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The Effect of Maternal Autonomic Regulation on fetal Hemodynamic Fluctuations and Growth

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Abstract

Objective. The development of fetal autonomic regulation has a crucial influence on fetal hemodynamics. Heart rate variability (HRV) captures the continual changes in sympathetic and parasympathetic balance.

The study was focused on the detection of the relationship between maternal HRV, fetal HRV, neonatal biometry, and Apgar score.

Methods. This study was performed among 71 pregnant women at 22-36 weeks. Fetal HRV data were collected using non-invasive fetal electrocardiography (NI-FECG) with the "Cardiolab Baby Card" system, which registered RR-intervals from the maternal abdominal wall over 30–60 minutes. Additionally, the study recorded neonatal biometry (birth weight, body length, and head circumference) and the 1-minute Apgar score.

Results. Maternal stress index (SI) demonstrated strong or moderate negative correlation with acceleration capacity (AC) maternal (r=-0.71; p<.001), deceleration capacity (DC) maternal (r=-0.78; p<.001), AC fetal (r=-0.42; p<.001), DC fetal (r=-0.37, p=.001), and STV (r=-0.27, p=.022). A positive moderate correlation was found in pairs Apgar score vs AC fetal (r=0.31; p=.009), DC fetal vs Apgar score (r=0.35, p=.003), STV (r=0.27; p=.024), and LTV (r=0.25; p=.038). A negative moderate correlation was detected between the SI fetal and Apgar score (r=-0.27; p=0.02). The multivariate logistic regression detected the relationship between maternal SI and neonatal body weight.

Conclusion. Maternal stress-related sympathetic activity is intrinsically linked to fetal autonomic regulation and neonatal outcomes. Utilizing maternal SI as a prognostic marker could provide clinicians with a fundamental tool for developing models to predict fetal growth restriction and ensure timely intervention.

Keywords: autonomic nervous regulation, heart rate variability, fetal growth restriction.

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INTRODUCTION

The maturation of the fetal autonomic nervous system is a critical factor in the regulation of fetal hemodynamics and development. During the end of the second trimester, "neurological maturation" occurs, which changes how the fetal heart rate reacts to movement. Heart Rate Variability (HRV) serves as a non-invasive instrument to investigate these autonomic tones, capturing the continuous shifts between sympathetic and parasympathetic balance. While fetal regulation is not a direct mirror of maternal mechanisms, evidence suggests a significant coupling between maternal and fetal autonomic tones, which may influence fetal growth[1]. The mother-placenta-fetus unit operates as a dynamic, multiscale network of physiological subsystems (cardiovascular, autonomic, endocrine, immune, neural). Transient, nonlinear couplings—mediated by placental conduction of hemodynamic fluctuations, hormones, cytokines, extracellular vesicles, and neurotransmitters—generate emergent adaptive states that orchestrate fetal neurodevelopment and maternal neuroplasticity. These interactions are bidirectional: maternal network dynamics program fetal brain networks, while fetal/placental signals reshape maternal brain architecture[2]. The issue of whether a maternal autonomic system is a controlling signal for fetal hemodynamics, growth, and maturation has not been resolved yet.

The nature of fetal and maternal cardiac coupling is not clearly understood. The opinion of disturbed fetal and maternal autonomic interaction in pre-eclampsia and other placenta-related disorders is known[3]. HRV is involved in the ergotrophic and trophotrophic reactions. The theory of possible dependence of fetal gestational age and the power of autonomic tone is already implemented. Therefore, HRV demonstrated a prospect in diagnosing fetal growth restriction (FGR)[4,5]. Probably, this method could be of use in the early detection of fetal excessive growth in gestational diabetes mellitus (GDM).

Fetal non-invasive electrocardiography (NI-FECG) is a prospective method for assessing fetal cardiac rhythm[6]. A range of HRV variables is rather wide. There are temporal or spectral parameters. Some of them refer to the reflection of sympathetic or parasympathetic branches of the autonomic nervous system. The sympathetic tone is increased in fetal deterioration. The complex parameter reflecting sympathetic stress-induced reaction is a stress index (SI)[7]. The phase rectified signal averaging – acceleration capacity and deceleration capacity (AC/DC) are useful in fetal well-being assessment[8]. Several parameters were obtained from cardiotocography (CTG) – short-term variations (STV) and long-term variations (LTV). They are known to be useful in diagnosing fetal distress in case of a nonreactive pattern of a non-stress test[9].

This study investigated the relationship between maternal HRV, fetal HRV, neonatal biometry, and Apgar scores.

MATERIALS AND METHODS

This descriptive cross-sectional study was performed among pregnant women who were admitted to Kharkiv Municipal Perinatal Center between 1 April 2024 and 31 October 2025. The data was obtained from the hospital automation system. Ethics approval was received from the Research Council and Ethical Committee of Kharkiv National Medical University, No28.0224p. Informed consent was obtained from all the patients. Patients from the department of maternal-fetal medicine were selected randomly. The technique of automated numbers was used. The FGR was diagnosed in case fetal weight parameters were lower than the 10th percentile according to ultrasound. The ultrasonic investigations were performed longitudinally following current clinical protocols [10]. Inclusion criteria: healthy pregnancy with appropriate fetal growth, FGR, GDM. Exclusion criteria: chromosomal abnormalities, multiple pregnancy, threatened preterm birth, severe pre-eclampsia, preexisting medical disorders like diabetes mellitus, metabolic syndrome, cardiac diseases, renal disease, and thyrotoxicosis.

The study was a descriptive cross-sectional investigation involving 71 pregnant women between 22 and 36 weeks of gestation. Participants were categorized into three groups: those with appropriate fetal growth, FGR, and GDM. Fetal HRV data were collected using NI-FECG with the "Cardiolab Baby Card" system, which registered RR-intervals from the maternal abdominal wall over 30–60 minutes. Several key metrics were analyzed: Stress Index (SI): a complex parameter used to reflect sympathetic stress-induced reactions. Phase-Rectified Signal Averaging (AC/DC): used to determine acceleration and deceleration capacities for assessing fetal well-being[11]. Short-term and Long-term Variations (STV/LTV): variables derived from cardiotocography (CTG) tracing to diagnose

potential fetal distress. Additionally, the study recorded neonatal biometry (birth weight, body length, and head circumference) and the 1-minute Apgar score. All recordings were performed during maternal rest after meals in a recumbent position.

The Statistical Package for Social Sciences (SPSS) program (IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.) was used for statistical analysis. Results were presented as means and standard deviations for numerical variables, and frequencies and percentages for categorical data. The suitability of numerical variables to normal distribution was evaluated using skewness values and histograms.

The Independent sample t-test was used in comparing numerical variables fitting normal distribution. Variables that did not conform to normal distribution were analyzed by the Mann-Whitney U test. The Chi-Square (or Fisher's exact) test was used for comparing categorical variables. Depending on their distribution, Spearman or Pearson correlation analysis was used to assess the relationships between numerical variables. For multivariate examinations, a logistic regression analysis with the entered model was used. Sample size was calculated using confidence level 95 % and margin of error 5 %. A p-value of <0.05 was considered sufficient for statistical significance.

RESULTS

The appropriate fetal growth was found in 52 patients. FGR was diagnosed in 12 cases. The possible fetal overgrowth (tendency to macrosomy) was detected in 7 pregnant women with GDM. The mean age among the study population was 26.82±7.64. The quantity of primigravidae was 63.38 %. The mean interval between HRV registration and delivery was 5.38±1.92 days. The data obtained showed differences between normal growth, FGR, and GDM subgroups (Table 1). The maximal quantitative changes of the maternal and fetal HRV parameters, neonatal biometry were detected in GDM. However, the small number of cases of GDM did not allow statistical significance to proceed. The lowest Apgar score was in the FGR subgroup.

Table 1. Mean values of the variables in the study population.

The correlation between maternal and fetal HRV, neonatal biometry, and Apgar score is shown in Table 2. The analysis revealed significant correlations between maternal and fetal parameters. Maternal SI correlations: the maternal SI demonstrated a strong negative correlation with maternal AC (r=-0.71) and DC (r=-0.78), as well as moderate negative correlations with fetal AC (r=-0.42), fetal DC (r=-0.37), and STV (r=-0.27). Apgar score relationships: fetal AC (r=0.31), DC (r=0.35), STV (r=0.27), and LTV (r=0.25) all showed positive moderate correlations with the 1-minute Apgar score. In contrast, the fetal SI was negatively correlated with the Apgar score (r=-0.27).

Table 2. The linear correlation and significance between the detected variables in the study population.

Growth and Biometry: while direct correlations between HRV and biometry were not initially apparent, multivariate logistic regression identified a significant relationship between the maternal SI and neonatal birth weight (p=0.044) (Table 3). The model itself was meaningful. The different models with maternal or fetal AC, DC, STV, and LTV did not show any relationships with biometry variables.

Table 3. Multivariate logistic regression model with SI coefficient.

DISCUSSION

Maternal HRV demonstrates a biphasic pattern of changes during pregnancy. Parasympathetic tone is activated till the second half of gestation. Then, sympathetic regulation resetting occurs with its gradual increase [12]. However, excessive growth of sympathetic tone is associated with pre-eclampsia [13]. Our study demonstrated the coupling between maternal SI and fetal HRV variables: AC, DC, and STV. The negative correlation supported the speculation that the maternal sympathetic branch of autonomic regulation could suppress fetal HRV. Thus, maternal adrenal stress-related influences can contribute to fetal deterioration. The obvious mechanism of this link is unknown. However, it could be thought that maternal sympathetic overactivity could affect fetal hemodynamic fluctuations through the placental bed [14, 15, 16].

The correlation between Apgar score and fetal AC, DC, SI, STV, and LTV supported the use of these variables in diagnosing fetal distress [17]. These data provide a prospect for the use of NI-FECG in electronic fetal monitoring.

Therefore, our study showed that maternal SI has a moderate negative correlation with fetal AC, DC and STV. This sympatho-adrenal mediated variable was a controlling signal for fetal HRV. Fetal AC, DC, STV,

Table 1. Mean values of the variables in the study population

Parameter	Fetal growth pattern	Frequency	Mean	Std. Deviation	Minimum	Maximum
AC maternal	Appropriate	52	8.51	2.88	3.54	17.44
	FGR	12	8.57	2.91	4.51	15.44
	GDM	7	7.62	2.47	3.75	10.69
DC maternal	Appropriate	52	8.7	2.8	4.29	17.13
	FGR	12	8.2	2.02	4.73	10.85
	GDM	7	7.39	1.83	3.9	9
AC fetal	Appropriate	52	1.98	0.53	1.18	3.71
	FGR	12	1.8	0.98	0.77	3.54
	GDM	7	1.99	0.7	1.35	3.31
DC fetal	Appropriate	52	2.37	0.64	1.24	4.04
	FGR	12	2.16	1.3	0.78	4.37
	GDM	7	2.28	0.89	1.46	3.98
SI maternal	Appropriate	52	165.35	104.85	32	523
	FGR	12	194.33	105.42	57	457
	GDM	7	225	237.25	59	744
SI fetal	Appropriate	52	1005.02	500.86	326	2623
	FGR	12	1494.92	1305.72	251	4569
	GDM	7	1187.86	703.17	460	2503
STV	Appropriate	52	7.41	2.55	2.5	15
	FGR	12	6.5	4.04	1.2	12.2
	GDM	7	7.3	3.27	2.8	11.9
LTV	Appropriate	52	37.71	14.21	17.1	95.6
	FGR	12	34.68	19.86	9.3	71
	GDM	7	37.61	17.62	13.7	64.4
Neonatal weight	Appropriate	52	3247.02	602.42	1800	4650
	FGR	12	1709.17	917.58	410	2680
	GDM	7	3800.71	914.45	2665	5530
Neonatal body length	Appropriate	52	50.94	4.17	39	61
	FGR	12	39.75	8.07	27	50
	GDM	7	51.71	4.86	47	62
Head circumference	Appropriate	52	34.5	1.74	30	37
	FGR	12	27.83	5.11	19	34
	GDM	7	35.57	1.4	34	38
Apgar score	Appropriate	52	7.87	0.91	5	9
	FGR	12	5.5	2.91	0	8
	GDM	7	7.43	1.13	5	8

and LTV demonstrated a positive moderate correlation with Apgar score. Fetal SI had a negative moderate correlation with Apgar score. The logistic regression did not find any relationship between fetal HRV and parameters of neonatal biometry. However, the link between maternal SI and body weight at birth was supported. This fact captured the regulatory trophotrophic influence of sympathetic regulation on fetal growth. The obtained result could be fundamental for developing prognostic models using maternal SI as a possible biophysical marker for FGR.

The results support the theory that maternal autonomic activity acts as a controlling signal for fetal HRV. Specifically, maternal sympathetic overactivity—often associated with stress or conditions like pre-eclampsia—may suppress fetal HRV by affecting hemodynamic fluctuations through the placental bed. This connection highlights the regulatory "trophotrophic" influence that the maternal sympathetic branch has on fetal growth. The high predictive value of fetal AC, DC, and SI for neonatal Apgar scores suggests that NI-FECG is a robust tool for electronic fetal monitoring and the early detection of distress. Furthermore, maternal SI shows great promise as a biophysical marker for diagnosing and managing fetal growth restriction[18].

The lower fetal HRV is a moderate-to-strong biomarker of short-term compromise and signals long-term neurodevelopmental vulnerability, especially in

Table 2. The correlation and significance between the detected variables in the study population

Parameter	Cor-relation	AC maternal	DC maternal	AC fetal	DC fetal	SI maternal	SI fetal	STV	LTV	Neonatal body weight	Neonatal body length	Head circum-ference	Apgar score
AC maternal	Cor-relation	1	0.95	0.19	0.15	-0.71	-0.01	0.05	0	-0.09	-0.07	-0.1	0.01
	p		<.001	.115	.204	<.001	.943	.685	.981	.453	.588	.411	.902
DC maternal	Cor-relation	0.95	1	0.21	0.18	-0.78	0.01	0.07	-0.02	-0.06	-0.05	-0.05	0.03
	p	<.001		.079	.132	<.001	.923	.581	.84	.635	.657	.681	.792
AC fetal	Cor-relation	0.19	0.21	1	0.91	-0.42	-0.64	0.91	0.81	-0.04	0.04	0.18	0.31
	p	.115	.079		<.001	<.001	<.001	<.001	<.001	.736	.747	.135	.009
DC fetal	Cor-relation	0.15	0.18	0.91	1	-0.37	-0.63	0.91	0.83	-0.05	0.05	0.15	0.35
	p	.204	.132	<.001		.001	<.001	<.001	<.001	.672	.682	.216	.003
SI maternal	Cor-relation	-0.71	-0.78	-0.42	-0.37	1	0.05	-0.27	-0.11	-0.03	-0.01	-0.06	-0.1
	p	<.001	<.001	<.001	.001		.674	.022	.342	.819	.925	.643	.411
SI fetal	Cor-relation	-0.01	0.01	-0.64	-0.63	0.05	1	-0.74	-0.84	0.1	-0.03	-0.14	-0.27
	p	.943	.923	<.001	<.001	.674		<.001	<.001	.413	.807	.254	.02
STV	Cor-relation	0.05	0.07	0.91	0.91	-0.27	-0.74	1	0.9	-0.05	0.04	0.19	0.27
	p	.685	.581	<.001	<.001	.022	<.001		<.001	.696	.746	.116	.024
LTV	Cor-relation	0	-0.02	0.81	0.83	-0.11	-0.84	0.9	1	-0.03	0.03	0.22	0.25
	p	.981	.84	<.001	<.001	.342	<.001	<.001		.8	.791	.067	.038
Neonatal body weight	Cor-relation	-0.09	-0.06	-0.04	-0.05	-0.03	0.1	-0.05	-0.03	1	0.78	0.76	0.46
	p	.453	.635	.736	.672	.819	.413	.696	.8		<.001	<.001	<.001
Neonatal body length	Cor-relation	-0.07	-0.05	0.04	0.05	-0.01	-0.03	0.04	0.03	0.78	1	0.65	0.62
	p	.588	.657	.747	.682	.925	.807	.746	.791	<.001		<.001	<.001
Head circum-ference	Cor-relation	-0.1	-0.05	0.18	0.15	-0.06	-0.14	0.19	0.22	0.76	0.65	1	0.5
	p	.411	.681	.135	.216	.643	.254	.116	.067	<.001	<.001		<.001
Apgar score	Cor-relation	0.01	0.03	0.31	0.35	-0.1	-0.27	0.27	0.25	0.46	0.62	0.5	1
	p	.902	.792	.009	.003	.411	.02	.024	.038	<.001	<.001	<.001	

high-risk pregnancies[5-7, 16]. This supports its role in Network Physiology-guided monitoring alongside maternal-fetal coupling and placental hemodynamics. Preserved or improving fetal HRV trajectories appear protective. Further pooled analyses would benefit from raw data sharing.

The limitations of the study include the following points: small Sample Size in Specific Subgroups. A primary limitation was the small number of cases within the GDM group, which consisted of only 7 participants. This small sample size prevented the researchers from achieving statistical significance for changes in maternal and fetal HRV parameters and neonatal biometry specifically within the GDM subgroup.

Restrictive Exclusion Criteria: the study's generalizability is limited by the exclusion of several high-risk conditions. Specifically, the research excluded patients with chromosomal abnormalities, multiple pregnancies, threatened preterm birth, and severe pre-eclampsia. It also excluded those with preexisting medical disorders such as diabetes mellitus, metabolic syndrome, cardiac diseases, renal disease, and thyrotoxicosis.

Temporal Gap Before Delivery: There was a mean interval of 5.38±1.92 days between the registration of HRV and the actual delivery. This time gap could potentially influence the correlation between the recorded HRV data and the final neonatal outcomes.

Initial Statistical Challenges: In simple correlation analysis, no significant relationship was initially detected between maternal or fetal HRV variables and neonatal biometry. A significant link between maternal SI and neonatal birth weight only became apparent when using multivariate logistic regression.

Single-Center Scope: The data were obtained exclusively from the Kharkiv Municipal Perinatal Center, which may limit the broader application of the findings to different populations or healthcare settings.

Table 3. Multivariate logistic regression model with SI coefficient

Parameter	Unstandardized Coefficients	Standardized Coefficients				95% confidence interval for B	
Model	B	Beta	Standard error	t	p	lower bound	upper bound
(Constant)	123.51		233.59	0.53	.599	-343.92	590.94
AC maternal	0.63	0.01	10.97	0.06	.954	-21.32	22.58
DC maternal	-30.22	-0.65	11.63	-2.6	.012	-53.48	-6.95
AC fetal	-56.92	-0.3	56.8	-1	.32	-170.58	56.75
DC fetal	-10.88	-0.07	44.75	-0.24	.809	-100.43	78.66
SI fetal	0.01	0.07	0.02	0.51	.61	-0.03	0.06
STV	4.31	0.1	15.25	0.28	.778	-26.21	34.84
LTV	-0.19	-0.02	1.74	-0.11	.912	-3.67	3.28
Neonatal body weight	-0.07	-0.51	0.03	-2.06	.044	-0.13	0
Neonatal body length	4.31	0.23	4.97	0.87	.389	-5.63	14.26
Head circumference	14.54	0.43	8.45	1.72	.091	-2.38	31.45
Apgar score	-12.33	-0.17	13.31	-0.93	.358	-38.96	14.3

CONCLUSION

Maternal stress-related sympathetic activity is intrinsically linked to fetal autonomic regulation and neonatal outcomes. Utilizing maternal SI as a prognostic marker could provide clinicians with a fundamental tool for developing models to predict fetal growth restriction and ensure timely intervention.

CLINICAL SIGNIFICANCE

The study establishes that the maternal SI can serve as a valuable biophysical marker for the early detection of FGR. It highlights that NI-FECG is an effective tool for monitoring fetal autonomic variables, such as acceleration and deceleration capacities, which are significant predictors of neonatal Apgar scores. These findings indicate that maternal sympathetic overactivity may influence fetal development through the placental bed, suggesting that maternal autonomic monitoring offers a novel pathway for identifying fetal distress and improving the management of high-risk pregnancies.

List of abbreviations.

FGR – fetal growth restriction; HRV – heart rate variability; NI-ECG – non-invasive electrocardiography; STV – short-term variation; AC – acceleration capacity; DC – deceleration capacity; SI – stress index; CTG – cardiotocography; SPSS – Statistical Package for Social Sciences.

Declarations

Ethics approval and patient consent to participate The study protocol received official ethics approval from the Research Council and Ethical Committee of Kharkiv National Medical University (No. 28.0224p). All participants provided informed consent prior to their inclusion in the study.

Patient Consent for publication Informed consent was obtained from all individual patients included in the study.

Animal Studies Not applicable. This research was conducted exclusively involving human participants, specifically 71 pregnant women.

Author contributions Igor Lakhno, as the primary and corresponding author, was responsible for the study's objective, methodology, data analysis using the Statistical Package for Social Sciences (SPSS), and the interpretation of the relationship between maternal and fetal heart rate variability.

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Conflict of interest

The sources do not explicitly state any conflicts of interest for the author.

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Availability of data and materials

The data utilized for this research were retrieved from the hospital automation system of the Kharkiv Municipal Perinatal Center. Additional information regarding data availability is not specified in the sources.

References

1. Widatalla N, Khandoker A, Alkhodari M, Koide K, Yoshida C, Kasahara Y, Kimura Y, Saito M. Similarities between maternal and fetal RR interval tachograms and their association with fetal development. Front Physiol. 2022 Nov 21;13:964755. doi: 10.3389/fphys.2022.964755.
2. Zöllkau J, Dölker EM, Schmidt A, Schneider U, Hoyer D. Dependencies between maternal and fetal autonomic tone. J Perinat Med. 2019 Apr 24;47(3):323-330. doi: 10.1515/jpm-2018-0221.
3. Lakhno I. Autonomic imbalance captures maternal and fetal circulatory response to pre-eclampsia. Clin Hypertens. 2017 Feb 8;23:5. doi: 10.1186/s40885-016-0061-x.
4. Smith V, Nair A, Warty R, Sursas JA, da Silva Costa F, Wallace EM. A systematic review on the utility of non-invasive electrophysiological assessment in evaluating for intra uterine growth restriction. BMC Pregnancy Childbirth. 2019;19(1):230. doi: 10.1186/s12884-019-2357-9.
5. Stroux L, Redman CW, Georgieva A, et al. Doppler-based fetal heart rate analysis markers for the detection of early intrauterine growth restriction. Acta Obstet Gynecol Scand. 2017;96(11):1322-1329. doi: 10.1111/aogs.13228.
6. Liu B, Ridder A, Smith V, et al. Feasibility of antenatal ambulatory fetal electrocardiography: a systematic review. J Matern Fetal Neonatal Med. 2023;36(1):2204390. doi: 10.1080/14767058.2023.2204390.
7. Hoyer D, Żebrowski J, Cysarz D, et al. Monitoring fetal maturation-objectives, techniques and indices of autonomic function. Physiol Meas. 2017;38(5):R61-R88. doi: 10.1088/1361-6579/aa5fca.
8. Rivolta MW, Barbieri M, Stampalija T, Sassi R, Frasch MG. Relationship Between Deceleration Morphology and Phase Rectified Signal Averaging-Based Parameters During Labor. Front Med (Lausanne). 2021 Nov 25;8:626450. doi: 10.3389/fmed.2021.626450.
9. Gatellier MA, De Jonckheere J, Storme L, Houfflin-Debarge V, Ghesquiere L, Garabedian C. Fetal heart rate variability analysis for neonatal acidosis prediction. J Clin Monit Comput. 2021;35(4):771-777. doi: 10.1007/s10877-020-00535-6.
10. Leal CRV, Rezende KP, Macedo EDCP, Rezende GC, Corrêa Júnior MD. Comparison between Protocols for Management of Fetal Growth Restriction. Rev Bras Ginecol Obstet. 2023;45(2):96-103. doi: 10.1055/s-0043-1764493.
11. Ponsiglione AM, Cosentino C, Cesarelli G, Amato F, Romano M. A Comprehensive review of techniques for processing and analyzing fetal heart rate signals. Sensors (Basel). 2021;21(18):6136. doi: 10.3390/s21186136.
12. Bester M, Joshi R, Mischi M, van Laar J, Vullings R. On the distinct differences in autonomic regulation between pregnant and non-pregnant women - a heart rate variability analysis. Physiol Meas. 2023;44(5). doi: 10.1088/1361-6579/acce1e.
13. Musa SM, Adam I, Lutfi MF. Heart Rate Variability and Autonomic Modulations in Preeclampsia. PLoS One. 2016 Apr 4;11(4):e0152704. doi: 10.1371/journal.pone.0152704.
14. Byron GS, Hilmert CJ, Strahm AM. Heart rate variability during pregnancy moderates the impact of depressive symptoms on fetal growth. J Affect Disord. 2025; 370:381-384. doi: 10.1016/j.jad.2024.11.008.
15. Lakhno I V. The hemodynamic repercussions of the autonomic modulations in growth-restricted fetuses. Alexandria Journal of Medicine. 2019; 53;4: 333-336. doi: 10.1016/j.ajme.2016.12.007.
16. Ghelmene EA, Serban N, Russu M. Sonographic placental aspects in fetal growth restriction. Medicina Moderna - Modern Medicine. 2024; 31(1):7-12. doi: 10.31689/rmm.2024.31.1.7
17. Chiera M, Cerritelli F, Casini A, Barsotti N, Boschiero D, Cavigioli F, Corti CG, Manzotti A. Heart Rate Variability in the Perinatal Period: A Critical and Conceptual Review. Front Neurosci. 2020;14:561186. doi: 10.3389/fnins.2020.561186.
18. Gashi A. Recommendations for prenatal Doppler in daily obstetric practice - a review. Medicina Moderna - Modern Medicine. 2023; 30(3):167-173. doi: 10.31689/rmm.2023.30.3.167.