

# Heart rate variability under endogenous intoxication before and after percutaneous transluminal coronary angioplasty in patients with angina pectoris

Cite as: AIP Conference Proceedings 2053, 030043 (2018); <https://doi.org/10.1063/1.5084404>  
Published Online: 19 December 2018

T. F. Mironova and V. A. Mironov



View Online



Export Citation

## ARTICLES YOU MAY BE INTERESTED IN

[High resolution heart rate variability analysis in patients with angina pectoris during coronary artery bypass graft surgery](#)

AIP Conference Proceedings 1915, 030013 (2017); <https://doi.org/10.1063/1.5017333>

[Heart rate variability in young men at rest and in autonomic stress testing](#)

AIP Conference Proceedings 2053, 040003 (2018); <https://doi.org/10.1063/1.5084441>

## Lock-in Amplifiers up to 600 MHz



Zurich  
Instruments



# Heart Rate Variability under Endogenous Intoxication before and After Percutaneous Transluminal Coronary Angioplasty in Patients with Angina Pectoris

T. F. Mironova<sup>1</sup> and V. A. Mironov<sup>2, a)</sup>

<sup>1</sup>*Ekaterinburg Medical Scientific Center of Preventive Treatment and Health Care of Industrial Workers, 30 Popova St., 620014, Ekaterinburg, Russia*

<sup>2</sup>*Ural State Medical University, 3 Repina St., 620028, Ekaterinburg, Russia*

<sup>a)</sup>Corresponding author: [vamironov2013@yandex.ru](mailto:vamironov2013@yandex.ru)

**Abstract.** The paper studies heart deregulation by high-resolution rhythmocardiography (RCG) and analyzes heart rate variability (HRV) in patients with stable angina pectoris (SAP) before and after of percutaneous transluminal coronary angioplasty (PTCA). HRV deregulations were defined in 57 patients (pts) in special form of small lengthening of 3-5 RR intervals (Fig. 2). They had a period of  $2.07 \pm 0.41$  s and a spectral power of  $0.218 \pm 0.016$  Hz in the high-frequency range in the wave HRV structure. Such waves were recorded in pts with chronic intoxication in different somatic diseases during inflammation. The comparison of HRV in pts with SAP and healthy control pts (Fig. 1), and also before and after PTCA, showed that HRV indices were the worst under endogenous intoxication. The most expressive regulative HRV breaches were detected after PTCA. This intoxication may be a predictor of inflammation during and after PTCA. The RCG diagnosis of the HRV waves was not defined by standard methods. Such HRV wave structure shows supplementary intoxication complications after PTCA in the pts, and it can deteriorate the PTCA results.

## INTRODUCTION

The urgency of the research stems from the need to improve diagnostics when carrying out intervention of cardiac revascularization of a myocardium by the PTCA method in pts with SAP. As an innovative investigation, the method of high-resolution rhythmocardiography (RCG) is applied to the diagnostics of heart deregulation, advancing standard clinical and paraclinical symptoms of the ischemic process, this being an obligatory background at any changes in the conditions of pts.

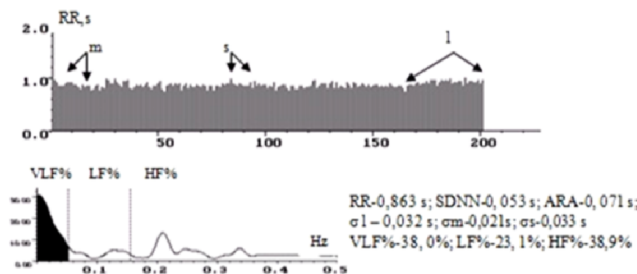
## MATERIALS AND METHODS

Patients with SAP were investigated by standard cardiological methods (ECG with loading, HM, EchoCG research, BP monitoring, CAG before PTCA), as well as by RCG before and after PTCA. 57 pts were selected according to primary RCG with the high-frequency waves of the HRV in the form of low-amplitude lengthening of 3-5 RR intervals, which was described in several studies [1, 2] as characteristic of endogenous intoxication in inflammatory pathology. RCG written before and after PTCA had a cardiosignal discretization of  $1000 \pm 3$  Hz. Computerized statistical and spectral analyses were performed in time and frequency domains with the application of a fast Fourier transformation and spectral Hamming and Parsen windows in order to determine the correlation of the shares of three factors regulating the heart rhythm – autonomic sympathetic (LF%), parasympathetic (HF%), and humoral-metabolic influence (VLF%). The following RCG indices were determined in the statistical analysis: average values of intervals (RR), their standard deviation (SDNN), the mean square dispersion of sympathetic, parasympathetic and humoral waves of HRV –  $\sigma_m$ ,  $\sigma_s$ , and  $\sigma_l$ , respectively. The RCG research was done at rest (Ph) and in the tests – Valsalva-Burker (Vm), Ashner (pA), active orthostatic (Aop), with a load corresponding to a

cardiac rate of 120 bpm ( $PWC_{120}$ ). The following indices were analyzed in the tests: maximal reaction to stimuli ( $\Delta RR$ ), time of its achievement ( $tAB$ ) and restoration ( $tr$ ). For control, 41 healthy men (Fig. 1) aged comparably to pts with SAP were examined by the same methods, including RCG. The research of HRV was performed by a CAP-RC-01-Micor computerized diagnostic complex (Reg. certificate No. 022b2005/2447-06). The nonparametric Spirmen method with a SPSS-12 package was used, as well as the Stat-6 program, Student's criteria, and the Z analog for nonparametric gross sampling.

## RESULTS AND DISCUSSION

The above-discussed HRV was recorded for all the 57 patients (Fig. 2); an evening increase in subfebrile temperature, an increase in leukocytes and monocytes. The same HRV was described in recent studies for cerebral tumors in the terminal stage [1] in combination with intoxication and anemia. Breaches in the immunological status were detected – a decrease in lymphocytes, CD4+ (%), CD16+ (%), and the CD4+/CD8+ ratio, an increase in leukocytes, etc. In pts with 1- and 2-type diabetes (SD) [2], the HRV waves in the high-frequency spectral range of  $0.23\pm 0.045$  Hz and  $0.24\pm 0.16$  Hz moderately and strongly correlated ( $r = 0.358-0.432$  and  $r = 0.711-0.731$ ) with symptoms of accumulated products of broken metabolism in humoral environments. The described HRV waves were termed nonparasympathetic (NPW). The direct correlation of the shares of humoral waves of the spectral power (VLF%) at rest and in all tests was found. A significant average negative correlation with all the HRV data and the level of blood urea ( $r = 0.570-0.620$ ) was revealed. NPW had a peak of spectral density in the spectral range of  $0.23\pm 0.045$  Hz and a period of 2.33 to 2.35 s. The high-frequency range of NPW was determined in 1500 RR interval sampling [2]. For 57 patients with SAP, NPW had a period of  $2.07\pm 0.41$  s and a spectral power peak of  $0.218\pm 0.016$  Hz in the high-frequency range. The results of comparison of the HRV indices before and after PTCA in the groups of pts without NPW and SAP and in the NPW+SAP group are presented in Tables 1 and 2. Statistic data for NPW+HRV – the RR interval, SDNN,  $\sigma_l$ ,  $\sigma_m$ , and  $\sigma_s$  – were the lowest. NPW in 57 pts with SAP had a period of  $2.07\pm 0.41$  s and a spectral power peak of  $0.218\pm 0.016$  Hz in the high-frequency range. Tables 1 and 2 show the results of comparison of all the data.



**FIGURE 1.** Rhythmicardiograms, spectrograms and HRV indices of a healthy man: m (LF) – sympathetic waves and their spectral share, s (HF) – parasympathetic waves, l (VLF) – humoral-metabolic HRV fluctuation.

The combination of SAP with intoxication is very bad for RTCA, and it assumes deterioration as a result of angioplasty. The dynamics of the HRV indices had a low level in the tests in ms units, which demonstrated a considerable oppression of the autonomic regulation of the heart rhythm or a pathological change in the effector pacemaker of SN at the synapses level (Table 1). The spectral HRV data (Table 2) show that the fast autonomic regulation of HRV (LF%, HF%) is oppressed, and the humoral influence (VLF%), slow, low and inadequate, has become the leading regulative factor. The humoral influence during SAP and intoxication is higher than without intoxication.

The reactions in the tests, the time of the achievement of maximal reactions and the time of restoration after stimuli were the most pathological; the greatest share of the humoral-metabolic influence (VLF%) on the background of the oppression of autonomic regulation was in pts with SAP in combination with endogenous intoxication after PTCA. The RCG symptom of an ischemic episode was revealed in the structure of HRV in pts with SAP (RCG fragments without waves on RCG, with stabilization of HRV). These fragments had a difference in the length of RR intervals, on the average,  $3.55\pm 1.02$  ms, these fluctuations being explainable by the hibernation of pacemaker cells of the sinus node (SN) at the time of an increase in occlusion of coronary arteries during the activation of endothelina-1; the circulation of SN deteriorates and pacemakers become unexcitable at this moment.

In all the 5 positions, NPW were recorded, as well as decreased reactions to stimuli in the tests, their slow achievement and restoration after the stimuli. On the spectrograms there is a peak of spectral density in the high-frequency range.

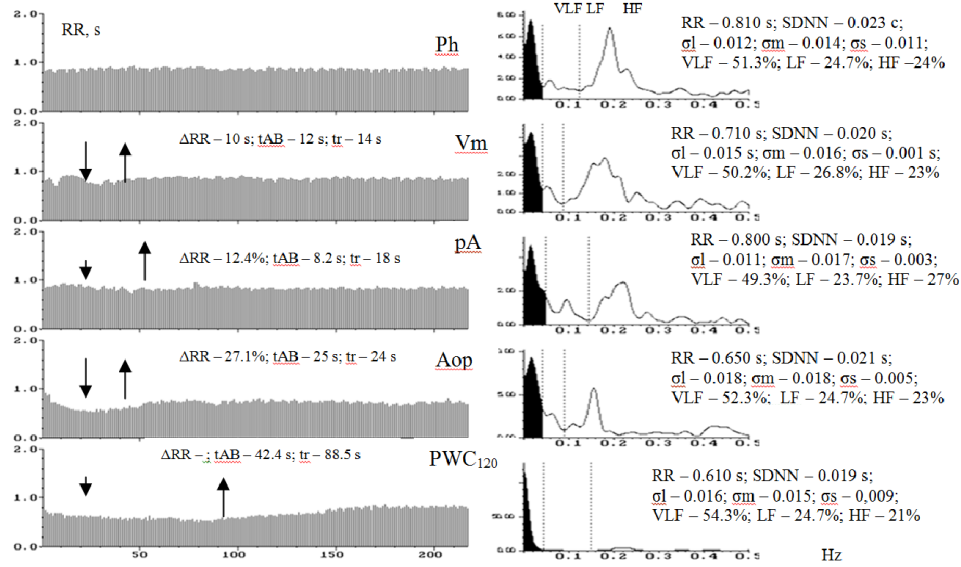


FIGURE 2. Rhythmocardiograms, spectrograms and HRV indices in a pt with SAP in the initial posture of rest (Ph) and in tests (Vm, pA, Aop, PWC<sub>120</sub>)

TABLE 1. Results of the statistical analysis of HRV in pts with SAP without NPV before PTCA (n=53, first line), in pts with SAD+NPV before PTCA (n=57, second line) and after PTCA (n=54, third line)

HRV indices, s and %, M $\pm\sigma$	Ph, initial posture at rest	Vm, Valsalva-Burker test	pA, Ashner test	Aop, active orthostatic	PWC <sub>120</sub> , load exercise
<b>RR, s</b> average interval	0.816 $\pm$ 0.088	0.811 $\pm$ 0.091	0.820 $\pm$ 0.111	0.803 $\pm$ 0.098	0.742 $\pm$ 0.071
	0.745 $\pm$ 0.073	0.769 $\pm$ 0.082	0.797 $\pm$ 0.101	0.751 $\pm$ 0.088	0.622 $\pm$ 0.100
	0.660 $\pm$ 0.033	0.695 $\pm$ 0.043	0.761 $\pm$ 0.033	0.680 $\pm$ 0.023	-
	Z1=6.75	Z1=2.62	Z1=0.8	Z1=3.0 Z2=19.6	Z1=7.5
	Z2=11.4	Z2=7.2	Z2=3.0		
<b>SDNN, s</b> Standard deviation of all intervals	0.025 $\pm$ 0.010	0.023 $\pm$ 0.003	0.025 $\pm$ 0.011	0.019 $\pm$ 0.007	0.028 $\pm$ 0.010
	0.020 $\pm$ 0.011	0.018 $\pm$ 0.001	0.021 $\pm$ 0.010	0.012 $\pm$ 0.005	0.016 $\pm$ 0.011
	0.018 $\pm$ 0.003	0.024 $\pm$ 0.005	0.017 $\pm$ 0.002	0.009 $\pm$ 0.004	
	Z1=2.6; Z2=1.4	Z1=11.6 Z2=9.5	Z1=2.1 Z2=3.6	Z1=6.36 Z2=3.65	Z1=4.44
<b><math>\sigma_l</math>, s</b> rms deviation of humoral-metabolic HRV waves	0.018 $\pm$ 0.005	0.017 $\pm$ 0.001	0.018 $\pm$ 0.003	0.012 $\pm$ 0.003	0.014 $\pm$ 0.008
	0.014 $\pm$ 0.004	0.021 $\pm$ 0.002	0.014 $\pm$ 0.005	0.008 $\pm$ 0.005	0.013 $\pm$ 0.007
	0.008 $\pm$ 0.003	0.020 $\pm$ 0.003	0.016 $\pm$ 0.002	0.007 $\pm$ 0.002	
	Z1=5.19 Z2=9.2	Z1=13.4 Z2=2.08	Z1=5.13 Z2=2.85	Z1=5.19 Z2=1.42	Z1=1.0
<b><math>\sigma_m</math>, s</b> rms deviation of sympathetic HRV waves	0.012 $\pm$ 0.004	0.011 $\pm$ 0.001	0.012 $\pm$ 0.001	0.011 $\pm$ 0.003	0.013 $\pm$ 0.003
	0.009 $\pm$ 0.003	0.014 $\pm$ 0.002	0.010 $\pm$ 0.002	0.009 $\pm$ 0.002	0.011 $\pm$ 0.004
	0.008 $\pm$ 0.001	0.015 $\pm$ 0.002	0.014 $\pm$ 0.002	0.012 $\pm$ 0.002	
	Z1=4.45 Z2=2.3	Z1=4.43 Z2=2.7	Z1=6.7 Z2=10.8	Z=4.5 Z2=8.1	Z1=2.98
<b><math>\sigma_s</math>, s</b> rms deviation of parasympathetic HRV waves	0.014 $\pm$ 0.004	0.012 $\pm$ 0.003	0.013 $\pm$ 0.004	0.007 $\pm$ 0.002	0.017 $\pm$ 0.004
	0.011 $\pm$ 0.003	0.016 $\pm$ 0.001	0.012 $\pm$ 0.001	0.004 $\pm$ 0.001	0.011 $\pm$ 0.001
	0.013 $\pm$ 0.002	0.015 $\pm$ 0.002	0.014 $\pm$ 0.001	0.006 $\pm$ 0.002	-
	Z1=4.43 Z2=4.2	Z1=9.28 Z2=3.3	Z1=1.66 Z2=6.1	Z1=9.86 Z2=0.06	Z1=10.6

In Tables 1 and 2 the results for SAP pts without NPW are compared with those for pts with SAP+NPW before PTCA (Z1) and the results for pts with SAP+NPW before and after PTCA (Z2), the Z criterion being used for gross sampling.

**TABLE 2.** The results of comparison of spectral HRV indices and indices of the stimulant periods in tests for SAP pts without NPW before PTCA (n=57, 1st line), with SAP+NPW before PTCA (n=57, 2nd line) and after PTCA (n=54, 3rd line)

<b>HRV indices, s and %, M±σ</b>	<b>Ph, initial posture at rest</b>	<b>Vm, Valsalva- Burker</b>	<b>pA, Ashner test</b>	<b>Aop, active orthostatic</b>	<b>PWC<sub>120</sub>, load exercise</b>
<b>VLF%</b>	46.6±10.1	46.8±10.08	47.08±10.9	50.8±11.72	36.81±10.91
spectrum share	58.3±9.03	59.5±5.81	64.2±4.05	63.7±4.05	77.0±7.8
of humoral-metabolic HRV waves	63.9±7.002 Z1=6.3 Z2=3.68	68.4±5.008 Z1=5.41 Z2=8.7	62.11±7.02 Z1=10.8 Z2=1.98	57.8±4.02 Z1=7.67 Z2=7.8	- Z1=16.4
<b>LF%</b>	17.7±3.8	17.6±3.6	18.4±4.4	23.3±10.6	23.7±5.4
spectrum share	20.3±2.5	24.1±2.5	24.8±13.4	37.3±5.2	22.0±3.1
of sympathetic HRV waves	16.01±4.02 Z1=25.2 Z2=6.8	26.5±3.01 Z1=11.0 Z2=4.61	19.03±4.2 Z1=3.02 Z2=8.01	28.7±3.02 Z1=8.75 Z2=10.8	- Z1=2.07
<b>HF%</b>	31.51±10.60	29.70±9.7	29.50±9.5	15.70±1.15	57.6±11.40
spectrum share	33.09±11.02	39.9±5.3	35.8±4.02	27.1±6.04	30.8±4.9
of parasympathetic HRV waves	24.01±5.02 Z1=2.05 Z2=5.6	27.05±3.02 Z1=8.16 Z2=15.8	28.5±3.03 Z1=5.06 Z2=10.8	21.02±3.02 Z1=14.0 Z2=6.8	- Z1=17.4
<b>ΔRR%</b>	-	8.71±3.5	7.21±2.52	-15.31±4.51	-20.81±8.21
maximal reaction to stimuli in tests		10.8±2.8 4.2±1.2	4.04±0.5 2.11±1.02	-4.5±0.51 -5.3±1.2	-8.9±1.8 -
		Z1=3.48 Z2=16.5	Z1=9.6 Z2=8.04	Z1=17.7 Z2=4.7	Z1=10.4
<b>tAB, s</b>	-	7.41±2.01	14.38±2.93	17.11±5.1	30.8±4.8
the time of achieving the maximal reaction		12.6±1.0 17.11±2.2	12.0±1.4 18.5±2.002	7.0±2.1 10.3±2.002	15.3±2.1 -
		Z1=17.8 Z2=14.0	Z1=5.53 Z2=20.3	Z1=13.48 Z2=8.04	Z1=22.1
<b>tr, s</b>		10.2±3.5	11.37±5.6	18.61±5.21	87.1±10.01
the time of recovery after stimuli		28.5±4.1 33.4±2.1	25.3±2.4 31.3±3.1	22.5±3.1 35.2±4.2	85.9±12.4 -
		Z=25.4 Z2=8.1	Z=16.9 Z2=1.7	Z=4.8 Z2=18.4	Z1=0.57

Thus, the most significant breach of heart regulation and a danger of complication during and after PTCA was found in pts with SAP and intoxication.

## CONCLUSION

1. RCG is noninvasive, fairly informative and adequate method for the examination of patients with stable angina pectoris before and after PTCA.
2. The worst HRV indices were recorded for patients with a combination of stable angina pectoris with HRV symptoms of endogenous intoxication.
3. A special HRV wave structure characterizing intoxication by high resolution RCG with electrocardiosignal discretization of 1000±3 Hz was detected, with a period of 2.07±0.41 s and a spectral frequency of 0.218±0.016 Hz, which essentially correlates with symptoms of intoxication.

## REFERENCES

1. V. Sadirin, *Immunological and Autonomous Breaches in Patients with Cerebral Tumors under Radiation Therapy* (Chelyabinsk, 2008).
2. T. F. Mironova, E. V. Nuzhdina, and V. A. Mironov, “Neurocardiological symptoms of heart rate variability at diabetic endotoxycosis,” in *Sixth International Symposium on Neurocardiology, NEUROCARD 2014* (Belgrad, Serbia, 2014), pp. 112–115.