VIA MEDICA

EDITORIAL

Ginekologia Polska
2024, vol. 95, no. 10, 759–762

ISSN 0017–0011, e-ISSN 2543–6767

DOI: 10.5603/gpl.103280

Fetal therapy guidelines — fetal echocardiography is of special value. Statement of the Polish Prenatal Cardiology Society

Maria Respondek-Liberska[1, 2], Maciej Slodki[1, 3], Oskar Sylwestrzak[1, 4], Iwona Strzelecka[1, 2]

[1]*Department of Prenatal Cardiology, Polish Mother's Memorial Hospital Research Institute in Lodz, Poland*
[2]*Department for Fetal Malformation Diagnosis and Prevention, Medical University of Lodz, Poland*
[3]*Faculty of Health Sciences, The Mazovian University, Plock, Poland*
[4]*Department of Obstetrics and Gynecology, Polish Mother's Memorial Hospital Research Institute in Lodz, Poland*

We have read with a great interest "Fetal therapy guidelines of the Polish Society of Gynecologists and Obstetricians — Fetal Therapy Section" by Kosinski et al. [1]. We would like to congratulate the Authors their effort and excellent results. This is the Polish guideline of fetal therapy presented during magnificent conference "Fetal therapy" in Warsaw 2024. The Authors of the guidelines are Polish experts of fetal therapy, perinatologists working in the tertiary centers for fetal medicine, with significant academic achievements and prominent medical experience. As the members of the Polish Prenatal Cardiology Society we would like to thank for including fetal echocardiography into the guidelines as important tool for assessing fetal heart anatomy and cardiovascular efficacy. We could read inter alia in the guidelines that:

— fetal echocardiography is of key importance in case of non-immune hydrops fetalis,
— fetal echocardiography is advised to evaluate fetal circulatory efficiency in case of sacrococcygeal teratoma.

Nevertheless, we would like to highlight that fetal echocardiography could be helpful in many more fetal pathologies [2, 3].

ABNORMAL AMNIOTIC FLUID VOLUME

Abnormal amniotic fluid volume is a quite common pregnancy complication. Polyhydramnios could be idiopathic or caused by congenital fetal defects, maternal diseases or genetic abnormalities. Cardiovascular malformations could also be significantly associated with polyhydramnios [4]. From fetal defects the Authors describe for example vascular rings that could obstruct fetal "airways". Vascular rings diagnosis is still challenging, but fetal echocardiography remains gold standard of diagnosis and monitoring this anomaly. The other, most common group of congenital defects causing polyhydramnion are gastrointestinal tract anomalies [5]. Still gastrointestinal diseases may influence fetal cardiovascular system, for example by increased pressure in abdominal cavity on inferior vena cava, that could also be assessed by fetal echocardiography. Polyhydramnion may be present in addition to cardiac problems (for instance at the imminent cardiovascular insufficiency), so fetal heart evaluation is essential for proper interpretation.

Oligohydramnios could have also been caused by fetal defects. The most common are urinary tract defects. As presented by many studies LUTO influence fetal cardiovascular system and in many cases the prognosis depends on cardiovascular efficiency [6].

What is more preterm prelabor rupture of membranes (pPROM) changes fetal environment enormously and many fetal cardiovascular disturbances have been described [7]. Concerning fetal growth restriction (FGR), those fetuses presents many cardiovascular abnormalities like: abnormal GSI, myocardial hypertrophy, abnormal mitral and tricuspid flow, etc. [8]. Fetal echocardiography could be used to establish fetal risk, answer whether pregnancy could be continued and help to predict prognosis.

NON-IMMUNE FETAL HYDROPS FETALIS

NIHF is associated with highly increased fetal and neonatal mortality [9]. As the Authors presented the most common underlying causes include fetal cardiovascular defects. Once more, the gold standard of diagnosis and monitoring of fetal cardiovascular insufficiency is fetal echocardiography. NIHF is a result of severe fetal heart failure. It may occur as a consequence of primary fetal heart defect [10], as abnormal

Corresponding author:
Oskar Sylwestrzak
Department of Prenatal Cardiology, Polish Mother's Memorial Hospital Research Institute in Lodz, Lodz, Poland
e-mail: sylwestrzakoskarpatryk@gmail.com

Received: 28.10.2024 Accepted: 29.10.2024

fetal heart anatomy result in somehow defective intracardiac bloodflows. Abnormal preload/afterload could worsen fetal cardiovascular efficiency with pregnancy advancing *e.g.* concomitant pulmonary and aortic severe stenosis or Ebstein anomaly [11]. Also many extracardiac conditions induce impaired myocardial function and may end up with NIHF [12]. In case of NIHF only fetal echocardiography enables to precisely predict prenatal (as NIHF may be complicated by fetal demise) or postnatal prognosis, plan future monitoring and schedule postnatal management. So in NIHF fetal echocardiography is obligatory and requires emergency examination regardless of gestational age.

FETAL HEMOLYTIC DISEASE

Even though immunoprophylaxis has decreased fetal alloimmunization and limited fetal hemolytic disease, it still remains serious obstetrical complication. Measurement of fetal peak systolic velocity of blood flow through middle cerebral artery is widely used to diagnose fetal hemolytic disease. It is easy to obtain, nearly in every pregnant women. However not only cerebral blood flows are abnormal in fetuses with anemia. As a response to anemia fetal increased cardiac output and vasodilatation has been proved. Also fetal atria response by increased contractility, which is presented by increased E wave velocity through mitral valve [13]. Due to increased need of tissue oxygenation, fetal myocardium overgrowths. By fetal echocardiography one may quantify fetal myocardial function in anemia more accurately. What is more fetal echocardiography has been used to monitor fetal blood transfusion [14]. The problem is even more complex if additional cardiac functional abnormalities occur [15].

FETAL PULMONARY MALFORMATIONS

Fetal lungs are directly connected with heart by pulmonary artery and pulmonary veins. These two organs are also located in the same cavity — the chest. So every enlargement or decrease in lung volume influence fetal heart (its size, location and axis) [16]. Fetal echocardiography could be carried out to exclude cardiac defects, assess the size of the heart and muscle contractility, diagnose possible atrioventricular regurgitation, and evaluate the blood flow using Doppler techniques. Pulmonary malformations can result in significant cardiac compression that alters hemodynamics and may result in hydrops on the basis of elevated central venous pressure. Assessment of fetal cardiovascular system could be also a clue in difficult fetal pulmonary diagnoses [17].

CONGENITAL DIAPHRAGMATIC HERNIA

Fetal echocardiography has been used for many years to predict postnatal outcome in case of congenital diaphragmatic hernia. Cardiac dysfunction is quite common consequence of congenital diaphragmatic hernia. Fetal cardiovascular insufficiency is a determinant of disease severity. Increased afterload and impairment left ventricular function have been observed [18]. Usually fetal compensatory mechanisms in response to diminished antegrade cerebral blood flow enables to monitor pregnancy and plan prenatal or postnatal management [19]. If there is no additional congenital heart defect, fetal prognosis is better, but still left heart structures may appear small, even if it usually normalizes after birth [20]. Influence of left sided fetal heart structures development have been studied for prediction of postnatal follow-up, with uncertain results [21]. That is why future more profound studies are needed. Certainly fetuses with congenital diaphragmatic hernia can suffer from both right and left ventricular dysfunction and the best tool to assess this dysfunction is fetal echocardiography. Especially nowadays, when neonatal pulmonary hypertension could be suspected [22–24]. Additional advantage of fetal echocardiography is a possibility to confirm fetal well-being after fetal endoscopic tracheal occlusion [25].

Fetal echocardiography is also obligatory in diaphragmatic hernia as the presence of additional structural heart defect changes the short-term and long-term prognosis in such pregnancy.

VEIN OF GALEN MALFORMATION

Prenatal treatment of a vein of Galen malformationhas been started as a clinical trial [26]. We look forward with hope, that interventional attempt could probably help in the future outcome of these fetuses. Hemodynamic profile in fetuses diagnosed with vein of Galen malformation has been studied and fetuses presented increased cardiothoracic ratio due to right ventricular dilatation, aortic isthmus retrograde flow starting in mid systole and throughout diastole, increased left and right ventricular and abnormal net pulmonary flow [27]. Non-survivors presents right ventricular dysfunction with tricuspid regurgitation [28]. The worse prognosis involve fetuses with hydrops fetalis. For this reasonfetal echocardiography is mandatory for the prediction of prognosis [29]. Especially because aggressive medical treatment of cardiac failure is needed if patient presents cardiac failure prenatally. Prognosis for newborns with vein of Galen malformation diagnosed prenatally is highlybased on fetal cardiovascular function and could potentially be better by prenatal treatment [30].

UROGENITAL MALFORMATIONS

The obstruction of fetal urinary tract may be located at the level of ureter, bladder or urethra. That cause dilatation of the upper part of the urinary tract above the obstruction. Data from Polish Mother's Memorial Hospital Research Institute in Lodz from 20 years ago showed that over 54%

of all fetuses with posterior urethral valve demonstrated functional abnormalities in circulatory system. Mortality of the fetuses with abnormalities in circulatory system was significantly higher than mortality of fetuses without circulatory abnormalities [31]. What is more lower urinary tract obstruction was associated with abnormal right ventricular filling and the correlation was found with β-2-microglobulin, α-1-microglobulin and potassium [32]. In severe cases with giant bladder fetuses presented higher incidence of cardiomegaly, myocardial hypertrophy and pericardial effusion [33]. In addition also left ventricular filling appeared to dysfunctional. That could be connected with redistributed cardiac output, what could contribute to the development of left-heart hypoplasia [34]. So it seems that urogenital malformations influence fetal cardiovascular system in many pathways, but finally they could worsen fetal cardiovascular efficiency and promote abnormal heart development.

FETAL OVARIAN CYST

Female fetuses with ovarian cysts usually have normal heart anatomy, but in about 14% of them there is congenital heart defect. Even if heart anatomy is normal there is a high prevalence of functional cardiovascular anomalies. Quite common is myocardial hypertrophy. Some of them presented monophasic inflow pattern of tricuspid valve, holosystolic tricuspid regurgitation and pericardial effusion. To reduce prenatal complications aspiration of the cyst could be performed. After prenatal aspiration of the cyst, the functional abnormalities in fetal echocardiography could recede, what could potentially present positive result of fetal therapy. Fetuses with ovarian cyst and functional anomalies more often required surgical procedures after the birth, whereas normal heart study was more often connected with spontaneous regression of ovarian cyst [35, 36].

TWIN-TO-TWIN TRANSFUSION SYNDROME

Twin-to-twin transfusion syndrome (TTTS) occurs in about 10% of monochorionic twin pregnancies. It is a severe complication that develops due to an intrauterine imbalance in intertwin blood exchange. Recipients present decreased ventricular shortening fraction, increased cardiac output, thickened myocardium, monophasic ventricular filling, shortened ventricular filling and prolonged isovolumetric relaxation time [37]. There is a high prevalence of tricuspid regurgitation and in severe cases mitral regurgitation [38]. Donors, on the other side, seem to usually have a normal cardiac function, with 5–10% present with abnormal Doppler waveforms in the ductus venosus, and 3% with tricuspid regurgitation or umbilical vein pulsations [37]. Fetal echocardiography has been also used to predict outcome in TTTS. In future TTTS recipients RV and LV Tei index were significantly higher, but in donors RV Tei index was lower [39]. In case of developing TTTS fetal echocardiography enables to recognize this complication before signs described in Quintero scale. In the case of a surgical procedure in TTTS Polish Prenatal Cardiology Society recommend control fetalechocardiography examinations a day before, post-procedure and within thenext 24 hours, on the 3rd and 7th day (in a hospital setting). Then at 14-day intervals (in an outpatient setting) [40].

CONCLUSIONS

Kosinski et al. [1] did a great effort to summary obstetricians' knowledge and experience what was presented in the form of excellent guidelines. The experts are perfectly acquainted with fetal echocardiography possibilities and use it in their clinical practice. We fully understand that Fetal Therapy Guidelines could not describe mainly utility of fetal echocardiography because the subject is too comprehensive. Polish Prenatal Cardiology Society Board in this commentary wanted to remind that fetal echocardiography is even more useful in the most difficult fetal cases that are usually managed by team of specialists. Use of fetal echocardiography could help to understand fetal physiology, predict outcome and monitor fetuses before and after prenatal invasive therapy.

It is our hope that in near future every referral obstetrical ward would have a fetal cardiologist on-side to discuss the most difficult problems in perinatology and to choose the best therapy option for our patients — fetuses [41–45].

Article information and declarations

Conflict of interest

All authors declare no conflict of interest.

REFERENCES

1. Kosinski P, Borowski D, Brawura-Biskupski-Samaha R, et al. Fetal therapy guidelines of the Polish Society of Gynecologists and Obstetricians — Fetal Therapy Section. Ginekol Pol. 2024; 95(4): 285–315, doi: 10.5603/gpl.100108, indexed in Pubmed: 38632880.
2. Słodki M, Respondek-Liberska M. Fetal echocardiography: one of the most important tools in fetal diagnosis and assessing wellbeing. J Clin Ultrasound. 2022; 50(5): 636–638, doi: 10.1002/jcu.23216, indexed in Pubmed: 35674056.
3. Sylwestrzak O, Strzelecka I, Słodki M, et al. Fetal echocardiography is not only used to detect congenital heart disease but also to monitor fetuses, especially those with different pathologies. Kardiol Pol. 2022; 80(9): 966–967, doi: 10.33963/KP.a2022.0159, indexed in Pubmed: 35758319.
4. Nowakowska A, Sylwestrzak O, Strzelecka I, et al. Prenatal echocardiography in Trisomy 18 — the key to diagnosis and further management in the second half of pregnancy. Ginekol Pol. 2023; 94(5): 366–373, doi: 10.5603/GP.a2023.0035, indexed in Pubmed: 37070966.
5. Kornacki J, Adamczyk M, Wirstlein P, et al. Polyhydramnios — frequency of congenital anomalies in relation to the value of the amniotic fluid index. Ginekol Pol. 2017; 88(8): 442–445, doi: 10.5603/GP.a2017.0081, indexed in Pubmed: 28930371.
6. Graupner O, Enzensberger C, Götte M, et al. Myocardial function in fetuses with lower urinary tract obstruction: Is there a cardiac remodeling effect due to renal damage? Prenat Diagn. 2019; 39(7): 495–504, doi: 10.1002/pd.5453, indexed in Pubmed: 30957256.

7. Pasieczna M, Kuran-Ohde J, Grzyb A, et al. Value of fetal echocardiographic examination in pregnancies complicated by preterm premature rupture of membranes. J Perinat Med. 2024; 52(5): 538–545, doi: 10.1515/jpm-2023-0448, indexed in Pubmed: 38639637.
8. Oluklu D, Menekse Beser D, Uyan Hendem D, et al. Assessment of fetal cardiac morphology and functional changes in early-onset and late-onset fetal growth restriction. Int J Gynaecol Obstet. 2023; 161(1): 241–249, doi: 10.1002/ijgo.14602, indexed in Pubmed: 36453150.
9. Sileo FG, Kulkarni A, Branescu I, et al. Non-immune fetal hydrops: etiology and outcome according to gestational age at diagnosis. Ultrasound Obstet Gynecol. 2020; 56(3): 416–421, doi: 10.1002/uog.22019, indexed in Pubmed: 32196790.
10. Ojala TH, Hornberger LK. Fetal heart failure. Front Biosci (Schol Ed). 2010; 2(3): 891–906, doi: 10.2741/s109, indexed in Pubmed: 20515832.
11. Gottschalk I, Gottschalk L, Stressig R, et al. Ebstein's anomaly of the tricuspid valve in the fetus — a multicenter experience. Ultraschall Med. 2017; 38(4): 427–436, doi: 10.1055/s-0042-107151, indexed in Pubmed: 27248797.
12. Kaczmarek P, Jaczewski B, Oszukowski P, et al. [Non-immune hydrops fetalis--prognostic factors based on fetal echo (analysis in 230 cases)]. Ginekol Pol. 2003; 74(10): 1112–1117, indexed in Pubmed: 14669404.
13. Bigras JL, Suda K, Dahdah NS, et al. Cardiovascular evaluation of fetal anemia due to alloimmunization. Fetal Diagn Ther. 2008; 24(3): 197–202, doi: 10.1159/000151338, indexed in Pubmed: 18753757.
14. Michel M, Schmitz R, Kiesel L, et al. Fetal myocardial peak systolic strain before and after intrauterine red blood cell transfusion — a tissue Doppler imagingstudy. J Perinat Med. 2012; 40(5): 545–550, doi: 10.1515/jpm-2011-0272, indexed in Pubmed: 22945275.
15. Luewan S, Tongprasert F, Srisupundit K, et al. The accelerated right ventricular failure in fetal anemia in the presence of restrictive foramen ovale. Diagnostics (Basel). 2022; 12(7), doi: 10.3390/diagnostics12071646, indexed in Pubmed: 35885551.
16. Reyna-Villasmil E, Briceño-Sanabria L, Briceño-Sanabria JC, et al. Prenatal ultrasound diagnosis of unilateral pulmonary agenesis. J Med Ultrasound. 2024; 32(3): 259–261, doi: 10.4103/jmu.jmu_24_23, indexed in Pubmed: 39310869.
17. Sliwka A, Sztyler-Krakowska M, Bielasik K, et al. Prenatal diagnosis of scimitar syndrome. Ginekol Pol. 2024 [Epub ahead of print], doi: 10.5603/gpl.99477, indexed in Pubmed: 39140350.
18. Sokołowski Ł, Pałgan M, Strzelecka I, et al. Prenatal diagnosis of right-sided congenital diaphragmatic hernia. Prenat Cardiol. 2023; 2023(1), doi: 10.5114/pcard.2023.137869.
19. Kosiv KA, Moon-Grady A, Hogan W, et al. Fetal cerebrovascular impedance is reduced in left congenital diaphragmatic hernia. Ultrasound Obstet Gynecol. 2021; 57(3): 386–391, doi: 10.1002/uog.21992, indexed in Pubmed: 32068925.
20. Moon-Grady AJ, Byrne FA, Lusk LA, et al. Expected small left heart size in the presence of congenital diaphragmatic hernia: fetal values and Z-scores for infants confirmed to have no heart disease postnatally. Front Pediatr. 2022; 10: 1083370, doi: 10.3389/fped.2022.1083370, indexed in Pubmed: 36561485.
21. Krekora M, Sokołowski Ł, Murlewska J, et al. Small prenatal diameter of the ascending aorta is associated with increased mortality risk in neonates with congenital diaphragmatic hernia. Arch Med Sci. 2023; 19(4): 1022–1027, doi: 10.5114/aoms/147768, indexed in Pubmed: 37560725.
22. Murlewska J, Sylwestrzak O, Respondek-Liberska M. Unfavorable postnatal outcome with significant dilation of the fetal main pulmonary artery near term. Birth Defects Res. 2021; 113(1): 55–62, doi: 10.1002/bdr2.1828, indexed in Pubmed: 33094922.
23. Sylwestrzak O, Murlewska J, Sokołowski Ł, et al. Increased maternal phenylalanine concentration may influence not only fetal heart structural development but also cardiovascular function and pulmonary tissue development in humans — a case report. Prenat Cardiol. 2023; 2023(1), doi: 10.5114/pcard.2023.135555.
24. Nowakowska A, Krekora M, Talar T, et al. Prenatal diagnosis of hypoplastic left heart syndrome with PAPVC and FO restriction and pulmonary hypertension – coexisting factors suggesting poor prognosis due to pulmonary hypertension with histopathological confirmation. Prenat Cardiol. 2022; 1: 25–30, doi: 10.5114/pcard.2023.126182.
25. Degenhardt J, Enzensberger C, Tenzer A, et al. Myocardial function pre- and post-fetal endoscopic tracheal occlusion (FETO) in fetuses with left-sided moderate to severe congenital diaphragmatic hernia. Ultraschall Med. 2017; 38(1): 65–70, doi: 10.1055/s-0041-108501, indexed in Pubmed: 27626241.
26. See AP, Wilkins-Haug LE, Benson CB, et al. Percutaneous transuterine fetal cerebral embolisation to treat vein of Galen malformations at risk of urgent neonatal decompensation: study protocol for a clinical trial of safety and feasibility. BMJ Open. 2022; 12(5): e058147, doi: 10.1136/bmjopen-2021-058147, indexed in Pubmed: 35613814.
27. Mendez A, Codsi E, Gonzalez Barlatay F, et al. Pulmonary hypertension associated with vein of Galen malformation. Fetal cardiac hemodynamic findings and physiological considerations. J Perinatol. 2022; 42(1): 143–148, doi: 10.1038/s41372-021-01297-y, indexed in Pubmed: 35022516.
28. Jhaveri S, Berenstein A, Srivastava S, et al. High output cardiovascular physiology and outcomes in fetal diagnosis of vein of galen malformation. Pediatr Cardiol. 2021; 42(6): 1416–1424, doi: 10.1007/s00246-021-02627-9, indexed in Pubmed: 33963894.
29. Turkyilmaz G, Arisoy R, Turkyilmaz S, et al. The outcome of the vein of Galen aneurysmal malformation cases diagnosed prenatally. J Obstet Gynaecol. 2022; 42(5): 1137–1141, doi: 10.1080/01443615.2021.2012439, indexed in Pubmed: 35020567.
30. Respondek-Liberska M, Słodki M. Prognosis for newborns with vein of Galen malformation diagnosed prenatally based on a new scale. Prenat Cardiol. 2019; 2019(1): 37–41, doi: 10.5114/pcard.2019.92671.
31. Grzesiak M, Respondek-Liberska M, Szaflik K, et al. [Fetal posteriorurethralvalve (PUV) in database of Department of the Diagnosis and Prophylaxis of Congenital Malformation Institute Polish "Mother's Memorial Hospital" in 1994–2002 and echocardiographic results]. Ginekol Pol. 2003; 74(10): 1088–1092, indexed in Pubmed: 14669400.
32. Graupner O, Enzensberger C, Götte M, et al. Myocardial function in fetuses with lower urinary tract obstruction: Is there a cardiac remodeling effect due to renal damage? Prenat Diagn. 2019; 39(7): 495–504, doi: 10.1002/pd.5453, indexed in Pubmed: 30957256.
33. Rychik J, McCann M, Tian Z, et al. Fetal cardiovascular effects of lower urinary tract obstruction with giant bladder. Ultrasound Obstet Gynecol. 2010; 36(6): 682–686, doi: 10.1002/uog.7664, indexed in Pubmed: 20503245.
34. Cohen J, Levasseur S, Simpson L, et al. Fetal cardiac findings and hemodynamic changes associated with severe lower urinary tract obstruction in utero. Ultrasound Obstet Gynecol. 2019; 54(6): 780–785, doi: 10.1002/uog.20271, indexed in Pubmed: 30908816.
35. Słodki M, Janiak K, Szaflik K, et al. [Fetal echocardiography in fetal ovarian cysts]. Ginekol Pol. 2008; 79(5): 347–351, indexed in Pubmed: 18624110.
36. Słodki M, Janiak K, Szaflik K, et al. Fetal echocardiography before and after prenatal aspiration of a fetal ovarian cyst. Ginekol Pol. 2009; 80(8): 629–631, indexed in Pubmed: 19824464.
37. Van Mieghem T, Lewi L, Gucciardo L, et al. The fetal heart in twin-to-twin transfusion syndrome. Int J Pediatr. 2010; 2010, doi: 10.1155/2010/379792, indexed in Pubmed: 20811613.
38. Rychik J, Tian Z, Bebbington M, et al. The twin-twin transfusion syndrome: spectrum of cardiovascular abnormality and development of a cardiovascular score to assess severity of disease. Am J Obstet Gynecol. 2007; 197(4): 392.e1–392.e8, doi: 10.1016/j.ajog.2007.06.055, indexed in Pubmed: 17904973.
39. Zanardini C, Prefumo F, Fichera A, et al. Fetal cardiac parameters for prediction of twin-to-twin transfusion syndrome. Ultrasound Obstet Gynecol. 2014; 44(4): 434–440, doi: 10.1002/uog.13442, indexed in Pubmed: 24919586.
40. Leszczyńska K, Preis K, Respondek-Liberska M, et al. Recommendations for fetal echocardiography in twin pregnancy in 2016. Prenat Cardiol. 2016; 6(1): 6–15, doi: 10.1515/pcard-2016-0001.
41. Respondek-Liberska M. Diagnostyka prenatalna USG/ECHO. Wady wymagające interwencji chirurgicznej. PZWL, Warszawa 2016.
42. Respondek-Liberska M. Diagnostyka prenatalna USG/ECHO. Zmiany czynnościowe w układzie krążenia płodu. PZWL, Warszawa 2019.
43. Respondek-Liberska M. Wady serca płodu. Diagnostyka i postępowanie. PZWL, Warszawa 2022.
44. Słodki M. Opracowanie modelu opieki nad ciężarną z wrodzoną wadą serca u płodu na podstawie nowego prenatalnego podziału wad serca. Rozprawa habilitacyjna. Wydawnictwo PWSZ w Płocku, Łódź 2012.
45. Słodki M, Copel JA, Rizzo G, et al. Endorsed by The International Prenatal Cardiology Collaboration Group. Fetal cardiology: is it time to establish a separate independent medicine subspeciality? Pediatr Cardiol. 2022; 43(7): 1676–1677, doi: 10.1007/s00246-022-02936-7, indexed in Pubmed: 35606573.