

***СТРУКТУРНО-ФУНКЦИОНАЛЬНЫЕ НАРУШЕНИЯ СЕРДЦА ЧЕЛОВЕКА  
ПРИ ТЕТРАДЕ ФАЛЛО***

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***STRUCTURAL AND FUNCTIONAL DISORDERS OF THE HEART OF PEOPLE  
WITH THE FALLOT'S TETRALOGY***

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Data, presented in modern literature, about some congenital defects was studied. This report presents some information about the Fallot's tetralogy, which is the most common congenital defect of the heart. This defect is 50-70% of the total number of clinically defined congenital defects of the heart.

Permanent anatomical features of the tetralogy of Fallot are: obstruction of the outflow tract of the right ventricle (RV) and/or hypoplasia of pulmonary artery; large ventricular septal defect approximately equal to the diameter of aortal opening; right-sided aortic arch; hypertrophy of the myocardium of the right ventricle.

Subvalvular fibromuscular stenosis of the output of the RV may have several options: low - on the border of trabecular part and the output of the RV; high- in the output of the RV; diffusely hypoplastic output of the RV, and is often combined with valve stenosis, hypoplasia of the pulmonary ring (60%), as well as the trunk and branches (70%) of PA. Thus in the output section of the right ventricle between the valvular and subvalvular stenosis forms an extension - "the third ventricle."

As a result of balanced pressure in both ventricles during systole blood flows from the right ventricle through a ventricular septal defect with a greater extent into the ascending aorta and less - in pulmonary artery.

Therefore, the minute volume of the systemic circulation is increased, and the pulmonary - significantly reduced – hypovolemia. Reset of the venous blood into the aorta and reduced pulmonary blood flow cause the development of hypoxemia.

In response to chronic hypoxemia develop compensatory mechanisms aimed at reducing it (develop systemic-pulmonary collaterals, polycythemia, slowing blood flow to tissues and increased tortuosity of microvessels for better diffusion of oxygen). After some time in patients, changes in

the structure of the pulmonary circulation (peripheral vasoconstriction due to intimal hyperplasia, the emergence of "multibarrelled " vessels, arteriovenous anastomoses as a reaction to chronic hypoxemia) occur. By 10 to 15 years of age there is a tendency to thrombosis of pulmonary vessels.

With moderate pulmonary stenosis occurs left-to-right reset of blood that clinically manifests as pale form of the tetralogy of Fallot. With age stenosis progresses in Diastolic phase of the defect.

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