

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



---

# Differential Diagnosis of Monotonous Fetal Heart Rate

---

Alexander Karpov, Anna Simakova, Oksana Frolova,  
Gregory Shiferson and Igor Yemelianov

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/intechopen.69988>

---

## Abstract

The aim was to explore the possibility to forecast a risk of hypoxic lesions in a monotonous fetal heart rate via ECG measurements by the methods of time and frequency analysis. The study involved 50 healthy pregnant women with singleton pregnancy at 37-41 weeks of gestation along with 17 pregnant women in the same period of gestation who had a monotonous fetal heart rate registered of various origin. The registration of fetal heart rates was performed using fetal monitor "Monica AN24" ("Monica Healthcare Ltd", United Kingdom), transabdominal, using ECG electrodes. The software package "Monica DK" has been used to retrieve the "beat-to-beat" data. Analysis of experimental data was carried out on the basis of LABVIEW® software (National Instruments®, USA). The analysis of time parameters for fetal hypoxia showed a sharp decline in the spread function and a sharp increase in the concentration function. Spectral analysis showed a significant decrease in the ratio of high- to low-frequency components of the spectrum. In the analysis of fetal ECG, the ST segment depression was noted, which is also indicative of fetal hypoxia.

**Keywords:** cardiotocography, ECG measurements, time HRV analysis, frequency HRV analysis, fetal hypoxia

---

## 1. Introduction

Cardiotocography (CTG) belongs to routine methods of fetal monitoring in modern obstetrics. Doppler heart monitors that use ultrasound to register fetal heart rate allow the assessment of heart rate variability (HRV) and designation of monotonous heart rate as an adverse prognostic parameter. Beyond that account must be taken of the fact that over 25% of cardiotocography (CTG) records in the antenatal period are defined as vague, i.e., deemed alarming. It basically refers to the monotonicity of fetal heart rate.

A monotonous heart rate is a rate with the amplitude not exceeding five heart beats per minute absent accelerations or decelerations. A series of successive heart rate values has a complex wave structure rather than presents a set of random numbers. Heart rate is largely affected by the autonomic nervous system. The impact thereof is manifested in altering various frequency components of heart rate oscillations [1]. The degree of the autonomic nervous system impact on the fetal cardiac function can be estimated by using standard time and frequency domain methods of HRV analysis. Recent years have seen the appearance of scientific papers on the interaction of the sympathetic and parasympathetic divisions of the autonomic nervous system [2–5]. However, ultrasonic CTG is limited in its further development due to the inability to provide its user with the beat-to-beat data required for HRV time and frequency analysis.

External noninvasive registration of fetal heart rate via ECG measurements is another method employed in fetal HRV study. Stable ECG signal generation alongside with fetal electrocardiogram morphology analysis has become the focus of numerous research investigations [6]. These are complemented with papers on the spectral analysis of transabdominal fetal electrocardiograms used for diagnosing fetal hypoxia during childbirth [7, 8]. Fetal electrocardiography is currently considered as an alternative to Doppler ultrasonic cardiotocography so far as the antenatal assessment of fetal condition is.

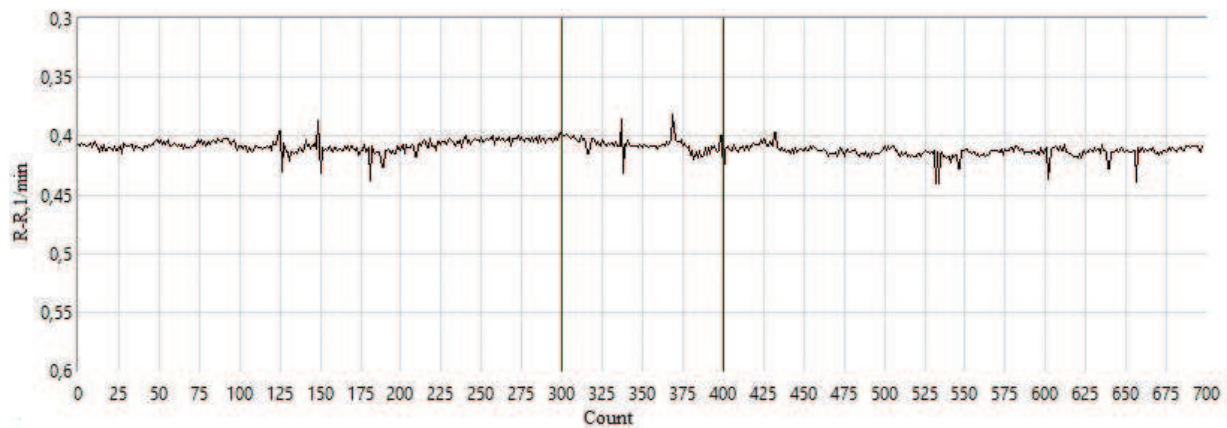
The present research is targeted toward the exploration of possibilities to predict the risks of hypoxic damage in the presence of monotonous fetal heart rate via ECG measurements with the application of time and frequency analysis methods.

## 2. Methods and materials

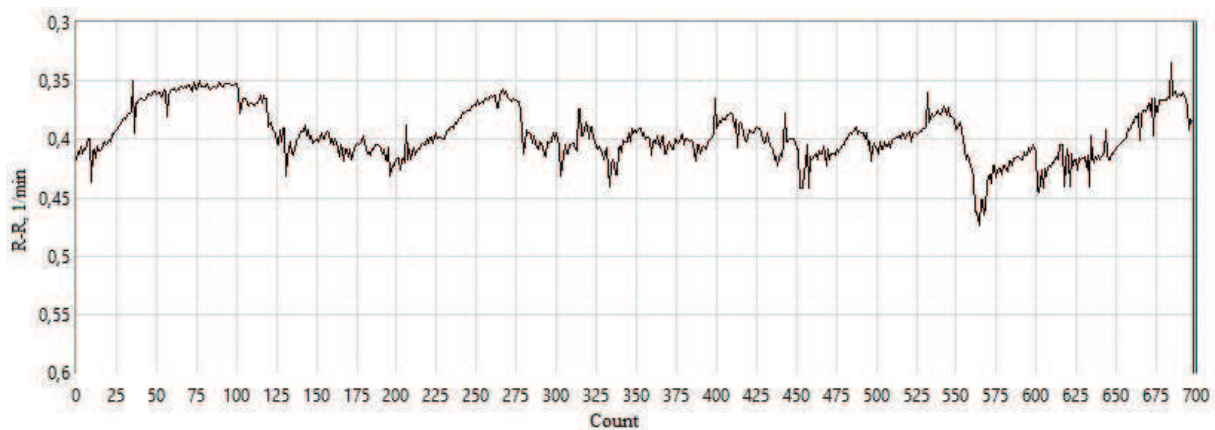
The sample of the research included 50 patients with normal singleton 37–41 week gestation admitted to the maternity department for delivery and 17 patients with the same gestation age diagnosed a monotonous fetal heart rate of varied etiology. Medical tests and measurements were conducted in the morning hours. Fetal heart rate was registered by using the “Monica AN24” (“Monica Healthcare Ltd” GBR)” fetal monitor, transabdominally, with the aid of ECG electrodes, the patient’s position being unrestricted. The “Monica DK” software package permitting to retrieve beat-to-beat data was used for analyzing the electrophysiological information.

Each fetal heart rate monitoring session lasted for 60 minutes, which corresponded to 7000 counts. To avoid mistakes in spectral analysis data interpretation, it is essential that the number of readings taken for comparison be equal. The following periods of the fetal cardiorythmogram were selected for analysis: a stationary period lasting for 5 minutes (700 counts, **Figure 1**), nonstationary period lasting for 5 minutes (700 counts, **Figure 2**), and integrated period lasting for 60 minutes (7000 counts, **Figure 3**).

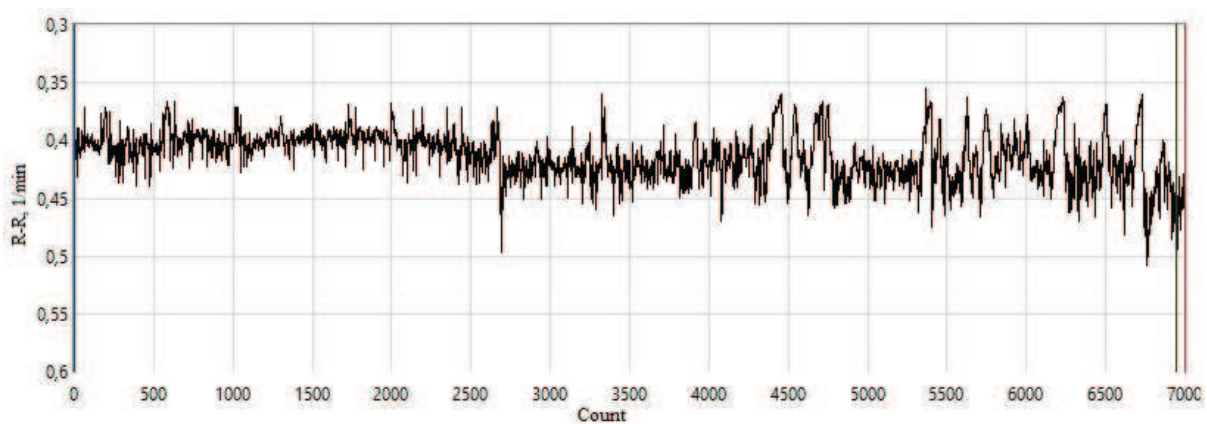
The background processing of data for temporal and spectral analysis consisted in the presentation of data in milliseconds (ms), elimination of artifacts (**Figures 4 and 5**), deletion of pop-up values using the “three sigma rule” (**Figures 6 and 7**), and estimation of stationarity using the Dx-statistics. Test data analysis was conducted on the basis of the LABVIEW® (National Instruments®, USA) software.



**Figure 1.** Stationary period of fetal cardiorythmogram. A duration of 700 counts (Y-axis—RR intervals, ms; X-axis—count).



**Figure 2.** Nonstationary period of fetal cardiorythmogram. A duration of 700 counts (Y-axis—RR intervals, ms; X-axis—count).



**Figure 3.** Integrated period of fetal cardiorythmogram. A duration of 7000 counts (Y-axis—RR intervals, ms; X-axis—count).

Normative time and frequency parameters for fetal HRV were determined in the group of patients with the normal gestation course. The parameters were defined as the upper (95%) and lower (5%) percentile limits of performance. All basic HRV functions were assessed:



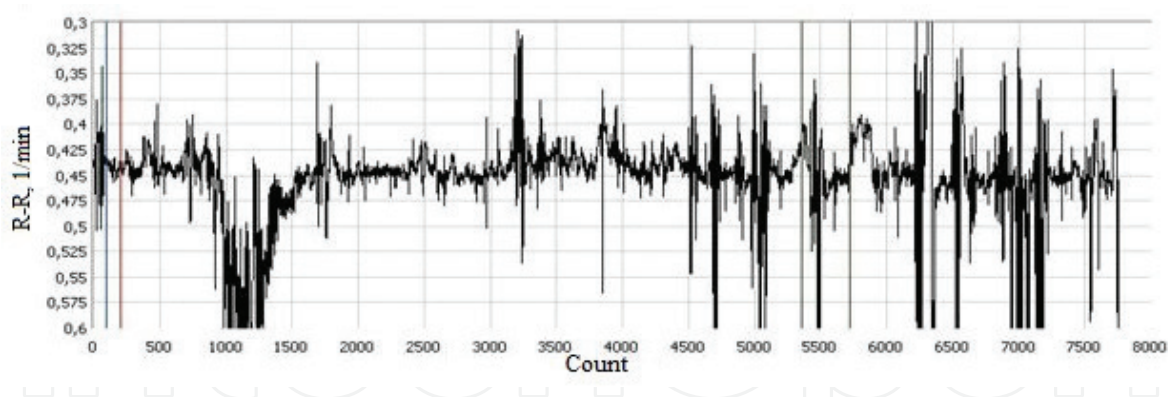


Figure 4. Fetal cardiorythmograms with artifacts (Y-axis—RR intervals, ms; X-axis—count).

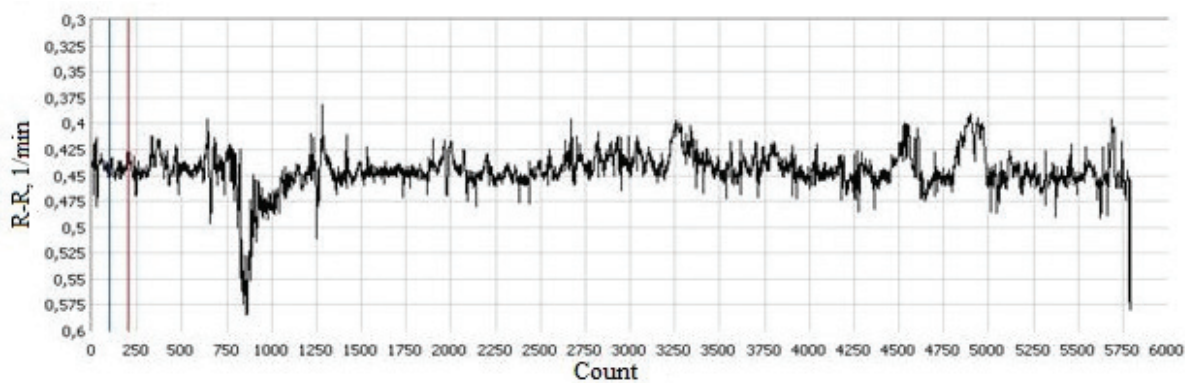


Figure 5. Fetal cardiorythmograms without artifacts (Y-axis—RR intervals, ms; X-axis—count).

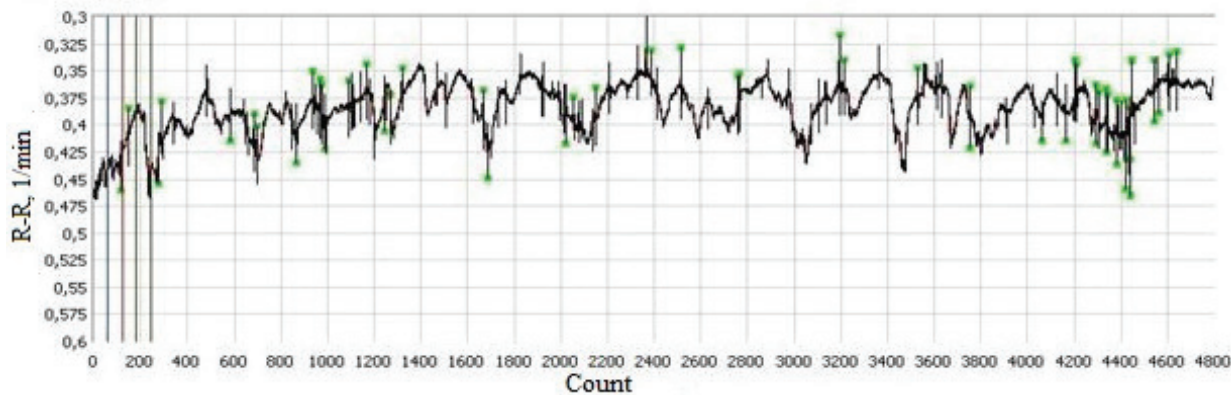
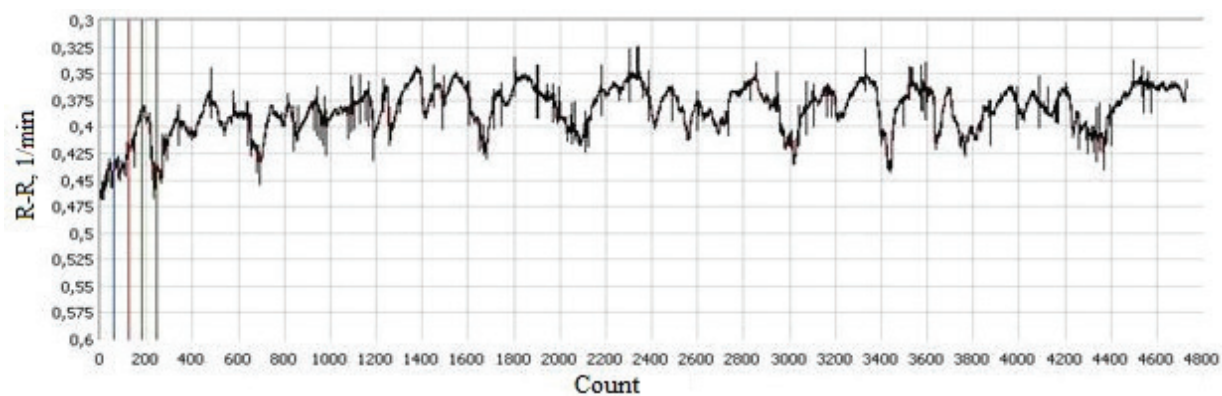


Figure 6. Fetal cardiorythmograms with pop-up values (green highlighting) (Y-axis—RR intervals, ms; X-axis—count).

scattering, concentration, and vegetal balance. The scattering function was tested by applying the indicators of standard RR interval distribution. The concentration function was assessed by mode parameters. Vegetal balance was tested utilizing the spectral analysis parameters as well as the fast-Fourier-transform algorithm. The ECG morphological analysis contained amplitude and time parameters. To overcome random interferences with the low ECG signal, we analyzed the average PQRST cycle (500–1000 cardiocycles) rather than every single cycle (Table 1).



**Figure 7.** Fetal cardiorythmograms without pop-up values (Y-axis—RR intervals, ms; X-axis—count).

The data correlation analysis permitted to identify five independent indicators to give the assessment of the fetal hypoxic damage risk by points (SDNN, RMSSD, AMo, Total Power, LF/HF). Assessment of fetal anoxic damage risk: 0–4 point—low; 5–6 point—medium; 7–8 point—high; 9–10 point—very high (**Table 2**).

The scattering function		
SDNN	ms	Standard deviation of all NN intervals
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals
SDANN	ms	Standard deviation of the averages of NN intervals in all 5-minute segments of the entire recording
NN15		Number of pairs of adjacent NN intervals differing by more than 15 ms in the entire recording; three variants are possible counting all such NN interval pairs or only pairs in which the first or the second interval is longer
PNN15%	%	NN15 count divided by the total number of all NN intervals
VR	ms	Difference between the maximum and the minimum RR intervals
The concentration function		
NModa		Number of cardio intervals corresponding to the mode value
AMo	%	Ratio of the amount of RR intervals with the values equaling the mode value to the total number of RR intervals, measured in percent
Moda <sub>range</sub>	%	Percent of cardio intervals in the mode range taken off the total number of RR intervals, measured in percent
AMo/PNN15	%	Ratio of AMo to the percentage of successive RR intervals with the difference exceeding 15 ms
St. George Index		Ratio of the histogram area to the amount of intervals with higher sampling rate duration
The spectral analysis		
VLF	ms <sup>2</sup>	Very low frequency range. Power in VLF range 0.02–0.08 Hz
LF	ms <sup>2</sup>	Low-frequency range. Power in LF range 0.08–0.2 Hz

IM*	ms <sup>2</sup>	Intermediate-frequency range. Power in IM range 0.2–0.4 Hz
HF	ms <sup>2</sup>	High-frequency range. Power in HF range 0.4–1 Hz
VLF% (AR)	%	Power in VLF range 0.02–0.08 Hz
LF% (AR)	%	Power in LF range 0.08–0.2 Hz
HF% (AR)	%	Power in HF range 0.4–1 Hz
Total power (AR)	ms <sup>2</sup>	Variance of all NN intervals
LF/HF (AR)		Ratio LF [ms <sup>2</sup> ]/HF[ms <sup>2</sup> ]
The ECG morphological analysis		
Wave P	mv	Wave amplitude P
Wave Q	mv	Wave amplitude Q
Wave R	mv	Wave amplitude R
Wave S	mv	Wave amplitude S
Wave T	mv	Wave amplitude T
Interval QRS	ms	Interval duration QRS
Interval ST	ms	Interval duration ST
Interval ST	mV	Segment ST Amplitude

Note: \*Parameters exceed the 5–95‰ limits.

**Table 1.** All basic parameters, HRV functions, and the ECG morphological analysis.

Variable//Point	0	1	2
SDNN	>95‰	5–95‰	<5‰
RMSSD	>95‰	5–95‰	<5‰
AMo	<5‰	5–95‰	>95‰
Total power	5–95‰	>95‰	<5‰
LF/HF	5–95‰	>95‰	<5‰

**Table 2.** Assessment of fetal anoxic damage risk.

### 3. Results

#### 3.1. Normative, time, and spectral HRV parameters

The gross findings for the patients with normal 37–41 week gestation are as such. Wide variability of the stationary/nonstationary period parameters taken into account, it is of utmost importance to determine the lower (5‰) and the upper (95‰) heart rate percentile limits.

The stationary period (5 minutes or 700 counts, **Table 3**) is characterized by the monotonicity of fetal heart rate continuing for 10–15 minutes in a 60-minute registration session (**Table 3**). It corresponds to the resting or dormant state of the fetus.

The nonstationary period (5 minutes or 700 counts, **Table 4**) is characterized by the fetal heart rate acceleration occurrences (**Table 4**). It corresponds to the motion activity of the fetus.

Integrated period (60 minutes or 7000 counts, **Table 5**) is characterized by the fetal heart rate acceleration and by the monotonicity of fetal heart rate.

It should be noted that time and frequency domain methods used to assess fetal HRV complement each other.

### 3.2. Time and spectral HRV parameters and ECG morphology in the presence of monotonous fetal heart rate

According to the etiological factor, three types of monotonous fetal heart rate can be identified:

1. Physiological—occurring because of a temporary decrease in the fetus regulatory centers activity and corresponding to the resting or dormant state of the fetus;
2. Pharmacological—caused by the suppression of activity of the central fetal heart rate regulation mechanisms (methyldopa and relanium) or by the block of impulse transmission to the sino-atrial node (atropine);
3. Hypoxic—conditioned by circulatory injuries in the presence of heart failure or intra-uterine infection.

Since heart rate monotonicity is a stationary cardiorythmogram, which means that the spectral properties of the signal do not change in time, the comparative study of the HRV time and frequency analysis data was conducted for the referential values of the stationary period only.

Scattering function								
	SDNN		rMSSD	NN15	pNN15%			VR
5‰	0.006		0.004	4	0.51			0.04
95‰	0.012		0.01	35	5.08			0.097
Concentration function								
	NModa		AMo	Moda <sub>range</sub>	AMo/PNN15			St. George Index
5‰	32		5.0	48	0.5			13
95‰	90		12.9	87	4.7			35
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	TP	LF/HF (AR)
5‰	0.3	1.9	1.3	5	49	12	4.3	1.15
95‰	2.9	18.8	5.8	11	78	40	18.5	3.96

**Table 3.** The stationary period. Lower (5‰) and upper (95‰) percentile limits of the fetal HRV time parameters.



Scattering function								
	SDNN	rMSSD	NN15	pNN15%	VR			
5‰	0.017	0.005	9	1.2	0.09			
95‰	0.033	0.01	71	10.3	0.16			
Concentration function								
	NModa	AMo	Moda <sub>range</sub>	AMo/PNN15	St. George Index			
5‰	19	2.7	29	0.2	29			
95‰	41	5.6	42	1.2	45			
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	TP	LF/HF (AR)
5‰	1.2	12	2.8	8	63	8	26	2.6
95‰	17.2	80	11	18	81	24	96	6.8

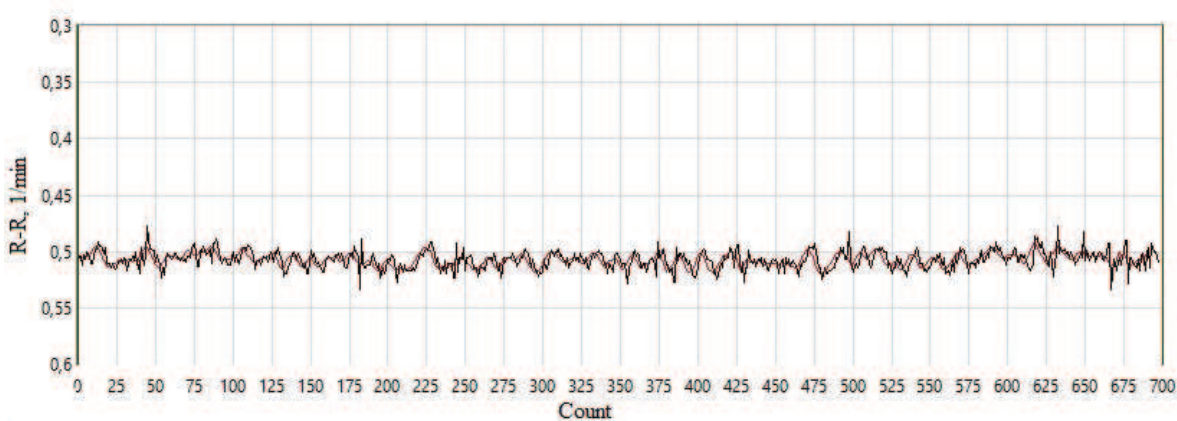
**Table 4.** The nonstationary period. Lower (5‰) and upper (95‰) percentile limits of the fetal HRV time parameters.

3.2.1. *Physiological monotonicity*

Physiological fetal heart rate monotonicity is registered during the dormant state of the fetus and continues for 10–15 minutes in a 60-minute cardiac cycle registration session. Here, we present data analysis of the patient with 40 week gestation: delivery of a baby boy, 3430 g, 54 cm, and 9/9 Apgar score and stationary period lasting for 5 minute(or 700 counts duration, **Figure 8** and **Table 6**).

Scattering function								
	SDNN	rMSSD	NN15	pNN15%	VR			
5‰	0.017	0.006	234	3.4	0.13			
95‰	0.03	0.011	621	10.8	0.19			
Concentration function								
	NModa	AMo	Moda <sub>range</sub> %	AMo/PNN15	St. George Index			
5‰	150	2.1	27	0.2	32			
95‰	318	4.4	49	1.6	54			
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	TP	LF/HF (AR)
5‰	20	122	38	9	65	15	178	2.7
95‰	99	478	138	15	73	23	697	4.6

**Table 5.** Integrated period. Lower (5‰) and upper (95‰) percentile limits of the fetal HRV time parameters.

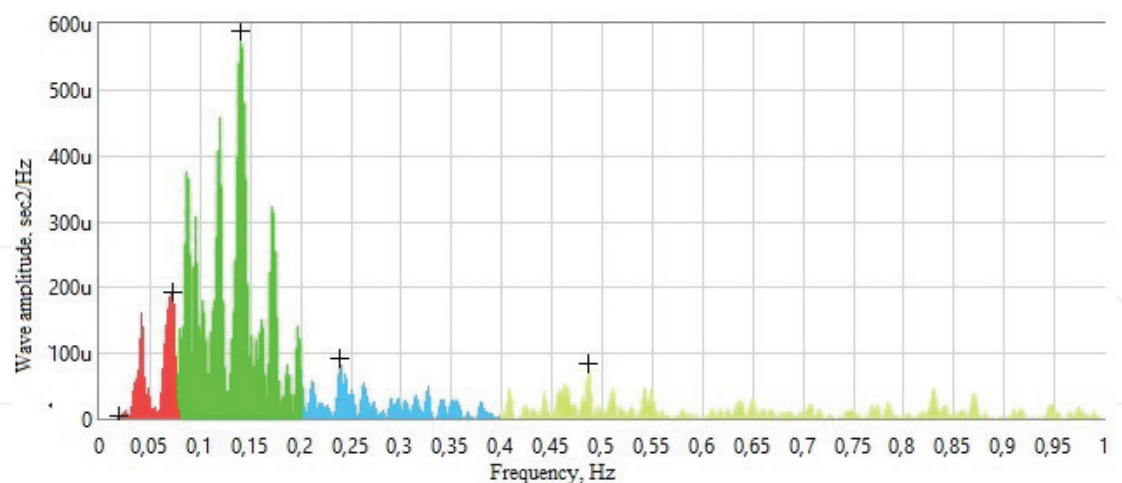


**Figure 8.** A gestation period of 40 weeks. Fetal cardiorythmograms, stationary period and physiological monotonicity (Y-axis—RR intervals, ms; X-axis—count).

The analysis of the time parameters revealed no impairment of the scattering or concentration functions (5–95%). The time analysis indices did not go beyond the percentile limits. The spectral analysis permitted to identify vegetal balance with the total power up (>95%) of the spectrum (**Figure 9**). However, it cannot be treated as a fetal hypoxia sign, which is

Scattering function								
	SDNN		rMSSD	NN15	pNN15%		VR	
5–95%	0.006–0.012		0.004–0.01	4–35	0.51–5.08		0.04–0.097	
sleep	0.008		0.007	23	3.3		0.06	
Concentration function								
	NModa		AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index	
5–95%	32–90		5.0–12.9	48–87	0.5–4.7		13–35	
sleep	51		7.3	52	2.2		27	
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	TP	LF/HF (AR)
5–95%	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
sleep	1.2	15	9.6*	5	58	37	26*	1.6
ECG morphology								
	P	Q	R	S	T	QRS	STint	ST
5–95%	0.39–2.14	–4.3 to –0.21	3.6–18.3	–7.96 to 0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
sleep	0.843	–2.41	11.8	–2.41	0	51	–	–0.12
Note: *Parameters exceed the 5–95% limits.								

**Table 6.** Time and frequency analysis parameters and ECG morphology in the presence of physiological monotonicity.

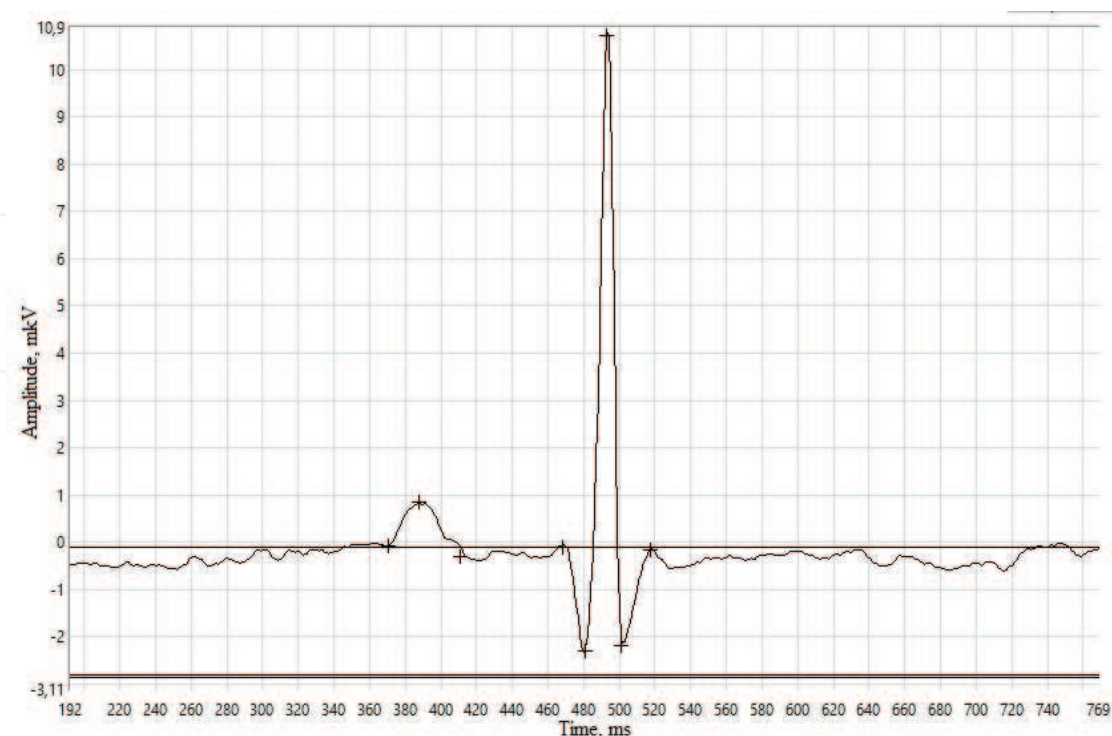


**Figure 9.** Spectral analysis of frequency (FFT method). A period of 40 week gestation, normal fetus. stationary period, physiological monotonicity.

confirmed by normal indicators of the fetus ECG (5–95%) (**Figures 8 and 10**). Facing this type of monotonicity, we can affirm that the risk of hypoxic damage assessed on the basis of time and frequency analysis parameters is low (4 points).

### 3.2.2. Pharmacological monotonicity: atropine

Atropine, a muscarinic receptor antagonist, reduces the vagal tone increasing the heart rate and raising conductivity along the His band. It is targeted toward the sino-atrial node causing



**Figure 10.** ECG. A period of 40 week gestation, normal fetus, stationary period, physiological monotonicity.

fetal heart rate monotonicity by increasing the activity of the sympathetic nervous system. The following is data analysis of the patient with 41 week gestation: planned labor induction; epidural anesthesia, cephalopelvic disproportion and caesarean section. Delivery of a baby boy, 4190 g, 56 cm, 8/9 Apgar score. Medicines used: atropine. Stationary period lasted for 5 minutes or 700 counts duration (**Figure 11** and **Table 7**).

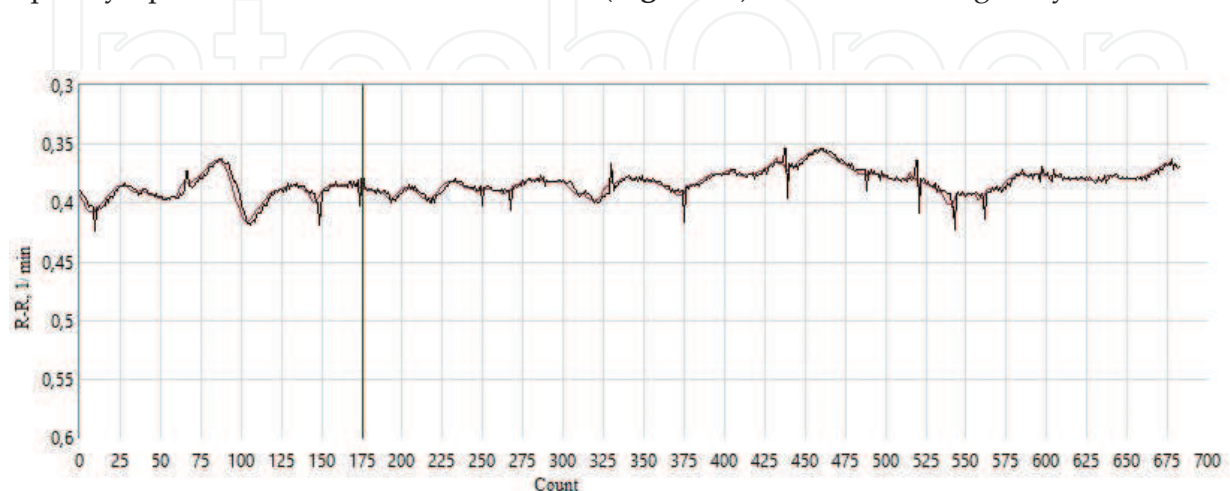
The analysis of the time domain parameters revealed no impairment of the scattering or concentration functions (5–95%). The time analysis indices did not go beyond the percentile limits. The spectral analysis permitted to identify the total power up (>95%) of the spectrum and the accentuated decrease of the high-frequency spectrum component (<5%) which reflects the weakening of parasympathetic influences over the heart (**Figure 12**). Accentuated sympathicotonia is confirmed by a significant increase in the high-to low-frequency spectrum components ratio (>95%). The fetal ECG parameters including the ST segment do not go beyond the percentile limits (5–95%), which speaks for the absence of fetal hypoxia (**Figure 13**). With this type of monotonicity, the risk of hypoxic damage assessed on the basis of time and frequency analysis parameters is moderate (5 points).

### 3.2.3. Pharmacological monotonicity: methyl dopa

Methyldopa, which is a stimulant of the alpha-adrenoceptors, causes fetal heart rate monotonicity by suppressing the activity of the central fetal heart rate regulation mechanisms.

The following is data analysis of the patient with 39 week gestation: arterial hypertension, labor induction, delivery of a baby girl, 3090 g, 52 cm, 9/9 Apgar score. Medicines used: methyldopa starting from week 32. Stationary period lasted for 5 minutes 700 counts duration, (**Figure 14** and **Table 8**).

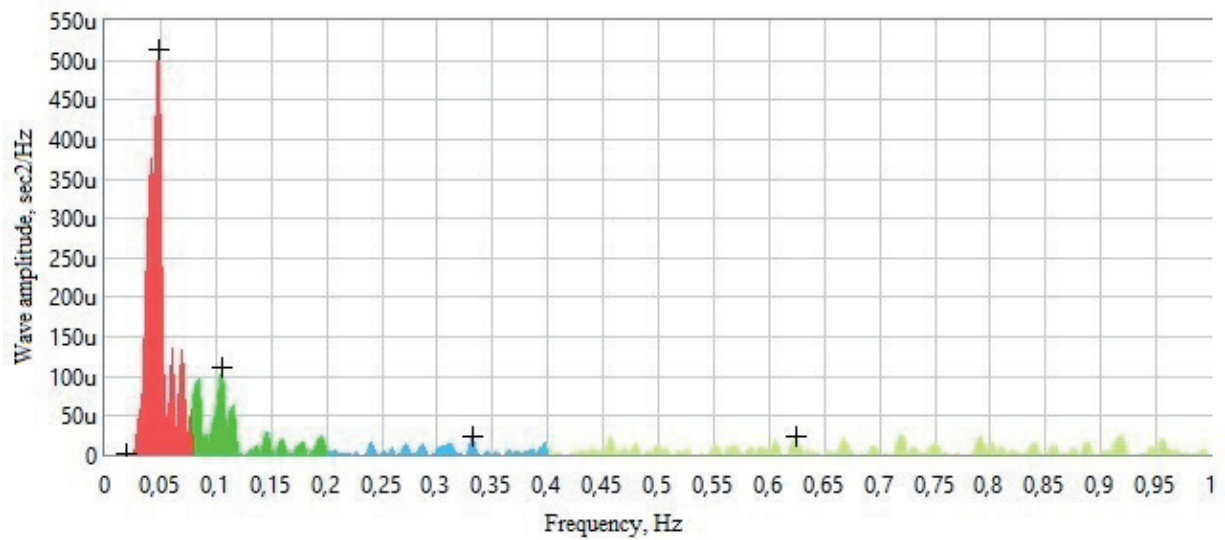
The analysis of the time parameters revealed the intensification of the sympathetic influences which resulted in the increase of the concentration function (>95%). The spectral analysis permitted to identify the increase of the high-frequency spectrum component (>95%) with a background of the total power down of the spectrum, which testifies for the predominance of parasympathetic influences over the heart (**Figure 15**). Accentuated vagotony is confirmed



**Figure 11.** A period of 41 week gestation, fetal cardiorythmograms, stationary period, pharmacological monotonicity, atropine effect (Y-axis—RR intervals, ms; X-axis—count).

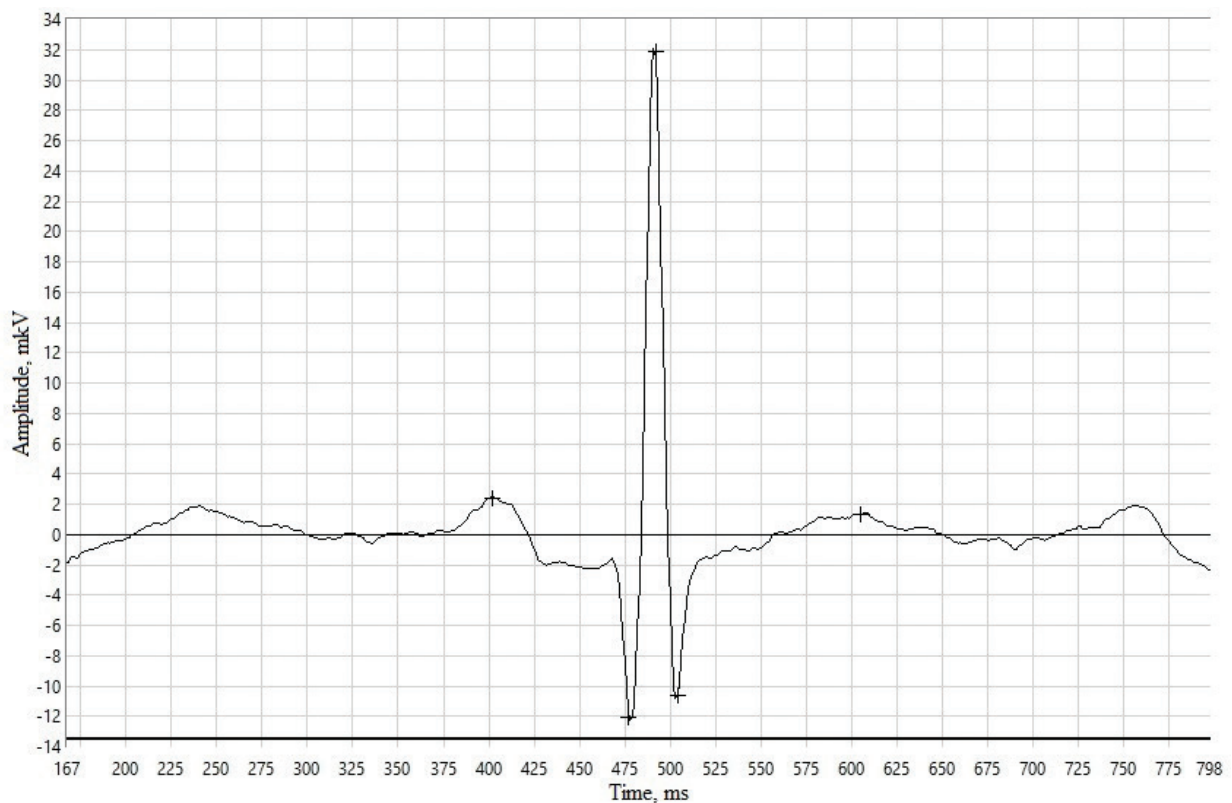
Scattering function								
	SDNN		rMSSD	NN15	pNN15%		VR	
5–95‰	0.006–0.012		0.004–0.01	4–35	0.51–5.08		0.04–0.097	
atropine	0.011		0.006	30	4.30		0.07	
Concentration function								
	NModa		AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index	
5–95‰	32–90		5.0–12.9	48–87	0.5–4.7		13–35	
atropine	57		8.2	58	1.9		22	
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	Power (AR)	LF/HF (AR)
5–95‰	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
atropine	1.6	14	1.9	13*	76	11*	19*	7.2*
ECG morphology								
	P mv	Q mv	R mv	S mv	T mv	QRS ms	Stint ms	ST mv
5–95‰	0.39–2.14	–4.3 to –0.21	3.6–18.3	–7.96 to –0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
atropine	3.08*	–11.8	32.4*	–10.1	1.64	51	33	–0.2
Note: *Parameters exceed the 5–95‰ limits.								

**Table 7.** Time and frequency analysis parameters and ECG morphology in the presence of pharmacological monotonicity— atropine.

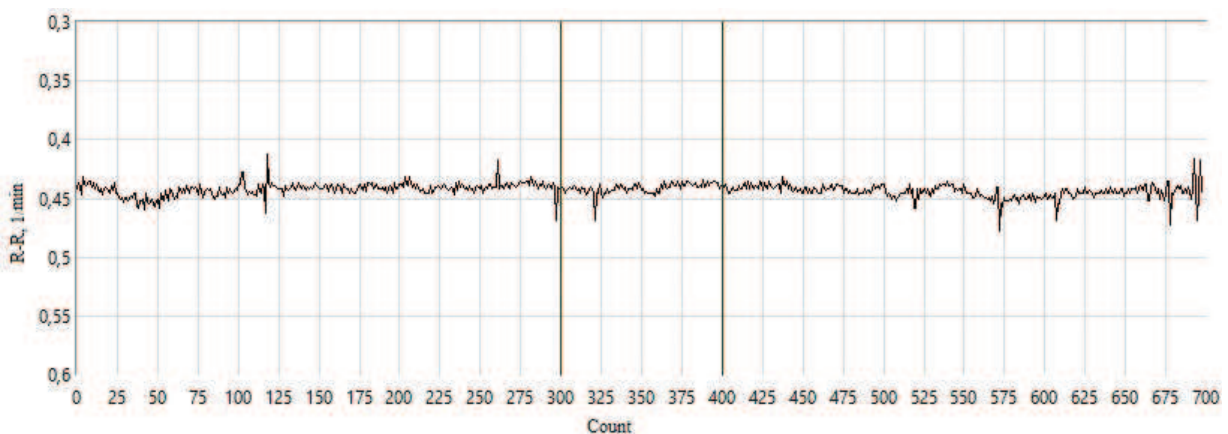


**Figure 12.** Spectral analysis of frequency (FFT method). A period of 41 week gestation, normal fetus, stationary period, pharmacological monotonicity, atropine.





**Figure 13.** ECG. A period of 41 week gestation, normal fetus, stationary period, pharmacological monotonicity — atropine.

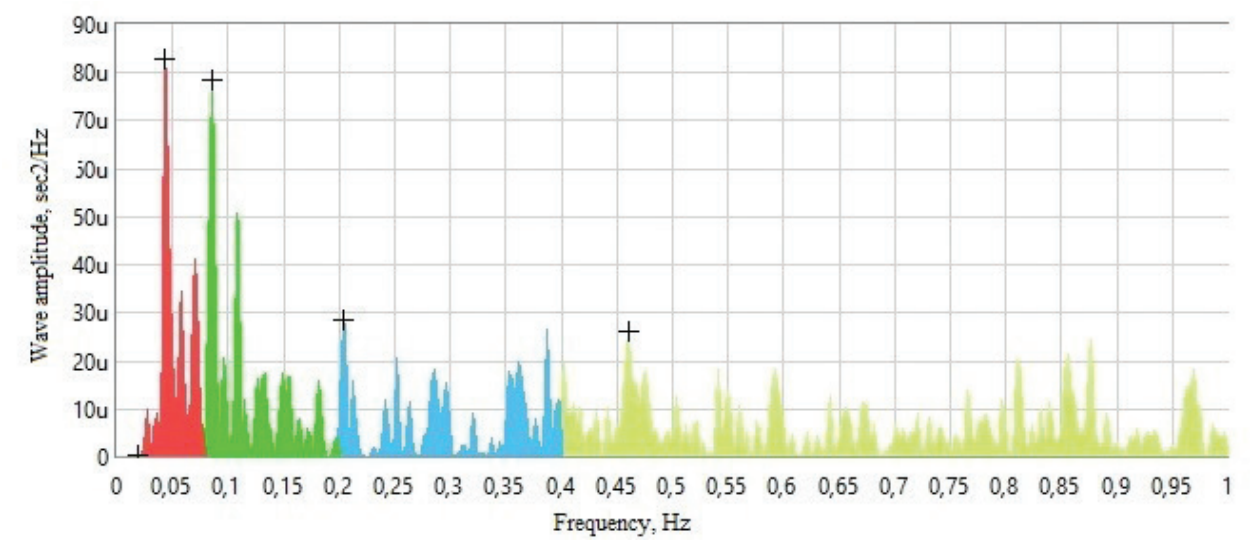


**Figure 14.** A period of 39 week gestation, fetal cardiogram, stationary period, pharmacological monotonicity, methyldopa effect (Y-axis—RR intervals, ms; X-axis—count).

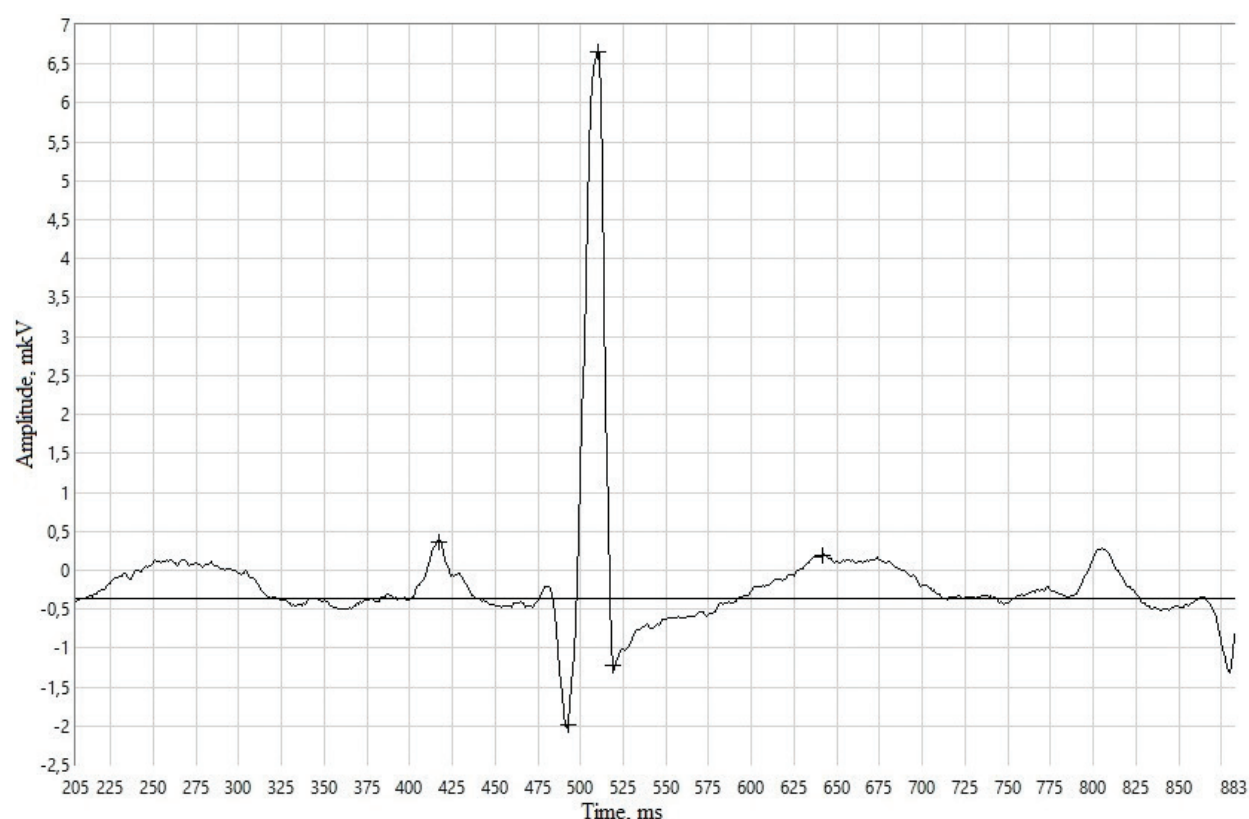
by a significant decrease in the high to low-frequency spectrum components ratio (<5%). However, it cannot be treated as a fetal hypoxia sign. The fetal ECG parameters do not go beyond the percentile limits (5–95%) (**Figure 16**). With this type of monotonicity, the risk of hypoxic damage assessed on the basis of time and frequency analysis parameters is moderate (6 points).

Scattering function								
	SDNN		rMSSD	NN15	pNN15%		VR	
5–95‰	0.006–0.012		0.004–0.01	4–35	0.51–5.08		0.04–0.097	
methyldopa	0.006		0.006	21	3.01		0.07	
Concentration function								
	NModa		AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index	
5–95‰	32–90		5.0–129	48–87	0.5–4.7		13–35	
methyldopa	137*		19.6*	61	6.5*		10*	
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	Power (AR)	LF/HF (AR)
5–95‰	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
methyldopa	0.134*	2.32	1.74	9.28	46*	45*	5.1	1.0*
ECG morphology								
	P	Q	R	S	T	QRS	STint	ST
5–95‰	0.39–2.14	–4.3 to 0.21	3.6–18.3	–7.96 to 0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
methyldopa	0.76	–1.62	7.09	–0.86	0.55	51	55	–0.2
Note: *Parameters exceed the 5–95‰ limits.								

**Table 8.** Time and frequency analysis parameters and ECG morphology in the presence of pharmacological monotonicity—methyldopa.



**Figure 15.** Spectral analysis of frequency (FFT method). A period of 39 week gestation, normal fetus, stationary period, pharmacological monotonicity, methyldopa.



**Figure 16.** ECG. A period of 39 week gestation, normal fetus, stationary period, pharmacological monotonicity, methylodopa.

### 3.2.4. Medication sleep (promedol and relanium)

Both promedol and relanium cause fetal heart rate monotonicity by depressing the central nervous system of the fetus. Here, you see data analysis of the patient with 40 week gestation, uterine scar, planned labor induction, medication sleep, delivery of a baby boy, 3600 g, 53 cm, 9/9 Apgar score. Medicines used: promedol and relanium. Stationary period lasted for 5 minutes or 700 counts duration (**Table 9** and **Figure 17**).

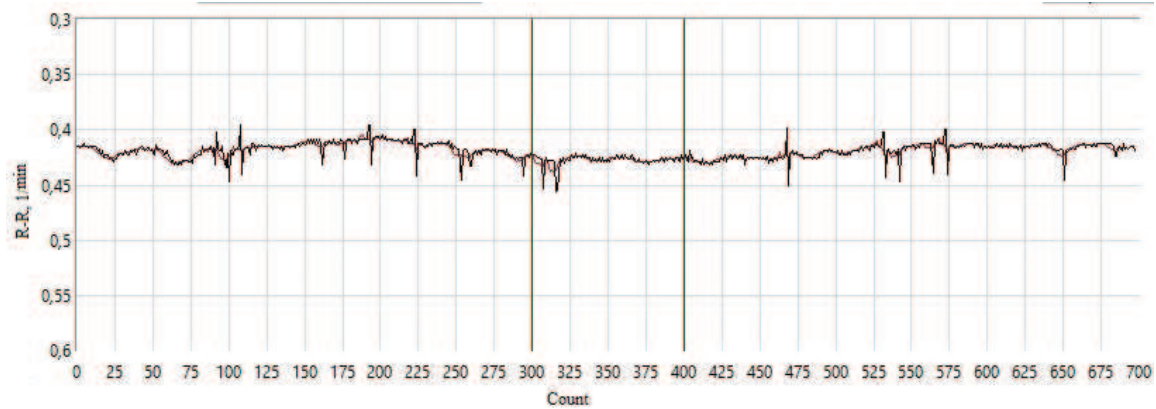
The analysis of the time parameters revealed no impairment of the scattering and concentration functions. The time analysis indices do not go beyond the percentile limits (5–95%). The spectral analysis permitted to identify vegetal balance with the normal spectrum power (5–95%) (**Figure 18**). The fetal ECG parameters do not go beyond the percentile limits (5–95%) (**Figure 19**). With this type of monotonicity, the risk of hypoxic damage assessed on the basis of time and frequency analysis parameters is low (3 points).

### 3.2.5. Hypoxic monotonicity

Lack of vegetal balance leads to an extreme increase of adrenergic influences over vascular walls, which ultimately results in the predominance of cholinergic influences, and this process causes circulatory hypoxia followed by a change in the arterial blood gas as well as pH.

Scattering function								
	SDNN			rMSSD	NN15	pNN15%		VR
5–95‰	0.006–0.012			0.004–0.01	4–35	0.51–5.08		0.04–0.097
Med. sleep	0.008			0.007	35	5.01		0.06
Concentration function								
	NModa			AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index
5–95‰	32–90			5.0–12.9	48–87	0.5–4.7		13–35
Med. sleep	84			12.0	67	2.4		17
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	Power (AR)	LF/HF (AR)
5–95‰	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
Med. sleep	0.398	3.47	3.05	5.69	55	39	6.5	1.37
ECG morphology								
	P	Q	R	S	T	QRS	ST <sub>int</sub>	ST
5–95‰	0.39–2.14	–4.3 to 0.21	3.6–18.3	–7.96 to 0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
Med. sleep	0.92	–1.54	13.51	–2.32	1.09	45	27	–0.07

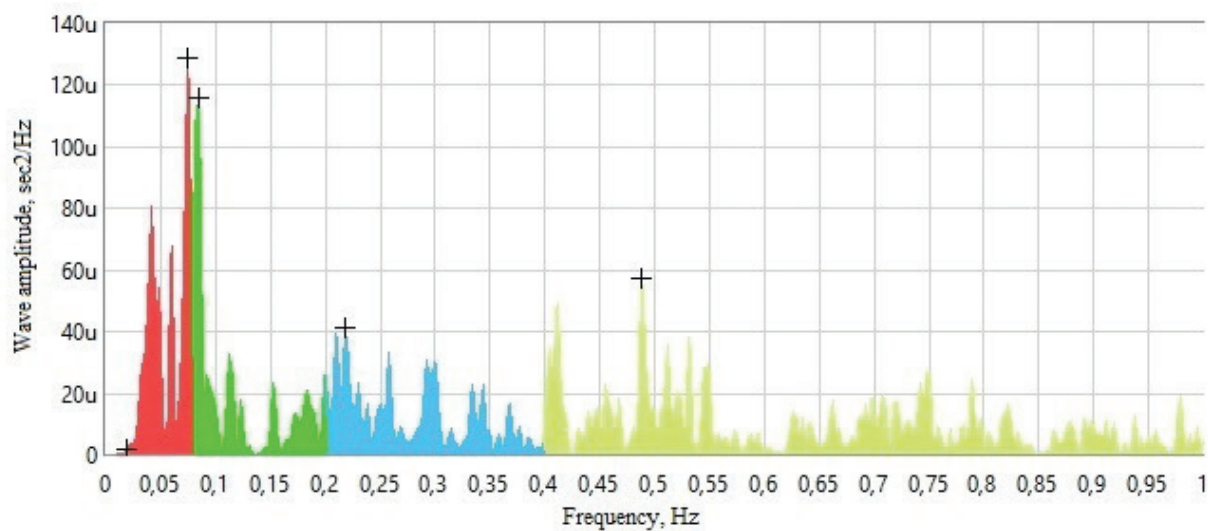
**Table 9.** Time and frequency analysis parameters and ECG morphology in the presence of pharmacological monotonicity—promedol and relanium.



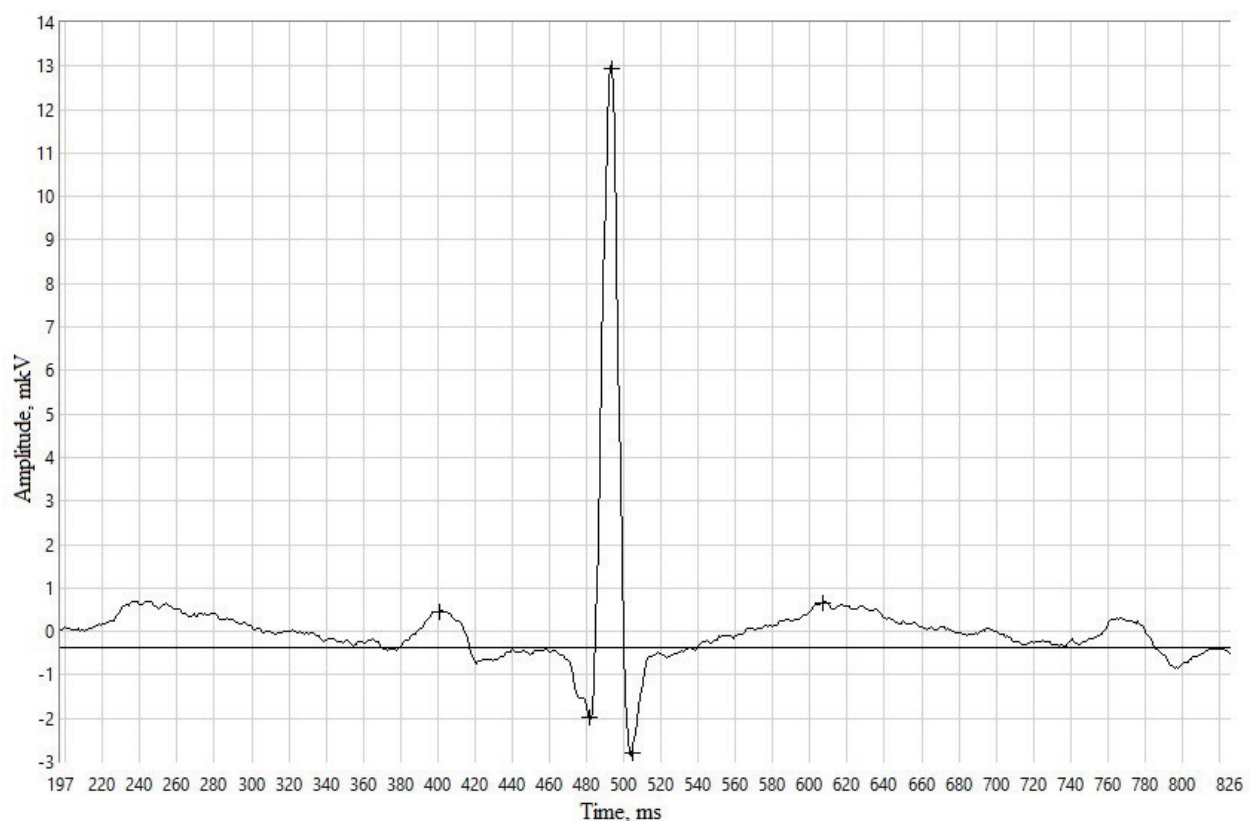
**Figure 17.** A period of 40 week gestation, fetal cardiorythmograms, stationary period, pharmacological monotonicity, promedol and relanium (Y-axis— RR intervals, ms; X-axis—count).

3.2.5.1. Heart failure

The following is data analysis of the patient with 40 week gestation: arterial hypertension, atrial septal defect (12 mm), inferior vena cava dilation (10 mm), ascite confirmed by ultra-sound fetometry. “Zero” blood flow in the umbilical artery, PI-1.77. Fetus weight: 3530 g. Caesarean section caused by fetal distress in labor. Stationary period lasted for 5 minutes or 700 counts (**Figure 20** and **Table 10**).



**Figure 18.** Spectral analysis of frequency (FFT method). A period of 40 week gestation, normal fetus, stationary period, pharmacological monotonicity, relanium and promedol.

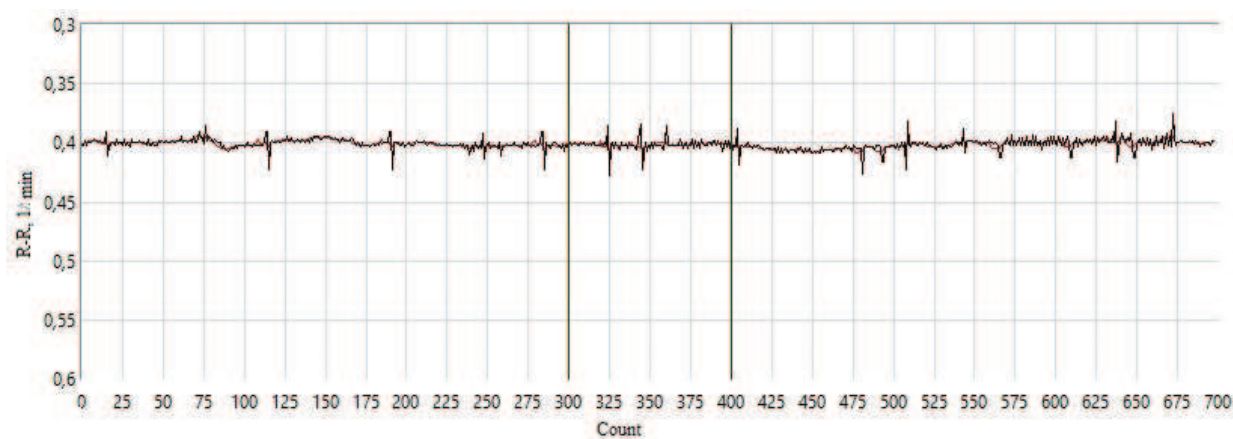


**Figure 19.** ECG. A period of 40 week gestation, normal fetus, stationary period, pharmacological monotonicity, relanium, promedol.

### 3.2.5.2. Fetal infection

The following is data analysis of the patient with 40 week gestation: monotonous fetal heart rate, labor induction, caesarean section caused by fetal head asynclitism; delivery of a baby boy, 3650 g, 51 cm, 6/7 Apgar score; venous blood pH: 7.054; neonatologist's diagnosis: congenital





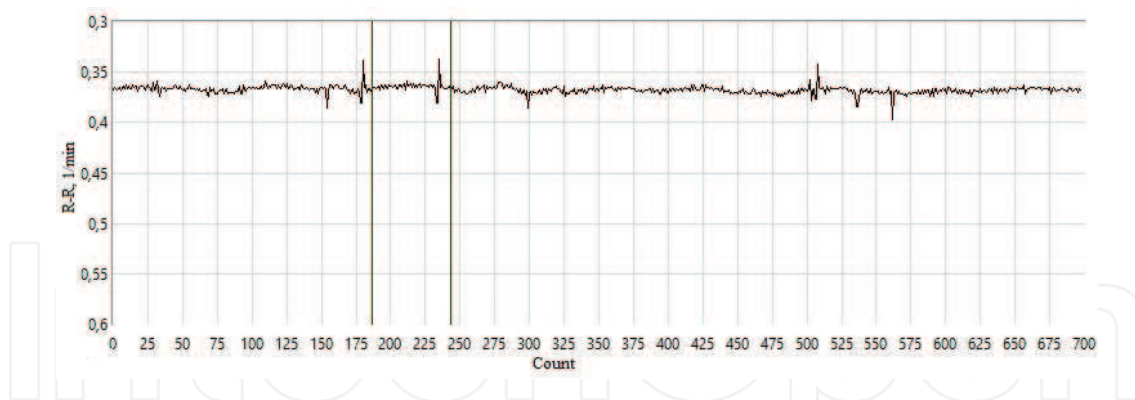
**Figure 20.** A period of 40 week gestation, fetal cardiorythmograms, stationary period, hypoxic monotonicity (Y-axis—RR intervals, ms; X-axis—count).

Scattering function								
	SDNN			rMSSD	NN15	pNN15%		VR
5–95‰	0.006–0.012			0.004–0.01	4–35	0.51–5.08		0.04–0.097
Hypoxia	0.005*			0.006	25	3.58		0.05
Concentration function								
	NModa			AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index
5–95‰	32–90			5.0–12.9	48–87	0.5–4.7		13–35
Hypoxia	173*			24.8*	50	6.92*		8*
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	Power (AR)	LF/HF (AR)
5–95‰	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
Hypoxia	0.13	0.487*	1.86	3.85	33*	63*	2.23*	0.53*
ECG morphology								
	P	Q	R	S	T	QRS	ST <sub>int</sub>	ST
5–95‰	0.39–2.14	–4.3 to –0.21	3.6–18.3	–7.96 to –0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
Hypoxia	1.14	–1.26	7.97	–1.71	0.654	52	42	–0.7*
Note: *Parameters exceed the 5–95‰ limits.								

**Table 10.** Time and frequency analysis parameters and ECG morphology in the presence of hypoxic monotonicity.

pneumonia; respiratory failure, stage III; mixed genesis shock; and stationary period lasted for 5 minutes or 700 counts (**Figure 21** and **Table 11**).

The analysis of the time parameters revealed a radical decrease of the scattering function (<5‰) alongside with a sharp increase of the concentration function (>95‰). This speaks for significant sympathetic influences over the cardiovascular system. The spectral analysis



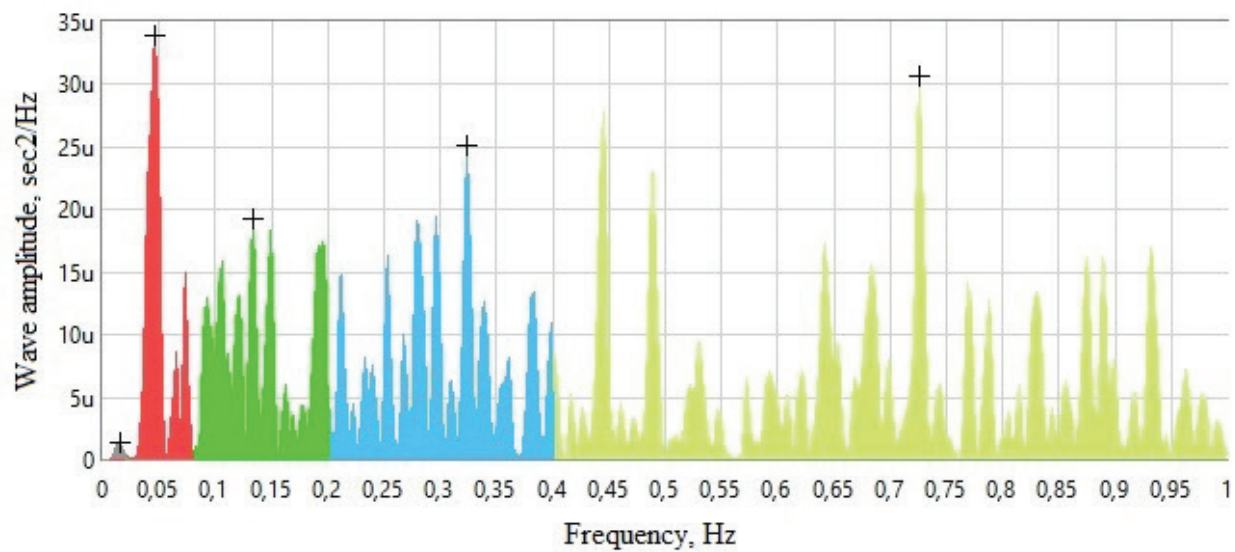
**Figure 21.** A period of 40 week gestation, fetal cardiorythmograms, stationary period, hypoxic monotonicity (Y-axis—RR intervals, ms; X-axis—count).

permitted to identify the increase of the high-frequency spectrum component (>95%) with a background of the total power down of the spectrum (<5%), which testifies for the predominance of parasympathetic influences over the heart (**Figure 22**). Accentuated vagotony is confirmed by a significant decrease in the ratio of high to low frequency components of the spectrum (<5%). Fetal ECG analysis shows depression of the ST segment, which is indicative of fetal hypoxia (<5%) (**Figure 23**). With this type of monotonicity, the risk of hypoxic damage assessed on the basis of time and frequency analysis parameters is very high (9 points).

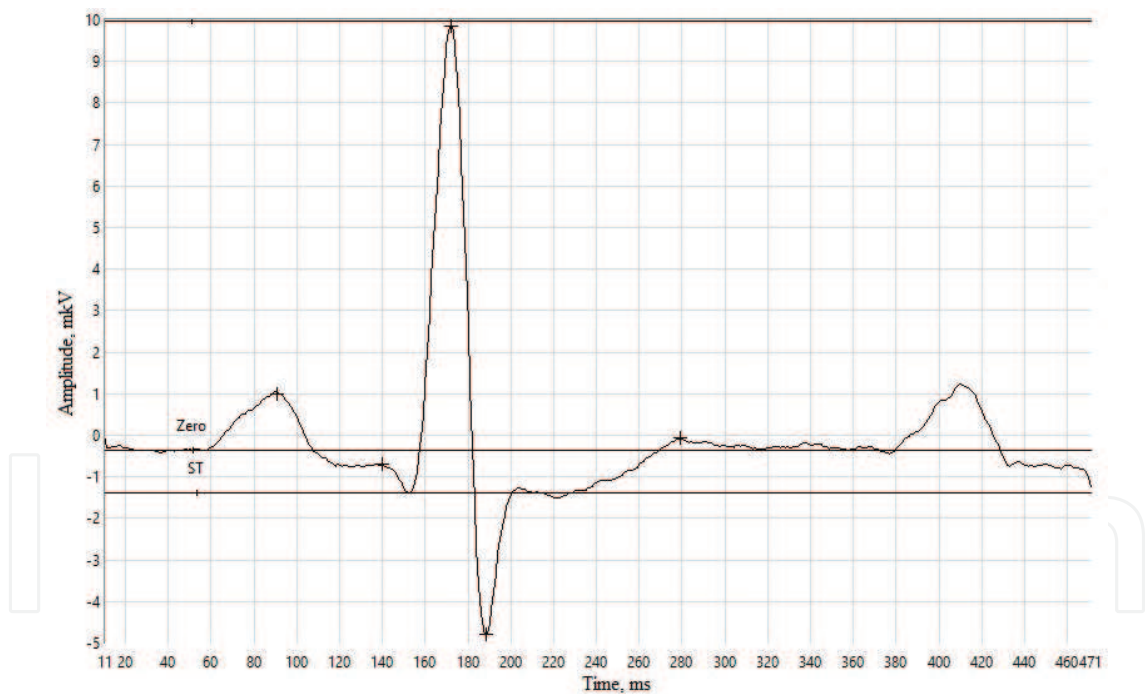
Scattering function								
	SDNN		rMSSD	NN15	pNN15%		VR	
5–95‰	0.006–0.012		0.004–0.01	4–35	0.51–5.08		0.04–0.097	
Hypoxia	0.005*		0.005	21	3.01		0.06	
Concentration function								
	NModa		AMo	Moda <sub>range</sub> %	AMo/PNN15		St. George Index	
5–95‰	32–90		5.0–12.9	48–87	0.5–4.7		13–35	
Hypoxia	125*		17.9*	51	5.95*		11*	
Spectral analysis								
	VLF	LF	HF	VLF% (AR)	LF% (AR)	HF% (AR)	Power (AR)	LF/HF (AR)
5–95‰	0.3–2.9	1.9–18.8	1.3–5.8	5–11	49–78	12–40	4.3–18.5	1.15–3.96
Hypoxia	0.18*	2.51	1.86	6.62	44*	50*	3.52*	0.88*
ECG morphology								
	P	Q	R	S	T	QRS	STint	ST
5–95‰	0.39–2.14	–4.3 to –0.21	3.6–18.3	–7.96 to –0.02	0.39–1.72	44–58	18–66	–0.2 to 0.6
Hypoxia	1.63	–3.01	20*	–7.71	–	52	54	–1.44*

Note: \*Parameters exceed the 5–95‰ limits.

**Table 11.** Time and frequency analysis parameters and ECG morphology in the presence of hypoxic monotonicity.



**Figure 22.** Spectral analysis of frequency (FFT method). A period of 40 week gestation. Fetometry: atrial septal defect (12 mm), inferior vena cava dilation (10 mm), ascite. “Zero” blood flow in the umbilical artery, PI-1.77. Stationary period and hypoxic monotonicity.



**Figure 23.** ECG. A period of 40 week gestation. Fetometry: atrial septal defect (12 mm), inferior vena cava dilation (10 mm), ascite. “Zero” blood flow in the umbilical artery, PI-1.77. Stationary period and hypoxic monotonicity.

## 4. Conclusion

Heart rate variability (HRV) is a CTG parameter considered important for fetal monitoring [9–12]. A monotonous heart rate is deemed an adverse prognostic parameter. A few scientific

papers devoted to fetal state distortion point at HRV decrease in the presence of fetal distress [6, 7]. However, other research investigations confirm that HRV decrease is possible during the dormant period of the fetus as well as when its nervous system is suppressed by pharmaceuticals. The understanding of the HRV physiological mechanism is not complete. It is widely accepted that the central nervous system dominates HRV regulation [13, 14]. The activity of the autonomic nervous system can be viewed as a brain function marker reflecting the regulatory capacity of the central nervous system [15]. Fetal autonomic nervous system activity is assessed on the basis of the time and frequency analysis of the RR intervals temporal series.

Beat-to-beat heart rate registration is a reliable source of data on the HRV spectrum [16, 17] and an indispensable prerequisite for correct interpretation of the fetal cardiogram. The complex wave structure of the fetal cardiorythmogram stipulates for the use of the time and frequency domain methods when analyzing vague CTGs. The majority of papers focused on the study of fetal state distortion by using the methods of time and frequency analysis have respect to the diagnostics of fetal distress during childbirth [7, 8, 12, 18–21]. We, however, believe that it is of utmost importance to monitor the fetus in the late weeks of gestation. We have defined percentile limits of the time and frequency analysis parameters required for fetal monitoring during the last weeks of pregnancy. The proposed diagnostic scale of fetal HRV time and frequency parameters permits to define the risk of anoxic damage when facing CTGs deemed alarming, including the cases of heart rate monotonous.

## Author details

Alexander Karpov<sup>1\*</sup>, Anna Simakova<sup>1</sup>, Oksana Frolova<sup>1</sup>, Gregory Shiferson<sup>2</sup> and Igor Yemelianov<sup>3</sup>

\*Address all correspondence to: [karpovay@medyar.ru](mailto:karpovay@medyar.ru)

1 Clinical Hospital, Yaroslavl, Russia

2 State Medical Academy, Yaroslavl, Russia

3 Yaroslavl State University PG Demidov, Russia

## References

- [1] Akselrod S, Gordon D et al. Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science*. 1981;**213**:220-222
- [2] Romano M, Luppieriello L, et al. Frequency and time domain analysis of foetal heart rate variability with traditional indexes: A critical survey. *Computational and Mathematical Methods in Medicine*. 2016. pp. 1-12
- [3] Van Laar JOE, Porath MM, Peters CHL. Spectral analysis of fetal heart variability for fetal surveillance: Review of the literature. *Acta Obstetrica et Gynecologica Scandinavica*. 2008;**87**:300-306

- [4] Van Leewen P, Geue D, et al. Changes in the frequency power spectrum of fetal heart rate in the course of pregnancy. *Prenatal. Diagnosis*. 2003;**23**:909-916
- [5] Manoj S, Kamalakar D, Desai and Mohan A. Gadani An estimate of fetal autonomic state by time spectral and nonlinear analysis of fetal heart rate variability. *International Journal of Computer Information Systems and Industrial Management Applications*. 2016;**8**:312-325
- [6] Graatsma EM, Jacod BC, van Egmont LAJ, et al. Fetal electrocardiography: Feasibility of long-term fetal heart recordings. *British Journal of Obstetrics and Gynaecology*. 2009;**116**:2: 334-338
- [7] Siira SM, Ojala TH, Vahlberg TJ, Jalonen JO, Valimäki IA, Rosen KG, et al. Marked fetal acidosis and specific changes in power spectrum analysis of fetal heart rate variability recorded during the last hour of labour. *British Journal of Obstetrics and Gynaecology*. 2005;**112**:418-423
- [8] Chung DY, Sim YB, Park KT, Yi SH, Shin JC, Kim SP. Spectral analysis of fetal heart rate variability as a predictor of intrapartum fetal distress. *International Journal of Gynaecology Obstetrics*. 2001;**73**:109-116
- [9] Pardey J, Moulden M, and Redman CWG. A computer system for the numerical analysis of nonstress tests. *American Journal of Obstetrics and Gynecology*. 2002;**186**(5):1095-1103
- [10] Sweha A, Hacker TW, and Nuovo J. Interpretation of the electronic fetal heart rate during labor. *American Family Physician*. 1999;**59**(9):2487-2500
- [11] Cabal LA, Siassi B, Zanini B, et al. Factors affecting heart rate variability in preterm infants. *Pediatrics*. 1980;**65**(1):50-56
- [12] Rantonen T, Ekholm E, Siira S, Metsälä T, Leino R, Ekblad U, et al. Periodic spectral components of fetal heart rate variability reflect the changes in cord arterial base deficit values: A preliminary report. *Early Human Development*. 2001;**60**(3):233-238
- [13] Sibony O, Fouillot JP, Bennaoudia M, Luton D, Blot P, and Sureau C. Spectral analysis of fetal heart rate in flat recordings. *Early Human Development*. 1995;**41**(3):215-220
- [14] Kero P, Anttila K, Ylitalo V, et al. Decreased heart rate variation in decerebration syndrome: Quantitative clinical criterion for brain death? *Pediatrics*. 1978;**62**:307
- [15] Hainsworth R. The control and physiological importance of heart rate. In: Malik M, Camm AJ, editors. *Heart Rate Variability*. Armonk, New York: Futura; 1995. pp. 3-19
- [16] Cerutti S, Civardi S, Bianchi A, Signorini MG, Ferrazzi E, Pardi G. Spectral analysis of antepartum heart rate variability. *Clinical Physics and Physiological Measurement*. 1989;**10**:27-31
- [17] van Laar JO, Warmerdam GJ, Verdurmen KM, Vullings R, Peters CH, Houterman S, Wijn PF, Andriessen P, van Pul C, Guid Oei S. Fetal heart rate variability during pregnancy, obtained from non-invasive electrocardiogram recordings. *Acta Obstetrica et Gynecologica Scandinavica*. 2014;**93**(1):93-101



- [18] Salamalekis E, Hintipas E, Salloum I, Vasios G, Loghis C, Vitoratos N, et al. Computerized analysis of fetal heart rate variability using the matching pursuit technique as an indicator of fetal hypoxia during labor. *Journal of Maternal-Fetal and Neonatal Medicine*. 2006;**19**:165-179
- [19] Ohta T, Okamura K, Kimura Y, Suzuki T, Watanabe T, Yasui T, et al. Alteration in the low-frequency domain in power spectral analysis of fetal heart beat fluctuations. *Fetal Diagnosis Therapy*. 1999;**14**:92-97
- [20] Cristian R, Alexandru P, Hariton C, Grigore TP, Dragos Nemescu. In: Spectral analysis of fetal heart rate variability associated with fetal acidosis and base deficit values. *Proceedings of 12th International Conference on Development and Application Systems*; 15-17 May 2014. pp. 210-213
- [21] Kwon JY, Park IY, Shin JC, Song J, Tafreshi R, Lim J. Specific change in spectral power of fetal heart rate variability related to fetal acidemia during labor: Comparison between preterm and term fetuses. *Early Human Development*. 2012;**88**(4):203-207

