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ARTERIAL HYPOXIA AS A FACTOR AFFECTING THE COURSE OF CONGENITAL HEART DEFECTS IN CHILDREN

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Abstract: The most common forms of congenital heart defects (CHDs) in children include ventricular septal defect (VSD), atrial septal defect (ASD), and Tetralogy of Fallot. The natural course of these defects is often complicated by the development of severe complications caused by arterial hypoxia. The high mortality rate associated with septal heart defects in children is primarily due to progressive congestive heart failure and pulmonary hypertension. In patients with cyanotic CHDs, the leading pathogenetic factor is diffuse hypoxemia. Insufficient arterial blood oxygenation exacerbates the clinical manifestations of the defect and contributes to the development of cardiac arrhythmias, which may be regarded as an additional adverse prognostic factor in congenital heart disease.
Keywords: congenital heart defects, arterial hypoxemia, VSD, ASD, Tetralogy of Fallot, ECG, cardiac arrhythmias.

Relevance: Congenital heart defects (CHDs) represent the most prevalent group of developmental anomalies in children and remain a leading cause of disability and mortality in the pediatric population [3,4,9]. Moreover, a further increase in the incidence of CHDs is projected. This trend may be partly attributed to advancements in diagnostic techniques, including improved training of ultrasound specialists and enhanced imaging technologies [6,7,10].

The prognosis of children with congenital heart defects (CHDs) depends on the type of defect, the degree of arterial hypoxia, the timeliness of diagnosis, and the presence of comorbid conditions [3,8].

The most prevalent defects belong to the so-called “big six,” including ventricular septal defect (VSD), atrial septal defect (ASD), and Tetralogy of Fallot [1,2,7]. The natural progression of these defects is often associated with the development of severe complications. In children with acyanotic CHDs, the primary causes of death are congestive heart failure and the development of pulmonary hypertension. In contrast, children with cyanotic CHDs have a high mortality rate due to profound hypoxemia and thromboembolic complications [1,3,5].

The key mechanisms underlying impaired cardiac hemodynamics in these defects include volume or pressure overload of cardiac chambers. Prolonged exposure to these conditions depletes compensatory mechanisms, leading to chamber dilation and hypertrophy, which subsequently results in heart failure. Heart failure, in turn, causes systemic hemodynamic disturbances, contributing to either pulmonary hyperemia or hipoemia. This leads to the clinical manifestations of circulatory hypoxia—regional in VSD and ASD, and diffuse in Tetralogy of Fallot [1,5,9,10].

Pulmonary hyperemia (pulmonary hypertension) in VSD and ASD is a significant risk factor for recurrent pneumonia, while diffuse hypoxia can lead to multiple organ dysfunction. Structural remodeling of the heart due to dilation and hypertrophy, along with hypoxic, ischemic, and metabolic disturbances in the myocardium, may contribute to conduction system disorders and be accompanied by various arrhythmias, which further aggravate the clinical course of CHDs

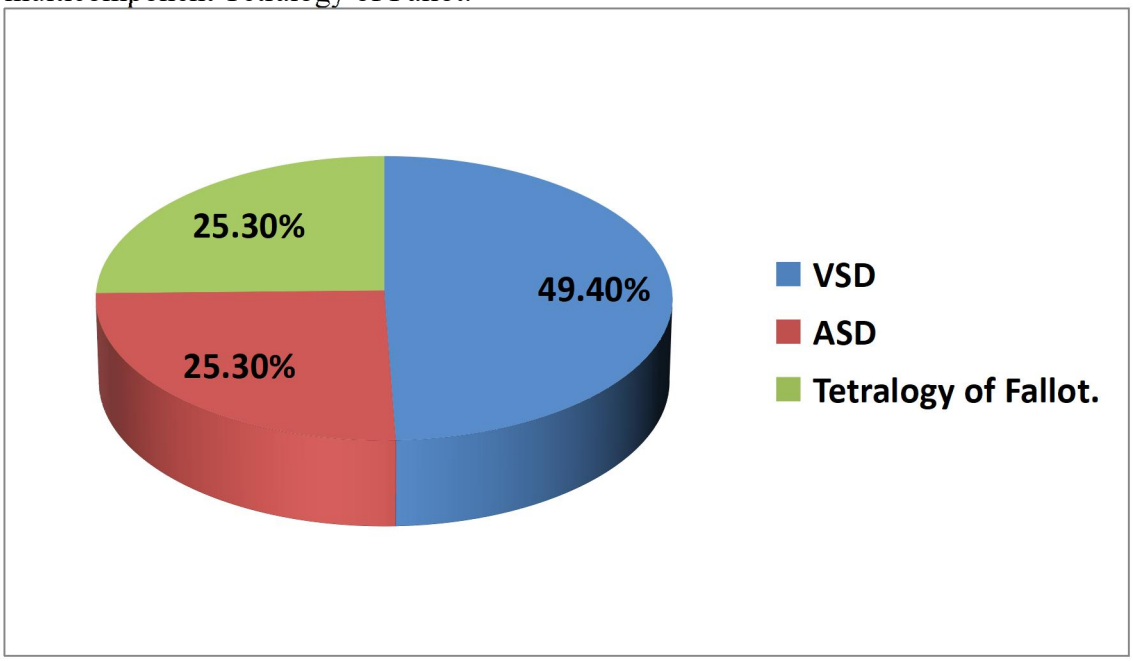
[1,4,5].

In this regard, our objective was to study the impact of arterial hypoxia on the course of congenital heart defects (CHDs) in young children.

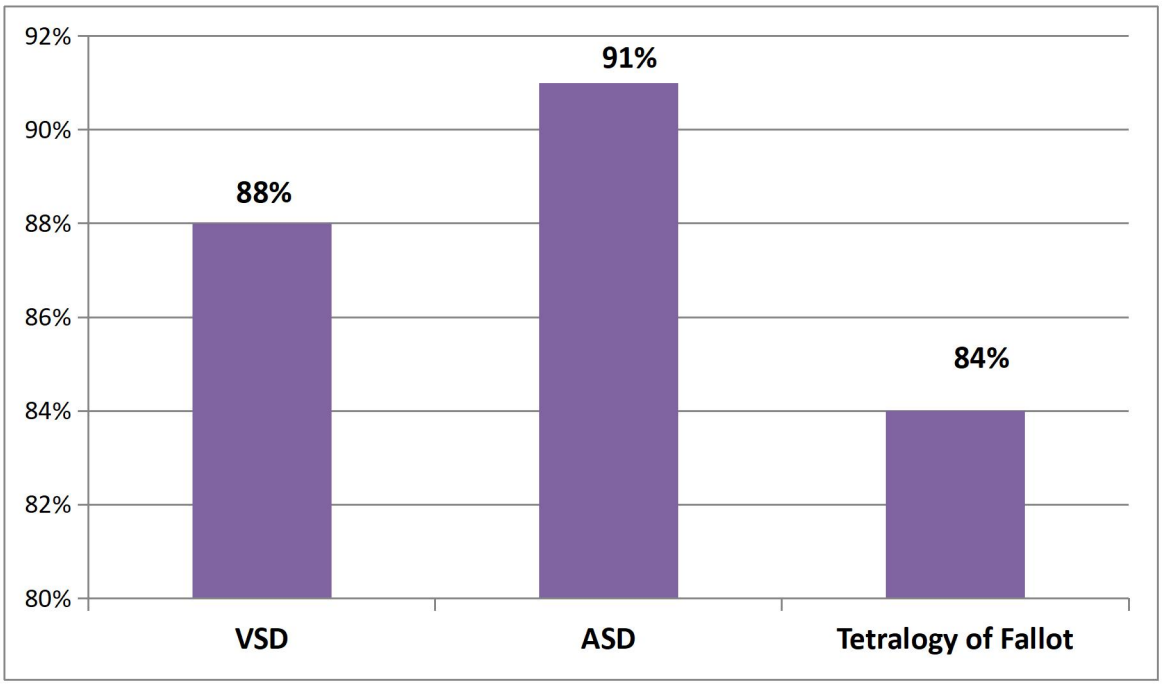
Results: The study and data collection were conducted at the Andijan Regional Multidisciplinary Children's Center. To address the objectives of this research, we examined children who received treatment in the Cardiorheumatology Department during the year 2024, along with a retrospective review of medical records.

The study included 71 children aged from 6 months to 3 years, of whom 72% were between 1 and 3 years old. In terms of gender distribution, boys predominated (55% compared to 45%), both in the group under 1 year of age and in the older age group.

Based on the hemodynamic patterns of the circulatory system, we selected patients with septal defects and pulmonary hypertension: specifically, ventricular septal defect (VSD) and atrial septal defect (ASD). The cyanotic defect included in our study was the complex, multicomponent Tetralogy of Fallot.



Upon hospital admission, 81,7% of the children were assessed as being in severe condition, primarily due to symptoms of grade II heart failure. Capillary blood oxygen saturation levels were measured in all children at the time of admission. The lowest oxygen saturation levels—ranging from 91% to 84%—were observed in children with Tetralogy of Fallot. In children with septal defects, the saturation levels were as follows: 94–88% in cases of ventricular septal defect (VSD) and 93–91% in atrial septal defect (ASD).



Frequent respiratory infections and repeated hospitalizations were observed in all of the children included in our study. The results we obtained are consistent with data reported in the literature.
The nature of complaints in all examined children with congenital heart defects (CHDs) was dependent on the degree of hypoxia. The most common and universal symptom, regardless of the type of hemodynamic disturbance, was dyspnea, which was recorded in 88,6% of children with ventricular septal defect (VSD), 77,8% of those with atrial septal defect (ASD), and in all children with Tetralogy of Fallot. In children with septal defects, the incidence of dyspnea was associated with excessive pulmonary blood flow, whereas in Tetralogy of Fallot, it was caused by arterial hypoxemia due to right-to-left shunting.
Hemodynamic disturbances in these heart defects were also reflected in the results of electrocardiographic (ECG) examinations. Regardless of the degree of arterial hypoxemia, abnormalities in cardiac automatism were represented by sinus tachycardia and sinus tachyarrhythmia. Increased electrical activity of the right ventricle was recorded in children with both ventricular and atrial septal defects.
Conduction disorders of varying frequency were observed in all examined children. Incomplete right bundle branch block (RBBB) was detected in 80% of children with VSD, 33.3% with ASD, and 50% with Tetralogy of Fallot. Complete RBBB was found exclusively in children with Tetralogy of Fallot (5,6%). First-degree atrioventricular (AV) block was registered in three children (8,6%) with large ventricular septal defects and in one child with Tetralogy of Fallot.

ECG changes in children with VSD (n=35).

ECG sign	Abs.	%
Sinus tachycardia	26	74,3%
Sinus arrhythmia	9	25,7%
Incomplete right bundle branch block (RBBB)	28	80%
AV block 1 degree	3	8,6%
High electrical activity of the right ventricle	7	20%
Left ventricular hypertrophy	18	51,5%
Right ventricular hypertrophy	17	48,5%

Tabl. 1
ECG changes in children with ASD (n=18).

ECG sign	Abs.	%
Sinus tachycardia	18	100%
Incomplete right bundle branch block (RBBB)	6	33,3%
High electrical activity of the right ventricle	9	50%
Right ventricular hypertrophy	11	61,1%

Tabl.2

ECG changes in children with tetralogy of Fallot (n=18).

ECG sign	Abs.	%
Sinus tachycardia	3	16,7%
Sinus tachyarrhythmia	8	44,4%
Incomplete right bundle branch block	9	50%
Complete right bundle branch block	1	5,6%
AV block 1 degree	1	5,6%
Right ventricular hypertrophy	18	100%

Tabl. 3

Electrocardiographic signs of right ventricular hypertrophy were observed in 48,5% of children with ventricular septal defect (VSD), 61,1% of those with atrial septal defect (ASD), and in all children with Tetralogy of Fallot. Signs of left ventricular hypertrophy were noted in 51,5% of children with VSD.

Structural changes in heart chambers and the dimensions of congenital defects were assessed based on echocardiography findings. Among children with VSD (35 cases), the largest proportion had defects measuring 4–8 mm (48,6%), followed by those with defects ≤4 mm (34,3%), and defects >8 mm in 17,1% of cases. For ASD (18 children), the distribution by defect size was as follows: up to 5 mm – 61,1% of cases, and 6–10 mm – 38,9%.

Among children with Tetralogy of Fallot, 66,7% had a ventricular septal defect measuring 6–10 mm, while the remaining children had defects larger than 10 mm. Regarding the localization of right ventricular outflow tract obstruction, 72,2% had narrowing in the infundibular region, while in 27,8% the stenosis was located at the level of the pulmonary valve.

Pulmonary hypertension in children with septal defects manifested on chest radiography as an enhanced pulmonary vascular pattern along the arterial branches. In contrast, in Tetralogy of Fallot, increased pulmonary translucency was due to a reduced vascular pattern. Cardiomegaly was identified in all examined children—74,6% due to right ventricular enlargement, and 25,4% due to left ventricular enlargement, the latter observed exclusively in children with VSD.

Thus, we established a correlation between arterial hypoxia and both the course and severity of clinical manifestations of CHDs. The lower the oxygen saturation in arterial blood, the more severe the clinical symptoms and the more pronounced the cardiac rhythm disturbances, which may serve as an additional prognostic risk factor in congenital heart defects.

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