

The impact of preeclampsia on fetal ECG morphology and heart rate variability

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Abstract

The aim of the investigation was a survey of the fetal HRV and ECG parameters in preeclampsia. It was performed fetal noninvasive ECG recordings in 94 pregnant women at 34-41 weeks of gestation and 66 of them were preeclamptic patients. The fetal deterioration in preeclampsia was characterized by lowered fetal heart rate variability and all its fractal components. The autonomic tone was diminished in direct proportion to the severity degree of preeclampsia. The mean value of short term vagal mediated parameters: RMSSD (root mean square of successive differences), pNN50 (the proportion of the number of pairs of successive NNs that differ by more than 50 ms divided by total number of NNs), HF (high frequency) and STV (short term variability) was also decreased. The relative predominance of the central sympathetic baroreflex mediated regulation of fetal hemodynamic was the main event in the preeclampsia induced scenario. The increased value of AMo (the amplitude of mode) and SI (stress index) was associated with abnormal myocardial adrenergic stimulation. It has induced pQ and QT shortening, increased T/QRS ratio and decelerations appearance. The augmented sympathetic tone played the significant role in fetal rigid rhythm and decelerations appearance and has formed the fetal myocardium hypoxic injury and suppressed sinus node response.

Key words: preeclampsia, fetal noninvasive ECG, heart rate variability, fetal distress

Fetal autonomic regulation spreads its influence on heart rate variability (HRV). This mechanism is associated with the sinus node response to continuous interaction of the sympathetic and parasympathetic tones [1, 4, 7]. The routine HRV assessment through antenatal cardiotocography (CTG) couldn't give a comprehensive ability to distinguish between fetal compromise and physiological autonomic balance changes according to several stationary conditions. It is known that fetus has periods with different activities as well as quiet and active sleep. During fetal trunk and extremities movements the increase of total power (TP) spectrum of HRV occurs. This process is associated with augmented low frequency (LF) and very low frequency (VLF) value. It indicates arising central sympathetic and humoral vasoactive response to fetal activity. The periods of fetal breathing are accompanied with high frequency (HF) predominance. This demonstrates the vagal nature of the cardiac and respiratory synchronization [7]. Fetal sleep has its suppressive influence on TP and all branches of HRV power spectrum.

So the HRV values have a very wide range that may complicate the interpretation [1, 7]. Additional investigation of the fetal ECG parameters could contribute to the advancement of the fetal functional condition diagnostics. It is proved that increased T/QRS ratio is a marker

of the fetal distress [9]. Several investigations have provided the information that fetal hypoxia could cause the shortening of QT interval [8].

Preeclampsia (PE) is a pathological condition that initiated by abnormal utero-placental hemodynamic [2, 5]. Fetal deterioration in PE is associated with the maternal systemic vasoconstriction. The peculiarities of the fetal cardiac function regulation in this severe condition may become a prospective direction of the scientific research [3, 6, 11].

The aim of the investigation was a survey of the fetal HRV and ECG parameters in preeclamptic patients.

Materials and methods

The study protocol was approved by the bioethics committee of the medical academy of postgraduate education. Observed pregnant women were informed about the methods of the study, its aim, indications and eventual complications before inclusion to the study. All patients gave written consent to participate in the investigation.

It was performed fetal noninvasive ECG recordings in 94 pregnant women at 34-41 weeks of gestation. All pregnant women were divided into several clinical groups. In group I there were 28 women with physiological pregnancy and normal fetal condition. In group II

it was observed 36 pregnant women with mild and moderate PE. It was 30 patients with severe PE in group III.

Fetal HRV and fetal ECG parameters were obtained with the application of the first Ukrainian fetal noninvasive ECG monitor “Cardiolab Babycard” (Kharkiv, Ukraine). The quality of the Ukrainian ECG recordings was assessed by international experts [10]. The registration was carried out during periods of fetal activity for 10 minutes long. It was estimated the value of TP and its

spectral compounds: VLF, LF and HF. The temporal characteristics of the fetal HRV: SDNN, RMSSD, pNN50, AMo and SI were determined. The CTG waveform analysis was performed with STV (short term variability) and LTV (long term variability) scoring. The fetal ECG parameters such as: pQ, QT intervals and QRS complex duration, T wave amplitude, T/QRS ratio were calculated (fig. 1).



Fig. 1. The “window” in the “Cardiolab Babycard” program. The sample of fetal ECG parameters evaluation

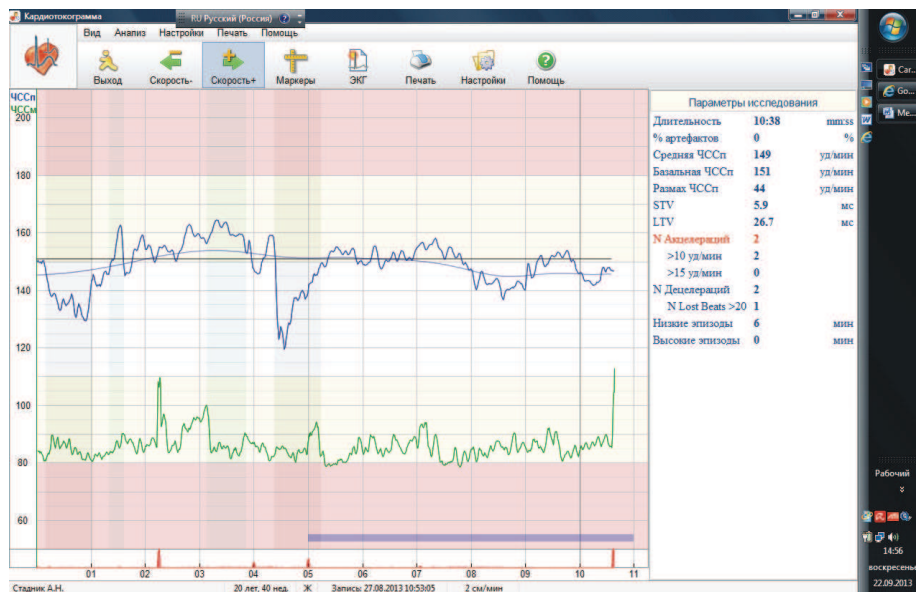


Fig. 2. The “window” in the “Cardiolab Babycard” program. Obtained CTG waveform (7 decelerations, basal rhythm – 151 per minute)

The results were processed by parametric statistical methods (mean – M, error – m) with the application of

statistics software package Excel adapted for biomedical research. The significance was set at $p < 0.05$.

Results

The obtained data has demonstrated the significant difference in fetal HRV parameters in the observed groups of patients (table 1). It was determined that TP and SDNN value was decreased in direct proportion to the severity degree of PE. So the autonomic tone was lower with the relative increase of LF branch in the patients of group II and group III. The predominance of the central sympathetic baroreflex mediated regulation of fetal hemodynamic was the main event in the PE induced scenario. The increased value of AMo and SI was associated with abnormal myocardial adrenergic stimulation. It has induced the metabolic starvation and

anaerobic processes activation. It was possible to speculate that the origin of the antenatal deceleration was presented in the pronounced augmentation of the sympathetic baroreflex (figure 2). The growth of the fetal heart rate range was not related to the increase of HRV but vice versa. The LTV value in PE was much more lower than in control group I (table 1).

The mean value of short term mediated parameters: RMSSD, pNN50, HF and STV was decreased in PE. The suppression of respiratory sinus arrhythmia was the predisposing condition to rigid rhythm formation. The lack of fetal vagal regulation was determined in observed patients with PE.

Table 1. Fetal HRV parameters in observed women

Index, units of measure	Group I	Group II	Group III
SDNN, ms	50.5 ± 9.4	30.6 ± 6.8*	12.3 ± 1.7*/**
RMSSD, ms	22.4 ± 3.4	14.2 ± 2.6*	8.1 ± 0.8*/**
pNN50, %	8.6 ± 1.0	5.6 ± 0.9*	2.1 ± 0.2*/**
SI, conv. units	140.6 ± 22.8	464.2 ± 52.4*	1450.2 ± 112.6*/**
AMo, %	38.2 ± 7.4	49.8 ± 6.2*	62.5 ± 6.6*/**
TP, ms ²	1634.8 ± 364.2	1048.4 ± 98.4*	384.8 ± 61.2*/**
VLF, ms ²	1346.2 ± 282.8	670.2 ± 84.6*	194.2 ± 23.8*/**
LF, ms ²	192.6 ± 31.1	312.2 ± 66.8*	143.6 ± 25.1*/**
HF, ms ²	95.2 ± 19.4	66.1 ± 14.9*	48.2 ± 14.1*/**
STV, ms	8.6 ± 2.0	6.2 ± 1.4*	3.8 ± 0.9*/**
LTV, ms	38.0 ± 9.4	26.4 ± 5.3*	20.4 ± 8.2*/**

* – the differences were statistically significant compared to the control group ($p < 0.05$)

** – the differences were statistically significant compared to the group II ($p < 0.05$)

Table 2. Fetal ECG parameters in observed women

Index, units of measure	Group I	Group II	Group III
pQ, ms	101.2 ± 16.4	105.2 ± 14.3*	86.7 ± 11.6*/**
QT, ms	202.4 ± 28.6	190.6 ± 17.4*	175.1 ± 23.2*/**
QRS, ms	64.0 ± 8.6	65.6 ± 10.4*	65.8 ± 0.2*
T, mcV	2.6 ± 0.4	4.5 ± 0.6*	7.2 ± 1.4*/**
T/QRS	0.04 ± 0.01	0.07 ± 0.02*	0.11 ± 0.04*/**

* – the differences were statistically significant compared to the control group ($p < 0.05$)

** – the differences were statistically significant compared to the group II ($p < 0.05$)

The pQ measurement has demonstrated gradual evolution of biphasic pattern (table 2). In case of mild and moderate PE the tendency to tachycardia was associated with prolonged pQ value. The deceleration pattern of CTG waveform in the group III was accompanied with significant pQ shortening. So the decrease of pQ consideration was a mirror of decelerations. A QT shortening was more obvious in the severe PE group and it has reflected myocardial hypoxic response to the increased

adrenergic stimulation. The value of QRS was almost stable. The rise of T wave has resulted in the increased T/QRS ratio. PE has spread its negative influence on sinus node and myocardium and changed bioelectrical processes.

Discussion

The obtained results have contributed to speculation that PE has formed the condition of sympathetic hyper-

stimulation. The onset of the fetal deterioration was initiated by humoral adrenergic substances and has mirrored in tachycardia. An increased fetal heart rate has its manifestation in the prolonged pQ [8-10]. The further baroreflex activation has changed fetal heart rate to the lower score. The decelerations and bradycardia have shortened pQ interval. A QT reduction was a significant marker of anaerobic myocardial metabolism and has presented a fetal distress. The lowered QT length and the rise of T wave amplitude were the symptoms of β -adrenergic receptors activation [8, 9]. The affected myocardium has reacted with high T/QRS ratio.

PE has induced the predominant role of catecholamines in the development of fetal distress. The activation of the sympathetic baroreflex and peripheral vasoconstrictor substances have negatively influenced on fetal hemodynamic regulation. It has resulted in myocardial hypoxic injury and suppressed sinus node response.

The application of fetal noninvasive ECG may contribute to the progression of the fetal well-being assessment. It could help to evaluate the influence of recent therapeutical approaches in perinatology on the fetal condition. Additional investigation of the maternal autonomic tone may provide more information about the role of maternal regulation in fetal growth.

Conclusions

- 1) The decreased fetal autonomic tone and predominance sympathetic regulation was marked in PE.
- 2) Fetal deterioration in PE was associated with pQ and QT shortening, increased T/QRS ratio and decelerations appearance.
- 3) Fetal distress was resulted in myocardial hypoxic injury and suppressed sinus node response.

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