group), 3592±274 ms<sup>2</sup>,  $p < 0.05$ .

Assessing the contribution of each part of ANS in the regulation of heart rate, we stated that patients with postinfarction cardiosclerosis are characterized by increased activity of sympathetic system that further increases in patients with PLVA. For example, in the control group «LF» value was 1753±38.9 ms<sup>2</sup>, in the second one – 2.215±59.8 ms<sup>2</sup>,  $p < 0.05$ . In the first and the third subgroups sympathetic activity was even higher, and the value of «LF» waves was 3103±93.6 ms<sup>2</sup> and 3295±45.4 ms<sup>2</sup>, respectively,  $p < 0.05$ .

Parasympathetic influences in patients with PLVA were weaker. For example, the lowest value of «HF» waves among research subgroups were stated for the second subgroup (individuals with PLVA and PCI). In this subgroup it was 436±21.7 ms<sup>2</sup>, which was significantly lower in comparison to the values of the control group, 1073±51.4 ms<sup>2</sup>, respectively  $p < 0.05$ . In the first and the third subgroups values of «HF» waves were slightly higher, but also significantly lower compared to patients without PLVA,  $p < 0.05$ .

Estimating the slow humoral-metabolic effects (waves in the range «VLF»), we found them to be more accented in patients with PLVA. Moreover, they prevailed in patients with PLVA

and drug treating tactic (the first research subgroup), in which the «VLF» value was  $1318 \pm 39.2 \text{ ms}^2$  and that was significantly greater, than in patients with PLVA and PCI (the second subgroup) and in patients with PLVA and CABG (the third subgroup),  $1056 \pm 23.3$  and  $1176 \pm 24.6 \text{ ms}^2$ , respectively,  $p < 0.05$ . According to the above mentioned results, percentages of specified parameters recorded changed as well.

Analysis of integral parameters revealed also some interesting patterns. Thus, in patients with PLVA prevailing of the autonomous centers over central mechanisms of regulation was stated. For example, the index of centralization («IC») was the lowest in patients of the first and the second research subgroups,  $2.9 \pm 0.3$  and  $2.3 \pm 0.1$ , respectively, which were significantly lower compared to the patients of control group,  $p < 0.05$ . It is interesting to mention and worse (lower) value of the so-called Baevsky index or stress index «SI» in patients with PLVA, characterizing the high activity of the sympathetic ANS. For example, it was  $92.1 \pm 3.27$  units in the control group, and only  $33.8 \pm 1.12$  units in the first research subgroup,  $p < 0.05$ . In other research subgroups «SI» was slightly higher, although significantly lower compared to data of the control group.

#### 4. Conclusions

One can conclude that patients with PLVA tend to tachycardia and have the higher occurrence of non-sinus complexes. They are characterized by increased sympathetic activity and higher humoral influences, prevalence of peripheral effects over the central ones, as well as significantly longer duration of QT interval. The use of CABG increases the rate of ectopic complexes of supraventricular and ventricular origin. The patients with PLVA and previous PCI have, in this context, the most favorable course of the disease. Prior revascularization of left ventricle by means of PCI may reduce aggressive sympathetic influences, as well as optimize the value of the stress index.

#### References

1. Antezano ES, Hong M. Sudden cardiac death. *J Intensive Care Med.* 2003; 18:313-329.
2. Bayes de Luna A, Coumel P, Leclercq JF. Ambulatory sudden cardiac death: mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *Am Heart J.* 1989; 117:151-159.
3. Miller JM. Diagnosis of cardiac arrhythmias / J. M. Miller, D. P. Zipes // *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* / J. M. Miller, D. P. Zipes. — Philadelphia, PA: Saunders Elsevier, 2011, 36.
4. Myerburg RJ, Castellanos A. Cardiac arrest and sudden cardiac death. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine.* Philadelphia: W. B. Saunders, 2001, 890-931.
5. Olgin JE. Approach to the patient with suspected arrhythmia / J. E. Olgin, L. Goldman, A. I. Schafer // *Goldman's Cecil Medicine* / In: L. Goldman, A. I. Schafer. — Philadelphia, PA: Saunders Elsevier, 2011, 62.
6. Pinto DS, Josephson ME. Sudden cardiac death. In: Fuster V, ed. *Hurst's The Heart.* New York: McGraw-Hill, 2001, 1015-1048.
7. Spirito P, Bellone P, Harris KM, Bernabo P, Bruzzi P, Maron BJ. Magnitude of left ventricular hypertrophy and risk of sudden death in hypertrophic cardiomyopathy. *N Engl J Med.* 2000; 342:1778-1785.

8. Zheng *et al.* State-Specific Mortality from Sudden Cardiac Death—United States 1999. *MMWR.* 2002; 51(6):123-126.