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DOPPLER ASSESSMENT OF FETAL DIASTOLIC DYSFUNCTION

Abstract. The article presents the results of a study of the pulmonary artery and vein in the assessment of cardiac dysfunction in fetuses with cardiomegaly. The Doppler in the artery and vein was obtained in one control volume. Acceleration time (AT), ejection time (ET), isovolumic contraction time (IVCT), isovolumic relaxation time (IVRT), duration of the A wave (Adur), atrial contraction time (ACT), the max velocity in the PV at the moment of opening of the atrioventricular valve (at point AVO), the max velocity in the PV in ventricular systole (at point S) and AVO/S, AT/ET, ACT/IVRT, IVRT/ET were also calculated. In diastolic dysfunction, there was a shortening IVRT, lengthening of the atrial time, and a decrease in the AVO/S ratio. Systolic dysfunction of the heart was characterized by a pronounced dicrotic notch in the arterial spectrum, a decrease in the wave after the closure of the semilunar valve/or retrograde blood flow in the ventricular diastole.

Keywords: diastolic dysfunction, fetus, Doppler sonography, pulmonary artery, pulmonary vein.

In recent years, many researchers have increasingly paid attention to the assessment of cardiac dysfunction in fetuses: methods such as Speckle tracking echocardiography are used, which have great prospects due to the possibility of obtaining measurements of longitudinal, radial and transverse deformation of the myocardium, Tissue Doppler (TDI), magnetic resonance imaging [1; 2]. However, unfortunately, there are still limitations in the widespread availability of these methods in the health care system.

Dopplerography is a relatively in expensive, accessible and non-invasive method in the diagnosis of myocardial dysfunction. To date, to assess global cardiac dysfunction in children and adults, including fetuses, the calculation of the MPI index (myocardial performance index) is widely used [3]. An analysis of the literature data on the use of the MPI index in fetuses showed a large scatter of the data obtained [4–6], which are apparently related to the technical aspects of the measurements. The development of new, effective, accessible, and easily reproducible methods for assessing fetal cardiac dysfunction is undoubtedly an urgent task of prenatal diagnosis. Our approach to the diagnosis of fetal cardiac dysfunction is based on the assessment of the Doppler spectrum of blood flow in the pulmonary arteries and veins.

Material and methods

In a prospective study, the main group included 69 pregnant women (5 patients with multiple pregnancies) who were observed at the Republican Scientific and Practical Center for Obstetrics and Gynecology of the Ministry of Health of the Republic of Uzbekistan, with a pathology of the heart in the fetus. A total of 74 fetuses were studied, including 51 fetuses (group 1) with cardiomegaly of various origins (CHD, FGR, fetal edema, hemolytic anemia of the fetus, twin-twin transfusion syndrome, diabetic macrosomia) and 23 fetuses with CHD without cardiomegaly (group 2). The control group consisted of 63 patients without extragenital pathology and with 10 to 70, on average 36.5 ± 6.5 percentile. Ultrasound examinations were carried out on Samsung WS80A (Korea) expert-class devices using convex probes with a frequency of 3.5–5 MHz.

All fetuses underwent standard fetometry, including biparietal head size, head and abdomen circumference, and thigh length. Cardio – femoral index (the ratio of the width of the heart at the level of the atrioventricular valves to the length of the thigh) was used to assess cardiomegaly, and Color Doppler mapping (CD) was used to study intracardiac hemodynamics. In all fetuses, when scanning through a four-chamber section, the pulmonary veins were removed, while the control volume of the spectral Doppler was set to capture both the pulmonary vein and the pulmonary artery. Using spectral doppler graph, the following parameters were measured: acceleration time (AT, acceleration time) and ejection time (ET, ejection time) in LA, isovolumic contraction time (IVCT), isovolumic relaxation time (IVRT, isovolumic relaxation time), duration of the A-wave in the PV spectrum (A dur, duration of the A wave), atrial contraction time (ACT), maximum velocity in the PV at the time of opening of the atrioventricular valve (at the AVO point, atrioventricular valve opening), maximum velocity in the PV in ventricular systole (at point S, systole) (Fig. 1), and also calculated such indicators as the ratio of velocity in the pulmonary artery at the moment of opening of the atrioventricular valve to the maximum velocity in ventricular systole AVO/S, the ratio of acceleration time to ejection time AT/ET, the ratio of atrial contraction time to isovolumetric relaxation time ACT/IVRT, and the ratio of isovolumetric relaxation time relaxation by IVRT/ET ejection time. Statistical data were calculated using the Statistics 23 program. The data of the first and second groups were compared with the control group, the differences in the groups were considered statistically significant at $p < 0.05$.

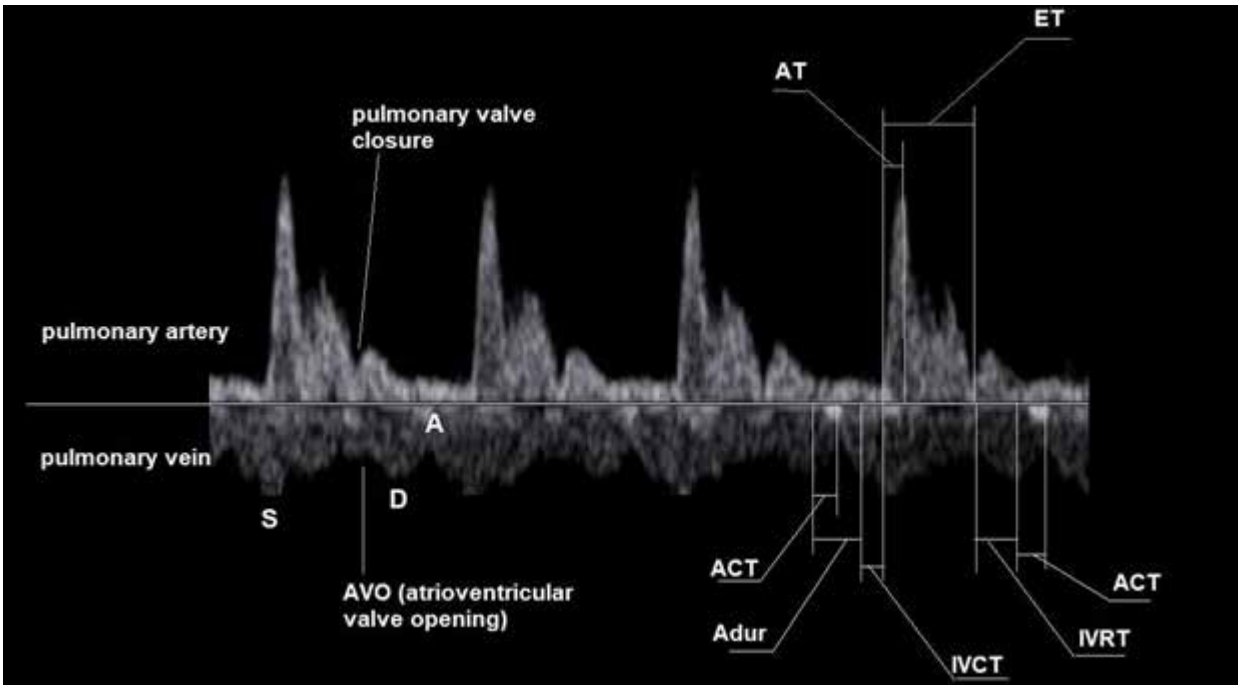

Figure 1. Doppler spectrum of blood flow in the pulmonary artery (above the isoline) and pulmonary vein (below the isoline)

Results

The indicators of gestational age of the first group were 28.2 ± 3.0, in the second group 26.04 ± 4.4 weeks, in the control group 29.47 ± 4.17 weeks. CFI in the control group 0.52 ± 0.02, in the first group 0.64 ± 0.04, in the second group 0.50 ± 0.01.

The mean value of AT in the first group was 33.0 ± ± 2.6 ($p < 0.05$), in the second group 30.2 ± 1.7 ($p = = 0.55$), in the control group 29.9 ± 1.9; ET – 180.0 ± ± 8.9 ($p < 0.05$), 175.6 ± 8.4 ($p = 0.90$), 175.9 ± 11.9; IVCT – 41.8 ± 4.0 ($p < 0.05$), 30.3 ± 7.5 ($p = 0.56$), 31.6 ± 8.3; IVRT – 72.7 ± 11.1 ($p < 0.05$), 82.0 ±

± 3.0 (p > 0.05), 84.1 ± 7.7; A dur 169.9 ± 30.0 (p < < 0.05), 163.8 ± 21.3 (p = 0.17), 157.6 ± 19.7; AST – 92.6 ± 11.4 (p < 0.01), 67.0 ± 5.7 (p< 0.05), 74.0 ± ± 16.2; AVO – 9.7±2.7 (p < 0.05), 12.0 ± 3.1 (p = = 0.79), 12.5 ± 3.2; S – 25.6 ± 3.6 (p < 0.05), 21.0 ± ± 4.2 (p = 0.81), 20.7 ± 3.2, respectively (table 1–3).

We also evaluated IVRT/ET, AST/IVRT ratios to assess diastolic dysfunction of the heart. The mean values of IVRT/ET in the first group were 0.41 ± ± 0.17 (CI 68% 0.24–0.58), in the second group 0.47 ± ± 0.03 (CI 95% 0.40–0.54), in the control group 0.48 ± 0.08 (95% CI 0.40–0.54), p-values for the first group compared with the control group were p < 0.01 (there are statistically significant differences between these groups), for the second group p = 0.12. For the ACT/IVRT indicator, the mean values in the first group were 1.32 ± 0.3 (CI 68% 0.68–1.95), in the second group 0.82 ± 0.09 in the control group – 0.90 ± 0.2 (95% CI 0.37–1.42), p<0.01 for the first group, p > 0.05 for the second group.

The presence of cardiomegaly in hemolytic anemia in the fetus was accompanied by changes in the spectrum of the pulmonary vein, caused by diastolic dysfunction (Fig. 2), there was a decrease in the ratio of the velocity at the point of opening of the atrioventricular valve to the maximum velocity during ventricular diastole AVO/S0.31 ± 0.03 with CI 95% of the control group 0.42–0.74. Atrial time A dur was also increased by 207.3 ± 11.1 ms (CI 95% of the control group 109–205 ms). IVRT/ET and AST/IVRT ratios were within 95% CI of the control group – 0.53 ± ± 0.03 (0.40–0.54) and 1.11±0.17 (0.37–1.42), respectively, despite the fact that the indicators of ACT 118.8 ± 17.6 ms (36–112 ms), IVRT 107.8 ± 5.4 ms (62–105 ms) and ET 206.7 ± 18.6 ms (146–205 ms) were higher than the values of the control group. The AT/ET ratio was increased to 0.22 ± 0.01 (95% CI 0.15–0.19 control group) (table 1–3). The group with fetal growth retardation had a wide range of values. In cases of an early form of IGR, there was a decrease in the IVCT value, an increase in the IVRT, AST and A dur values, while AT, ET corresponded to the values of the control group. The spectrum of the pulmonary vein in the late form of IGR showed shortening of IVRT, ET, AST and lengthening of A dur. The AT values had a spread from 23 ms to 57 ms, averaging 33.1 ± ± 5.3 ms (24.2–34.7). The mean ET in this subgroup was 166.3±18.9 ms (146–205 ms), IVCT 39 ± 5.5 ms (9.4–54.3 ms), IVRT 74.6±16.4 ms (62 .4–105.8 ms), ACT 90.5 ± 24.9 ms (36–112 ms). In diabetic macrosomia, a normal spectrum of the pulmonary artery and vein was recorded, with only a slight deviation in the indicators of the systolic function of the heart – an increase in the AT/ET ratio, apparently due to volume overload. Mild valvular regurgitation was noted in the pulmonary valve. The mean value in the group with diabetic macrosomia was AT 30.4 ± 1.1 ms (24.2–34.7), ET 192 ± 9.2 ms (146–205 ms), IVCT 45.7 ± 4.0 ms (9.4–54.3 ms), IVRT 70.7 ± 16.4 ms (62.4–105.8 ms), ACT 90.5 ± 24.9 ms (36–112 ms). In fetuses with a large weight for gestational age, in the absence of diabetes in a woman, systolic AT function and the AT/ET ratio were increased, indicating volume overload of the heart during macrosomia. The spectrum of the umbilical artery, ductus venosus, and middle cerebral artery were within normal limits.

Diastolic dysfunction of the heart in fetuses with twin-twin transfusion syndrome was also manifested by an increase in atrial time, acceleration time, and a decrease in isovolumetric contraction time. Changes in the parameters of the pulmonary vein AT, ET, IVST, IVRT correlated with CFI. With fetal edema (pronounced hydrothorax), if the CFI was not increased, then the spectrum of the pulmonary vein and artery remained normal (Fig. 3). In the venous duct, there was an increase in the pulsatile index PI 0.84 with CFI 0.49. The spectrum of the pulmonary artery and vein indicated the absence of cardiac dysfunction: IVRT / ET 0.52 (95% CI of the control group 0.40–0.54), AST / IVRT 0.64 (95% CI of the control group 0.37–1.42), AVO/S0.52 (95% CI of control group 0.42–0.74), AT/ET 0.19 (95% CI of control group 0.15–0.19).

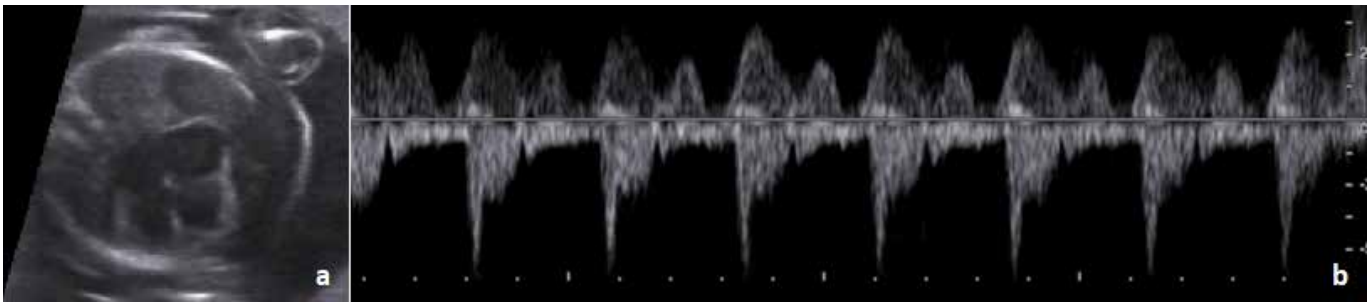

Figure 2. Diastolic dysfunction in a fetus with hemolytic anemia at 35 weeks of gestation, cardiomegaly (CFI 0.65). In the spectrum of the pulmonary vein (above the isoline), there was a decrease in the ratio of the velocity at the point of opening of the atrioventricular valve to the maximum velocity during ventricular diastole AVO/S0.25 (0.42–0.74) and an increase in atrial time A dur 207 ms (109–205 ms). a) cardiomegaly b) Doppler spectrum of pulmonary artery and vein

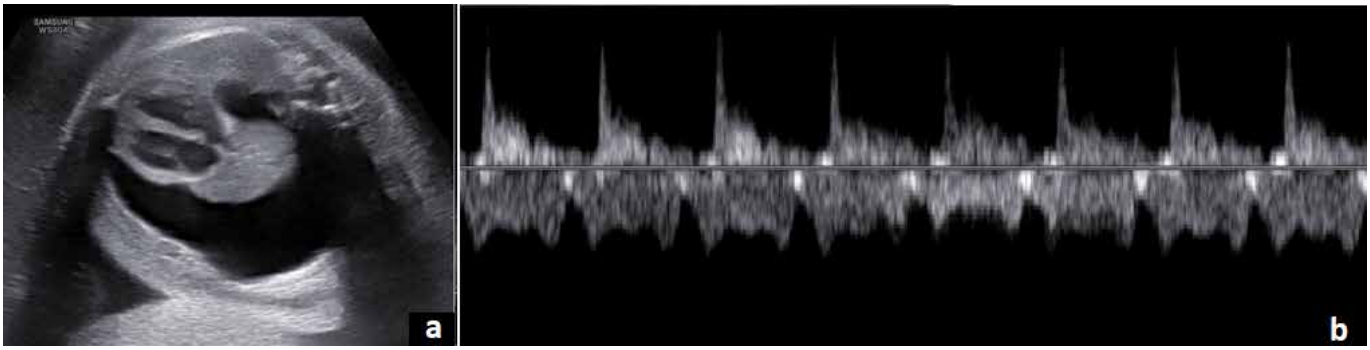

Figure 3. Doppler spectrum of blood flow in the pulmonary artery (above the isoline) and vein (below the isoline) in a fetus of 30 weeks of gestation with severe idiopathic hydrothorax, without cardiomegaly. The spectrum of the pulmonary vein is normal

Discussion

The proposed method for assessing cardiac dysfunction is based on obtaining the blood flow of the pulmonary artery and vein in the same spectrum. Technically, this is easy to do, since the pulmonary veins in the area of confluence with the left atrium are located next to the pulmonary arteries. In a four-chamber section, it is necessary to bring out the pulmonary veins on one side and capture the pulmonary vein together with the pulmonary artery into the control volume. Normal spectra of the pulmonary artery and vein in the second and third trimesters are shown in Figure 4.

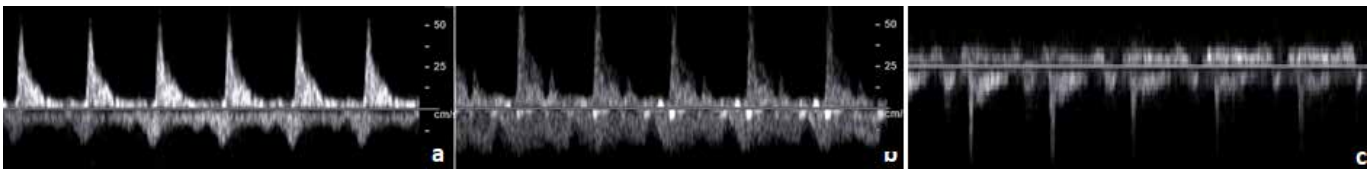

Figure 4. Spectrum of the pulmonary artery and vein at 20 weeks (a) and 37 weeks (b) of gestation during normal pregnancy. In the third trimester of pregnancy in the spectrum of the pulmonary artery, the closing point of the pulmonary valve and the antegrade wave following it become more pronounced due to the elasticity of the vessel walls. Reverse in the pulmonary vein (c) in a fetus of 19 weeks of gestation with a normal course of pregnancy

Using the spectrum of the pulmonary artery and vein, we estimated the **M**PI (myocardial performance index). The time of isovolumetric relaxation was calculated from the point of opening of the atrioventricu-

Table 1.– Comparison of mean values of AT, ET, IVCT, IVRT in the pulmonary artery and vein in the first and control groups

	AT, ms			ET, ms			IVCT, ms			IVRT, ms		
	First group	Control group	p-value	First group	Control group	p-value	First group	Control group	p-value	First group	Control group	p-value
CHD, n=11	33.2±5.0	29.9±2.6	p>0.05	170.0±11.4	175.9±11.9	p>0.05	41.8±3.4	31.6±8.3	p>0.05	83.6±5.3	84.1±10.8	p>0.05
Early FGR, n=13	33.15±8.8	29.9±2.6	p>0.05	166.3±16.9	175.9±11.9	p>0.05	39.0±7.3	31.6±8.3	p>0.05	74.6±21.3	84.1±10.8	p>0.05
Fetal edema (not immune), n=3	33.0±3.6	29.9±2.6	p>0.05	191.3±25.7	175.9±11.9	p>0.05	43.6±1.1	31.6±8.3	p>0.05	102.3±7.6	84.1±10.8	p>0.05
Fetal hemolytic anemia, n=7	43.8±6.7	29.9±2.6	p<0.05*	206.7±30.1	175.9±11.9	p<0.05*	43.7±14.9	31.6±8.3	p>0.05	107.8±7.7	84.1±10.8	p<0.05*
TTTS donor, n=5	33.0±1.0	29.9±2.6	p>0.05	177.6±6.2	175.9±11.9	p>0.05	41.8±6.8	31.6±8.3	p>0.05	87.0±3.3	84.1±10.8	
TTTS recipient, n=5	31.6±1.8	29.9±2.6	p>0.05	179.2±8.9	175.9±11.9	p>0.05	41.8±7.2	31.6±8.3	p>0.05	57.2±2.1	84.1±10.8	p<0.05*
Diabetic macrosomia, n=7	30.4±1.6	29.9±2.6	p>0.05	192.4±11.1	175.9±11.9	p>0.05	45.7±5.7	31.6±8.3	p>0.05	70.7±1.7	84.1±10.8	p>0.05

p < 0.05 there are statistically significant differences*

AT: acceleration time, ET: ejection time, IVCT: isovolumic contraction time, IVRT: isovolumic relaxation time

Table 2.– Comparison of mean values of ACT, Adur, AVO, S in the pulmonary artery and vein in the first and control groups

	ACT, ms			A dur, ms			AVO, cm/sec			S, cm/sec		
	First group	Control group	p-value	First group	Control group	p-value	First group	Control group	p-value	First group	Control group	p-value
1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
CHD, n=11	157.4±23.6	157.6±19.7	p>0.05	75.2±3.8	74.0±16.2	p>0.05	15.2±3.4	12.2±3.2	p>0.05	29.3±3.5	20.7±3.2	p<0.05*
Early FGR, n=13	167.2±29.1	157.6±19.7	p>0.05	90.5±24.9	74.0±16.2	p>0.05	7.8±1.4	12.2±3.2	p>0.05	21.7±2.7	20.7±3.2	p>0.05
Fetal edema is not immune, n=3	210.6±7.1	157.6±19.7	p<0.05*	96.6±0.4	74.0±16.2	p>0.05	7.3±0.9	12.2±3.2	p>0.05	25.0±0.6	20.7±3.2	p>0.05
Fetal hemolytic anemia, n=7	207.3±11.1	157.6±19.7	p<0.05*	118.8±17.5	74.0±16.2	p<0.05*	8.3±1.2	12.2±3.2	p>0.05	27.0±3.2	20.7±3.2	p>0.05

1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
FFTS donor, n=5	176.0±4.0	157.6±19.7	p>0.05	106.4±10.4	74.0±16.2	p<0.05*	8.6±1.4	12.2±3.2	p>0.05	23.6±2.2	20.7±3.2	p>0.05
FFTS recipient, n=5	207.8±8.1	157.6±19.7	p<0.05*	98.4±3.6	74.0±16.2	p>0.05	9.0±1.2	12.2±3.2	p>0.05	28.8±3.7	20.7±3.2	p<0.05*
Diabetic macrosomia, n=7	157.4±23.6	157.6±19.7	p>0.05	75.2±3.8	74.0±16.2	p>0.05	15.2±3.4	12.2±3.2	p>0.05	29.3±3.5	20.7±3.2	p<0.05*

p < 0.05 there are statistically significant differences*

ACT: atrial contraction time, A dur: duration of A wave, AVO: atrioventricular opening, S: systole

Table 3.– Comparison of the average ratios of IVRT/ET, AST/IVRT, AVO/S, AT/ET in the pulmonary artery and vein in the first and control groups

	IVRT/ET			**ACT/IVRT**			**AVO/S**			**AT/ET**		
	First group	**Control group**	**p-value**	**First group**	**Control group**	**p-value**	**First group**	**Control group**	**p-value**	**First group**	**Control group**	**p-value**
CHD, n=11	0.49±0.02	0.48±0.08	p>0.05	0.90±0.10	0.90±0.26	p>0.05	0.51±0.08	0.58±0.08	p>0.05	0.19±0.03	0.17±0.02	p>0.05
Early FGR, n=13	0.45±0.14	0.48±0.08	p>0.05	1.12±0.26	0.90±0.26	p>0.05	0.35±0.05	0.58±0.08	p<0.05*	0.20±0.05	0.17±0.02	p>0.05
Fetal edema is not immune, n=3	0.54±0.09	0.48±0.08	p>0.05	0.95±0.07	0.90±0.26	p>0.05	0.29±0.04	0.58±0.08	p<0.05*	0.18±0.04	0.17±0.02	p>0.05
Fetal hemolytic anemia, n=7	0.53±0.08	0.48±0.08	p>0.05	1.11±0.21	0.90±0.26	p>0.05	0.31±0.03	0.58±0.08	p<0.05*	0.22±0.09	0.17±0.02	p<0.05*
FFTS donor, n=5	0.49±0.02	0.48±0.08	p>0.05	1.23±0.19	0.90±0.26	p>0.05	0.36±0.05	0.58±0.08	p<0.05*	0.19±0.01	0.17±0.02	p>0.05
FFTS recipient, n=5	0.31±0.01	0.48±0.08	p<0.05*	1.72±0.10	0.90±0.26	p<0.05*	0.31±0.02	0.58±0.08	p<0.05*	0.17±0.01	0.17±0.02	p>0.05
Diabetic macrosomia, n=7	0.36±0.01	0.48±0.08	p>0.05	1.35±0.11	0.90±0.26	p<0.05*	0.32±0.02	0.58±0.08	p<0.05*	0.15±0.01	0.17±0.02	p>0.05

p<0.05 there are statistically significant differences*

lar valve in the venous spectrum (or the closing of the semilunar valve in the arterial spectrum) to point D, the start point of atrial contraction. When calculating the isovolumetric contraction, the ejection onset point was considered as the end point, and certain difficulties arose when determining the IVCT reference point, since it was difficult to accurately determine the time of closing of the atrioventricular valve on the spectrum. As you can see in (Fig. 1), A point is clearly visible on the spectrum, the end of atrial contraction, then the atrium relaxes and an antegrade blood flow begins in the pulmonary vein, which accelerates to the beginning of systole. Hypothetically, we took half the time from point A "end of atrial systole" to the point of the start of ejection time in the arterial spectrum (or point S in the venous spectrum).

In the first group, the MPI index averaged 0.65 ± ± 0.11 with a 95% CI 0.43–0.87, in the second group it was 0.64 ± 0.06 (95% CI 0.50–0, 78) and in the control group 0.66 ± 0.10 (95% CI 0.45––0.87), when comparing both groups with the control p > 0.05, no statistically significant difference was found. This is apparently due to the heterogeneity of the group with cardiomegaly, which included fetuses with growth retardation, diabetic fetopathy, immune and non-immune edema, etc. To clarify the data, studies in a homogeneous group are required. We have demonstrated that IVCT, IVRT and ET measurements can easily be obtained using the spectrum in the pulmonary artery and vein.

Thus, the data of our study showed that diastolic dysfunction can be judged by the shortening of the time of isovolumetric myocardial contraction, the lengthening of the atrial time, and the decrease in the AVO/S ratio. Systolic dysfunction of the heart was characterized by a pronounced dicrotic notch in the spectrum, a decrease in the wave after the closure of the semilunar valve/or retrograde blood flow in the ventricular diastole.

Our results allow us to state that the study of the Doppler spectrum of blood flow in the pulmonary artery and vein (in a single control volume) makes it possible to assess the presence and nature of cardiac dysfunction in fetuses with cardiomegaly.

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