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MODERN DIAGNOSIS OF TETRALOGY OF FALLOT

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Abstract: *Instrumental diagnostics of tetralogy of Fallot includes PCG, electrocardiography, ultrasound of the heart, chest X-ray, catheterization of the heart chambers and ventriculography. Surgical treatment of tetralogy of Fallot can be palliative (placing intersystem anastomoses) and radical (complete surgical correction of the defect). Tetralogy of Fallot is a combined congenital anomaly of the heart, characterized by stenosis of the outflow tract of the right ventricle, a defect of the interventricular septum, dextroposition of the aorta and hypertrophy of the right ventricular myocardium. Clinically, tetralogy of Fallot is manifested by early cyanosis, developmental delay, shortness of breath and dyspnea-cyanotic attacks, dizziness and fainting.*

Key words: *Causes of tetralogy of Fallot, Classification of tetralogy of Fallot, Features of hemodynamics in tetralogy of Fallot, Symptoms of tetralogy of Fallot, Diagnosis of tetralogy of Fallot, Treatment of tetralogy of Fallot, Tetralogy of Fallot prognosis.*

Introduction: Tetralogy of Fallot is a complex congenital heart defect of the "blue" type, the morphological basis of which is four signs: obstruction of the right ventricular outflow tract, a large VSD, right ventricular hypertrophy and displacement of the aorta. The most structurally related tetralogy of Fallot is the triad of Fallot (pulmonary artery stenosis, atrial septal defect, and right ventricular hypertrophy) and the pentad of Fallot (tetralogy of Fallot and ASD). Tetralogy of Fallot may be associated with other cardiac and vascular anomalies: right-sided aortic arch, coronary artery anomalies, pulmonary artery stenosis, patent ductus arteriosus, complete patent atrioventricular canal, accessory left superior vena cava, and partially anomalous pulmonary vein. In cardiology, tetralogy of Fallot occurs in 7-10% of all congenital heart defects and accounts for half of all cyanotic-type defects. A detailed

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anatomical description of the defect as an independent nosological form was first given in 1888 by the French pathologist E.LA Fallot, who later gave it his name.

Research methods and materials: Tetralogy of Fallot is formed as a result of a violation in the process of cardiogenesis at 2-8 weeks. embryonic development. The development of the defect can be caused by infectious diseases of a pregnant woman in the early stages of pregnancy (measles , rubella , rubella); taking medications (sleeping pills, sedatives, hormonal drugs, etc.), drugs or alcohol; exposure to harmful production factors. The influence of heredity in the formation of congenital heart disease can be traced. Tetralogy of Fallot is most often found in children with Cornelia de Lange syndrome (Amsterdam dwarfism), which includes mental retardation and numerous developmental anomalies (clown face, choanal atresia, ear deformity, cleft palate, strabismus, myopia, astigmatism, optic nerve and hypertension. deformity, syndactyly of the feet, reduced number of fingers, malformations of internal organs, etc.).

The cause of tetralogy of Fallot is a malrotation of the conus arteriosus (counterclockwise), which causes the aortic valve to move to the right relative to the pulmonary valve. In this case, the aorta is located above the interventricular septum ("riding aorta"). Malposition of the aorta leads to displacement of the pulmonary trunk, which becomes slightly longer and narrower. The rotation of the conus arteriosus prevents its septum from connecting with the interventricular septum, which leads to the formation of a VSD and subsequent enlargement of the right ventricle. Given the nature of the right ventricular outflow tract obstruction, anatomical variants of tetralogy of Fallot are divided into four types: embryological, hypertrophic, tubular, and multicomponent. Tetralogy of Fallot type I is embryological. The obstruction is caused by anterior and leftward displacement of the conical septum and/or its low position. The zone of maximal stenosis is at the level of the muscular ring of demarcation. The fibrous ring of the pulmonary valve is practically unchanged or moderately hypoplastic. Tetralogy of Fallot type II is hypertrophic. The obstruction is based on the displacement of the conical septum forward and to the left and/or its low location, as well as pronounced hypertrophic

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changes in its proximal segment. The zone of maximum stenosis corresponds to the level of the opening of the outlet of the right ventricle and the muscular ring of demarcation. Tetralogy of Fallot type III is tubular. The obstruction is caused by an uneven division of the common arterial trunk, resulting in a severely hypoplastic, narrowed, and shortened pulmonary conus. In this type of tetralogy of Fallot, hypoplasia of the fibrous ring or valvular stenosis of the pulmonary trunk may occur. Tetralogy of Fallot type IV is multicomponent. The cause of obstruction is a significant elongation of the conical septum or a high degree of deviation of the septal-marginal trabecula of the moderator band. According to their hemodynamic characteristics, three clinical and anatomical forms of tetralogy of Fallot are distinguished: 1) with atresia of the pulmonary artery orifice; 2) cyanotic form with stenosis of the orifice of varying degrees; 3) cyanotic form.

Results: With moderate obstruction, the total peripheral resistance is higher than the resistance of the stenotic outflow tract, so a left-to-right shunt develops, which leads to the development of the cyanotic (pale) form of tetralogy of Fallot. At the same time, with the development of stenosis, first a cross-flow, and then a venoarterial (right-to-left) blood flow appears, which means that the defect changes from a “white” form to a “blue” form. Depending on the time of onset of cyanosis, five clinical forms and, accordingly, the same number of periods of manifestation of tetralogy of Fallot are distinguished: early cyanotic form (appearance of cyanosis in the first months or first year of life), classical (appearance of cyanosis in the second or third year of life), late-cyanotic (with cyanotic onset), cyanotic (appearance of cyanosis at 6-10 years of age) and cyanotic (pale) form. In severe forms of tetralogy of Fallot, cyanosis of the lips and skin appears at 3-4 months and is stable at 1 year of age. Cyanosis is aggravated by eating, crying, straining, emotional stress, and physical exertion. Any physical activity (walking, running, active play) is accompanied by increased shortness of breath, weakness, tachycardia, and dizziness. The usual position of patients with tetralogy of Fallot after exercise is lying down. Children with tetralogy of Fallot may lag behind in physical (stage II-III hypotrophy) and motor development; They suffer from frequent recurrent acute respiratory viral infections,

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chronic tonsillitis, sinusitis, and recurrent pneumonia. Adult patients with tetralogy of Fallot may develop pulmonary tuberculosis. The most severe manifestation of the clinical picture of tetralogy of Fallot is dyspnea-cyanotic attacks, which usually appear at the age of 2-5 years. The attack develops suddenly, the child is accompanied by anxiety, increasing cyanosis and shortness of breath, tachycardia, weakness and loss of consciousness. The development of apnea, hypoxic coma, and convulsions with the subsequent manifestation of hemiparesis are possible. Dyspnea-cyanotic attacks develop as a result of a sharp spasm of the infundibular part of the right ventricle, which leads to the outflow of the entire volume of venous blood through the defect in the interventricular septum into the aorta and increased hypoxia of the central nervous system.

Discussion: When objectively examining patients with tetralogy of Fallot, attention is paid to pallor or cyanosis of the skin, thickening of the phalanges of the fingers ("drumsticks" and "hourglasses"), a forced posture and adynamia; less often - deformation of the chest (heart tails). Percussion reveals a slight expansion of the borders of the heart in two directions. Typical auscultatory signs of tetralogy of Fallot include a coarse systolic murmur in the II-III intercostal space on the left side of the sternum, weakening of the II tone over the pulmonary artery, etc. A complete auscultatory picture of the defect is recorded using phonocardiography. Chest X-ray reveals moderate cardiomegaly, a typical slipper-shaped heart, and absent lung signs. The ECG pattern is characterized by a significant rightward shift of the EOS, hypertrophic changes in the right ventricular myocardium, and incomplete blockade of the right bundle branch of His. With the help of ultrasound of the heart, all anatomical components of tetralogy of Fallot are directly determined: the degree of pulmonary stenosis, the magnitude of aortic displacement, the size of the VSD, and the severity of right ventricular hypertrophy. Heart sounding allows you to determine the high pressure in the right ventricle, the saturation of arterial blood with oxygen, and the passage of the catheter from the right ventricle to the aorta. When conducting aortography and pulmonary arteriography, collateral blood flow, the presence of PDA and pulmonary artery pathology are determined. If necessary, left ventricular ,

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selective coronary angiography , MSCT and MRI of the heart are performed . Differential diagnosis of tetralogy of Fallot is made with transposition of the great vessels, bilateral origin of the aorta and pulmonary artery from the right ventricle, single-ventricular heart, and two chamber heart. All patients with tetralogy of Fallot undergo surgical treatment. In the development of dyspnea cyanotic attacks, drug therapy is indicated: inhalation of humidified oxygen, intravenous administration of reopoliglyukin, sodium bicarbonate, glucose and euphyllin. If drug therapy is ineffective, immediate aortopulmonary anastomosis is necessary. The surgical approach to tetralogy of Fallot depends on the severity of the defect, its anatomical and hemodynamic variant, and the age of the patient. Newborns and young children with severe tetralogy of Fallot require palliative surgery in the first stage, which reduces the risk of complications during subsequent radical correction of the defect. Types of palliative (bypass) operations for tetralogy of Fallot include: creation of a Blalock-Taussig subclavian-pulmonary anastomosis, intrapericardial anastomosis of the ascending aorta and the right pulmonary artery, creation of a central aortopulmonary anastomosis by creating an anastomosis between a synthetic or biological lung and the left pulmonary artery, etc. Open infundibuloplasty and balloon valvuloplasty operations are used to reduce hypoxemia.

Conclusion: Radical repair of tetralogy of Fallot involves performing VSD plastic surgery and eliminating the obstruction of the right ventricular outflow tract. It is usually performed between six months and three years of age. Specific complications of operations performed for tetralogy of Fallot may include anastomotic thrombosis, acute heart failure, pulmonary hypertension, right ventricular aneurysm, AV block, arrhythmia, and infective endocarditis. The natural course of the defect largely depends on the degree of pulmonary stenosis. A quarter of children with severe tetralogy of Fallot die in the first year of life, half of them in the neonatal period. The average life expectancy without surgery is 12 years, less than 5% of patients live to 40 years. The cause of death in patients with tetralogy of Fallot is most often cerebral thrombosis (ischemic stroke) or brain abscess . The long-term results of radical repair of tetralogy of Fallot are good: patients are able-bodied and

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socially active, and tolerate physical activity satisfactorily. However, the later the age at which radical surgery is performed, the worse the long-term results. All patients with tetralogy of Fallot should be monitored by a cardiologist and cardiac surgeon, and antibiotic prophylaxis for endocarditis should be given before dental or surgical procedures that are potentially at risk for developing bacteremia.

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