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A MODERN VIEW ON THE CLASSIFICATION AND DIAGNOSIS OF THE OPEN ARTERIAL DUCT (REVIEW)

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Abstract. We have analyzed domestic and foreign sources, on the basis of which a variant of systematization of the existing author's classifications of the patent arterial duct (PAD) is proposed and the main diagnostic studies used to identify a functioning duct are described. Mandatory diagnostic measures for suspected PAD include assessment of the clinical picture (subjective and objective examination data) as well as non-invasive methods (radiography, electrocardiography, echocardiography, including three-dimensional variant, multispiral computed tomography (MCT), perfusion index, and infrared spectroscopy). The important role of laboratory diagnostic methods was noted: it was shown that the level of plasma B-type natriuretic peptide (NT-proBNP) and the level of cardiac troponin T (cTnT) can be used as biomarkers indicating the presence of PAD and allowing the determination of treatment approaches.

Key words: patent arterial duct; anatomy; epidemiology; classification; diagnostics; fetal communications.

СОВРЕМЕННЫЙ ВЗГЛЯД НА КЛАССИФИКАЦИЮ И ДИАГНОСТИКУ ОТКРЫТОГО АРТЕРИАЛЬНОГО ПРОТОКА (ОБЗОР)

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Резюме. Нами проведен анализ отечественных и зарубежных источников, на основании чего предложен вариант систематизации существующих авторских классификаций открытого артериального протока (ОАП), а также описаны основные диагностические исследования, применяемые для выявления функционирующего протока. В число обязательных диагностических мер при подозрении на ОАП входят оценка клинической картины (субъективные и объективные данные осмотра), а также неинвазивные методы (рентгенография, электрокардиография, эхокардиография, в том числе трехмерная, мультиспиральная компьютерная томография (МСКТ), индекс перфузии, инфракрасная спектроскопия). Отмечена важная роль лабораторных методов диагностики: было показано, что уровень плазменного натрийуретического пептида B-типа (NT-proBNP) и уро-

вень сердечного тропонина Т (cTnT) могут использоваться в качестве биомаркеров, указывающих на наличие ОАП и позволяющих определять подходы к лечению.

Ключевые слова: открытый артериальный проток; анатомия; эпидемиология; классификация; диагностика; фетальные коммуникации.

INTRODUCTION

Currently, there is no unified statistical record of the incidence of patent (open) ductus arteriosus (PDA) in the Russian Federation. This omission is due to a combination of factors, in particular, the lack of clear criteria for pathological PDA and a unified internationally recognised classification, as well as underestimation of the social significance by the medical community. For example, the last revision of the "Patent Ductus Arteriosus" clinical guidelines took place back in 2018, during which there were no significant changes in their content compared to the previous version [3]. At the same time, the statistics given in the text of the recommendations contain information only from 1996: "Isolated ductus arteriosus occurs approximately 0.14–0.3 per 1,000 live births, 7 % in the structure of all congenital heart disease (CHD) and 3 % in the structure of critical congenital heart disease" [3, 5, 12].

At present, the use of the above data is inappropriate for a number of reasons. Firstly, the goals to reduce the infant mortality rate in the structure of total mortality have been achieved, which is due to the implementation of the National Project "Health Care" and Presidential Decree N 240 of 29.05.2017. "On the Announcement of the Decade of Childhood". [14, 22, 31, 32]. Secondly, there is a positive trend to overcome the demographic hole of the 1990s: there is an increase in the birth rate from 1996 to 2020, including by nursing more children with extremely low birth weight (ELBW) [21, 31]. Thus, in 2018, 97,106 newborns were born with various degrees of prematurity, of which only 2.38 % (n = 2312) died [31]. The considered indicator in 2014 was 1.5 times higher (3.67 % with n = 4071). Given the fact that the incidence of PDA is inversely proportional to gestational age, it can be assumed that the current incidence of PDA is much higher [2, 4, 7, 10, 20]. In addition, statistics published by researchers from countries with different socioeconomic levels of development also show a higher prevalence of PDA (China, 1.97 per 1000 live births; South Korea, 8.1 per 1000 live births; USA, 10 per 1000 live births) [17, 46, 63].

The life expectancy of this group of patients depends on the time of development of fatal complications, which, first of all, are a rupture of the ductus against the background of its calcification or an aneurysmal dilation, development of bacterial endocarditis and/or a severe heart failure. The actual value of the described index does not exceed 40 years [12, 13, 35, 38, 39, 71].

PDA CLASSIFICATIONS: A VIEW THROUGH THE TIME

Several classifications have been proposed to evaluate the structure of the PDA. For example, Hans Bunkle (1980) distinguished the following types of the patent ductus arteriosus: funnel-shaped, tubular and terminal [5].

After the analysis of domestic and foreign sources, we propose the following approach to systematisation of the existing author's classifications of the open ductus arteriosus (Fig. 1): 1 — mixed; 2 — haemodynamic; 3 — descriptive; 4 — angiographic.

The mixed classifications include the classification of J.P. Hubbart (1943) and G. Tikov (1969). The Hubbart classification is based on clinical data and data of instrumental diagnostics, thanks to which it is possible to determine the

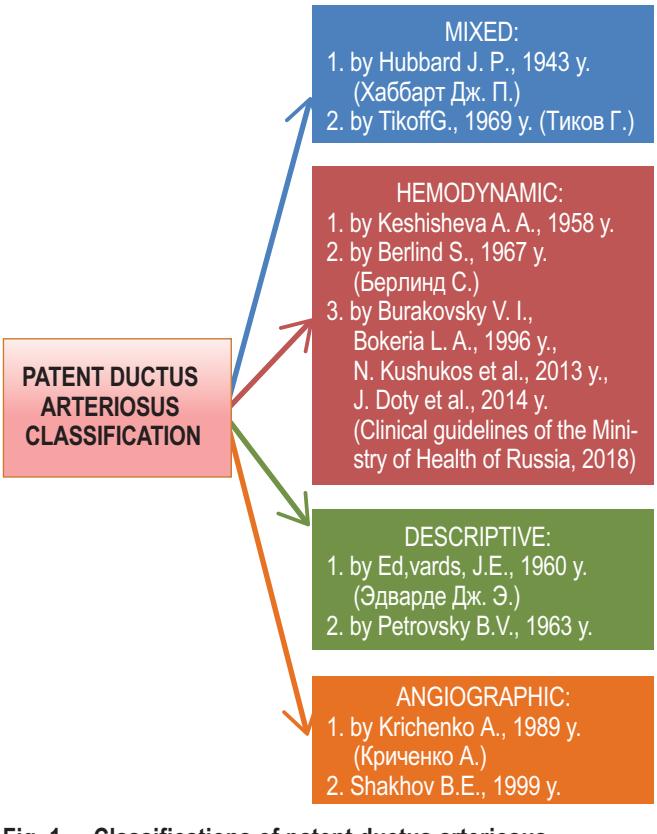

Fig. 1. Classifications of patent ductus arteriosus

degree of compensated consequences of ductal functioning (Table 1) [50].

In turn, the classification according to G. Tikov (Table 2) represents the division of PDA into types 1–4, in which the 1st and 2nd are small defects, and the 3rd and 4th are large defects [72]. The classification is based on the pathophysiological mechanisms of a functioning ductus arteriosus (DA), its complications and haemodynamic changes in the circulatory system [72].

The haemodynamic classifications include the classifications of Keshisheva (1974), Berlind (1967) and the classification from the clinical guidelines on PDA (2018).

According to Keshisheva, three stages of haemodynamically significant PDA are distinguished according to the level of severity of complications [13]:

- Stage I — uncomplicated nonocclusion of the arterial duct, in which pulmonary hypertension does not make changes in the clinical picture;
- Stage II — complicated ductus arteriosus non-enclosure, in which pulmonary hypertension makes changes in the clinical picture;
- Stage III — patent ductus arteriosus complicated by pulmonary hypertension and bacterial endarteritis.

Table 1

Classification of patent ductus arteriosus according to J.P. Hubbart [50]

Patent ductus arteriosus	Compensated	Uncompensated
Cyanosis	Absent	Absent*
Symptom of drum sticks	Absent	Absent
Murmur	Increased systolic and decreased diastolic murmurs	
Localisation of maximum murmur	The lung area	
Diastolic BP	Normal or slightly reduced DBP	Reduced DBP
Blood stasis in the lungs	Absent or negligible	Expressed
Deviation of the electrical axis of the heart	Absent or slight to the right / left	

* Cyanosis may be due to congestive insufficiency.

Table 2

Classification of the types of patent ductus arteriosus according to G. Tikov: based on the size of the defect and pulmonary vascular resistance [72]

A patent ductus arteriosus				
Type	I	II	III	IV
Defect size[1]	Small	Small	Large	Large
Pulmonary vascular resistance	Normal	Elevated	Elevated, but less than systemic vascular resistance	Elevated, greater than systemic vascular resistance
Pulmonary hypertension	Absent	Yes (mild to moderate)[2]	Yes[3]	
Involved ventricle (loading type)	Left (volume load)	Left (volume load) and right (pressure load)	Left (volume loading) and right (pressure loading)	Left (pressure loading) and right (volume loading)
Murmur	Continuous	Continuous or atypical	Atypical	Atypical
Subacute bacterial endocarditis	May occur	May occur	Occurs rarely[4]	
PBF: SBF[5]	Variable, in adults usually <2:1	Variable, in adults usually <2:	Variable, greater than 1:1	Variable, lower than 1:1
Pulse pressure	Wide	Wide or normal	Variable, but can be wide	Variable

[1] By “small” and “large” we mean sizes smaller or larger than those necessary to ensure mandatory equality between the pulmonary and systemic arterial circuits throughout the cardiac cycle.
[2] There is no mandatory equality of systemic and pulmonary arterial pressure.
[3] Mandatory equality of systemic and end-diastolic pressures in the pulmonary artery.
[4] Typically, the area affected is outside the juxtaductal zone.
[5] The ratio of pulmonary and systemic blood flow.

Table 3

Classification of patent ductus arteriosus according to S. Berlind [35]

A patent ductus arteriosus				
Group	1	2	3	4
Pulmonary BP, mm Hg.	Normal	30–69	70–89	≥90

In turn, according to the classification of S. Berlind (1967), four stages are distinguished depending on the level of pressure in the pulmonary artery (Table 3) [35].

In 2018, the working group for the preparation of clinical guidelines of the Association of Cardiovascular Surgeons of Russia, headed by Academician L.A. Bokeria, presented a classification of the severity of the malformation. It was based on the works of V.I. Burakovsky, L.A. Boqueria (1996), Nicholas Kushukos et al. (2013), and John Doty et al. (2014) [3, 5, 56, 77]:

- Stage I — pulmonary artery (PA) pressure in systole does not exceed 40 % of arterial pressure;
- Stage II — pressure in the PA is 40–75 % of arterial pressure (moderate pulmonary hypertension);
- Stage III — PA pressure is more than 75 % of arterial pressure (marked pulmonary hypertension with the preservation of left-right discharge of blood);
- Stage IV — PA pressure equals or exceeds the systemic pressure (severe pulmonary hypertension, which leads to the occurrence of right-to-left effusion).

We should not forget about the classification of natural functioning of the patent ductus arteriosus created by V.I. Burakovsky and L.A. Bokeria (1996), where 3 stages are defined [3, 5]:

1) the primary adaptation;
2) the relative compensation;
3) sclerotic changes of pulmonary vessels.

PDA is also divided, according to the clinical guidelines "Haemodynamically significant ductus arteriosus in the preterm newborn"[1], according to the degree of haemodynamic significance, into mild, moderate and severe (Table 4).

Haemodynamically significant patent ductus arteriosus will be if:

1) the diameter of the duct is greater than 1.5 mm;
2) there is a left-right discharge on the duct;
3) one of the criteria is present according to echoCG and Dopplerometry data (Table 4).

Two classifications can be attributed to descriptive classifications — those of J.E. Edwards (1967) and B.V. Petrovsky (1963). Edwards distinguishes 3 types (based on the shape of the duct): cylindrical, funnel-shaped, and terminal [41, 76]. B.V. Petrovsky's classification is based not on anatomical norm (shape, size and position), but on the basis of complications and concomitant pathology of isolated and combined DA [24]. Thus, isolated ductal obstruction is divided into uncomplicated and complicated (bacterial endarteritis, pulmonary hypertension, ductal or pulmonary trunk aneurysm, cardiac decompensation). Concomitant DA is divided into enhancing functional disorders, e.g. in atrial or interventricular septal defect, aortopulmonary fistula, Eisenmenger's disease, Lautembasch disease, and compensating functional disorders caused by malformations, e.g. Tetralogy of Fallot, Cantrell's disease and Corvisar's symptom complex.

B.V. Petrovsky and A.A. Keshisheva (1963) also distinguished the cylindrical form of the PDA, truncated cone form, and slit-shaped form [24].

The authors describing and systematising various forms of the duct to a greater extent were based on its study during sectional examination or during surgical interventions, which was regarded by other authors as a distortion of the true parameters of the duct because "the influence of vascular tone on its shape is not taken into account" [23, 66]. The authors of angiographic classifications are sceptics of this kind. Such an approach to the definition of PDA types corresponding to their anatomical structure allows fixing the image on X-ray film and further visually assessing the contours of the open arterial duct [23].

Modern classifications developed on the basis of X-ray contrast enhancement and related to angiographic classifications are the classification of A. Krichenko (1989) and its modification developed by B.E. Shakhov et al. (1999).

A. Krichenko singled out 5 types of the duct according to its shape and location. The classification looks as follows (Fig. 2) [57]:

Type A[2]: conical shape (the narrowest place of the duct is its pulmonary part; there is a well-differentiated aortic ampulla (analogue of the funnel-shaped type of the duct)).

Type B[3]: short duct, narrowest in the aortic part.

Type C: tubular shape without narrowing.

Type D: tubular shape with multiple narrowings.

Type E: unusual configuration of the duct — elongated conical shape with multiple constrictions (stenosed part, located away from the tracheal margin).

[1] Razumovsky A.Yu. Haemodynamically significant arterial duct in a premature neonate, Russian public organisation to promote the development of neonatology "Russian Society of Neonatologists" (RSN), Public organisation "Russian Association of Perinatal Medicine Specialists" (RAPMS) / A.Yu. Razumovsky, D.N. Dyagterev, R.R. Movsesyan [et al]. — Moscow: Ministry of Health of the Russian Federation, 2020. — C. 83. (In Russian).

[2] According to the location of the pulmonary end of the ducts at types A and B relative to the trachea, three subgroups were distinguished in each group: 1 — pulmonary opening of the duct is located to the right of the trachea; 2 — in the middle of the tracheal shadow; 3 — to the left of the trachea.

[3] The same

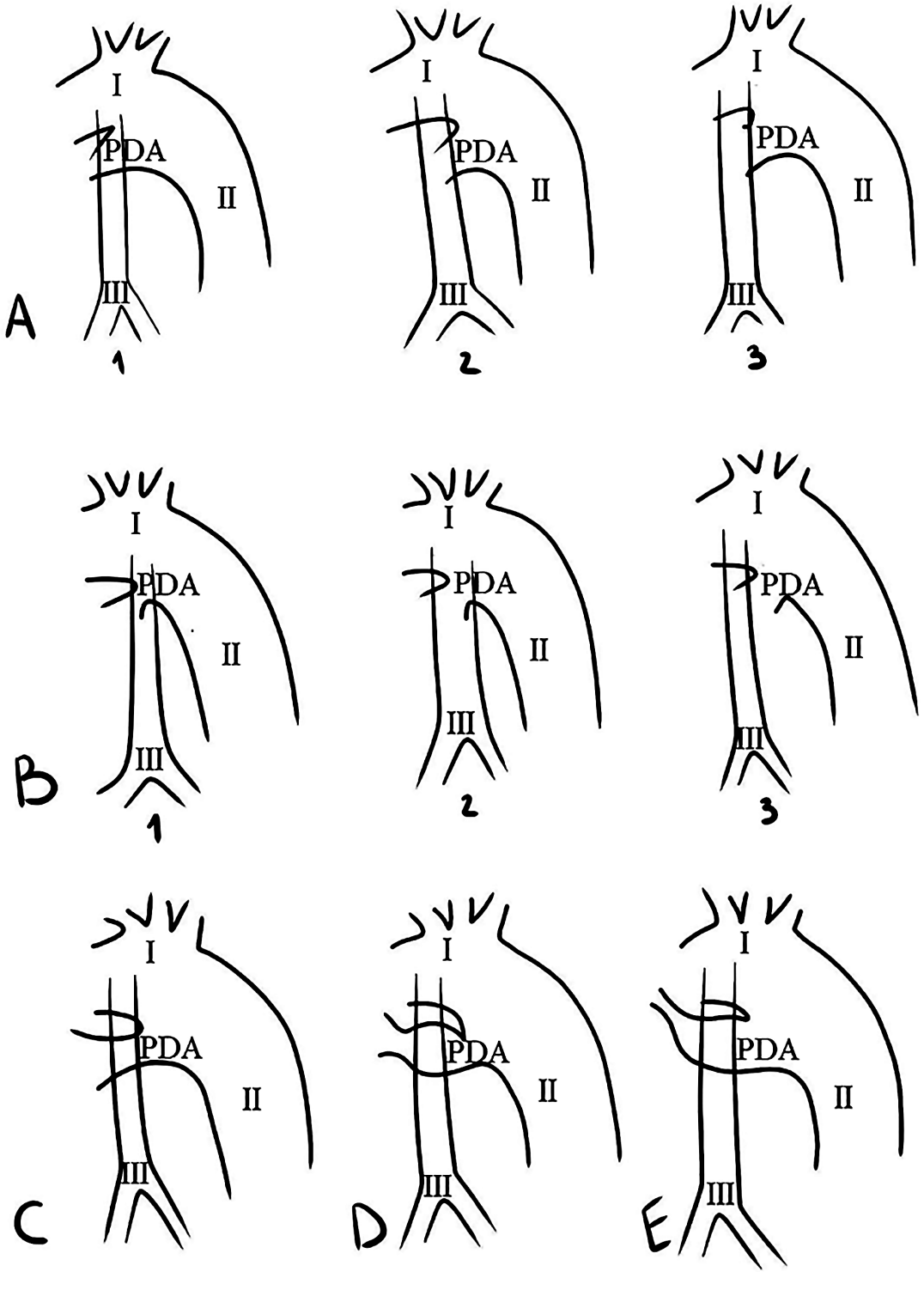

Fig. 2. Angiographic classification of the open ductus arteriosus: A — type A; B — type B; C — type C; D — type D; E — type E; 1 — pulmonary opening of the duct is located to the right of the trachea; 2 — in the middle of the tracheal shadow; 3 — to the left of the trachea; I — aortic arch; II — descending part of the aorta (thoracic aorta); III — trachea; PDA — open arterial duct (patentus ductus arteriosus) (by: [57])

A kind of contrast and at the same time a modification of A. Krichenko's classification is a variant of the authors' team led by B.E. Shakhov (Fig. 3). In the authors' opinion, the already known method is not without a number of drawbacks: it does not distinguish DA types, which are combinations of funnel-shaped and tubular types. B.E. Shakhov

and co-authors note that A. Krichenko, for example, "lacks quantitative characteristics of the duct, which does not allow to accurately select the parameters of the occlusive spiral during the manipulation of endovascular occlusion of the open arterial duct" [23]. At the same time, the use of tracheal shadow as a reference point is non-functional, because the X-ray anatomical picture of the thoracic cavity organs changes when the body is rotated.

The modernised classification is described on the basis of the analysis of angiographic images (n = 119), according to the results of which 3 types of the duct were proposed (Fig. 3) [23]:

- Type I open arterial duct type — predominantly funnel-shaped — represents a duct in which the ratio of the length of the duct ampulla to the total length of the duct is greater than 2/3 inclusive.
- Type II of the open arterial duct — intermediate type — represents the duct in which the ratio of the ductal length to the total length of the duct varies in the range from 1/3 to 2/3 (the most frequent type according to the results of the study (n = 52) — 44 %). It should be noted that according to the method of A. Krichenko (1989) there is no allocation of patients with open ductus arteriosus of intermediate type.
- The III type of the duct — predominantly tubular — is a duct in which the ratio of the ductal length to the total length of the duct is less than 1/3 inclusive.

PDA DIAGNOSTICS

At the current stage of the diagnostic search, the physician should resort to the full range of necessary clinical and instrumental methods of examination. The mandatory diagnostic measures in suspected PDA include assessment of the clinical picture (subjective and objective examination data), as well as non-invasive methods (radiography, electrocardiography, echocardiography, including three-dimensional, multispiral computed tomography (MSCT), perfusion index, infrared spectroscopy) and laboratory diagnostics.

CLINICAL PICTURE

Complaints in PDA are nonspecific [1, 3, 10, 11, 16, 33]. The severity of the clinical picture depends, to a greater extent, on the size of the duct and the stage of haemodynamic disorders [3, 10, 16, 55]. The manifestations range from asymptomatic to extremely severe [48, 52].

The main manifestations in PDA are [3, 11, 13, 16, 19, 19, 33]:

1) dyspnoea of varying intensity;
2) fatigue occurring during physical activity;
3) cyanosis: a) which occurs only when shouting, pushing, thus causing cyanosis of the lower half of the body (the iliac region and lower limbs); b) the permanent cyanosis occurs only in adults, with irreversible changes in shunt blood flow and sclerotic form of pulmonary hypertension;
4) delayed physical development, e.g. growth retardation;
5) heart irregularities (an increased apical thrust, high, rapid or racing pulse, tachycardia);
6) frequent pulmonary infections;
7) in premature infants: tachypnoea or apnoea (non-specific in the presence of respiratory distress syndrome (RDS), abdominal bloating, regurgitation, impaired digestion (mesenteric blood flow 'stealing' syndrome), hepatomegaly, increased oxygen demand, increased parameters of artificial lung ventilation (ALV) in complicated forms of PDA course.

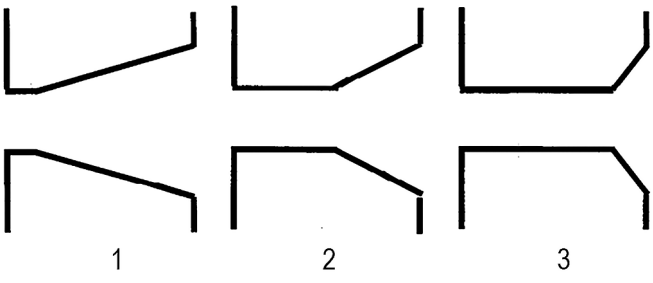

Fig. 3. Angiographic types of patent ductus arteriosus: 1 — funnel-shaped type; 2 — intermediate type; 3 — tubular type (according to: [23])

OBJECTIVE EXAMINATION DATA

On auscultation, a characteristic systolo-diastolic ("machine") murmur is heard, which was first observed and described by Scottish physician George Gibson in 1898. [25, 71]. The murmur can be systolic (~60% of PDA cases) or systolo-diastolic (~20 % of PDA cases), depending on the difference between aortic and pulmonary artery pressure [25]. The murmur is typically localised at the base of the heart in the second to third intercostal space on the left and may be conducted dorsally. There is a group of "silent ductus" — "mute" or "silent" ducts, occurring in 11–20 % of cases, when the characteristic murmur is not heard [14].

NON-INVASIVE METHODS

Electrocardiography and chest radiography are of little diagnostic value. In the *chest radiography,* specialists describe congestive full bloody pulmonary fields (decreased lung pneumatisation, atelectasis of the apical lobes) with a significant increase in the heart shadow due to its left chambers [3, 11, 12, 33, 35, 58, 60]. The pulmonary arteries may be dilated.

Electrocardiographic study (ECG) more often shows only increased left-sided potentials (a poorly informative method of diagnosing PDA in premature and preterm neonates) [3, 5, 33, 64, 72, 77]. However, with marked hypertension of the small circle, signs of hypertrophy of the right heart chambers may appear [11, 33]. In a number of studies, the authors found that only 22% of cases have ECG changes.

The "gold standard"[1] of PDA diagnosis, and at the same time the most reliable and valid study, is *transthoracic echocardiography (echocardiography)* [1, 3, 4, 11, 18, 25, 30, 46, 49, 49, 50, 54, 69]. In this study, it is important to assess the anatomical parameters of the duct — size (length and diameter), shape, and position [6, 18, 26]. EchoCG has been proved to have a higher sensitivity in diagnosis (Table 4) compared to clinical data (manifestations of PDA are observed on average on the 4th day from the moment of birth) [3, 11, 26, 42, 49].

PDA can be visualised from two accesses — the parasternal and the suprasternal [18, 25, 26]. Aortopulmonary and coronary-pulmonary fistulas are visualised only from the parasternal access, which may be an important ultrasound differential diagnostic sign [26].

Postnatal echocardiography is indicated in all preterm newborns during the first 48 hours when they are undergoing ventilatory support, when surfactant is administered and when pulmonary haemorrhage develops [1, 3, 11, 49, 54, 54, 60, 66]. A repeat study is performed 48 hours after the previous one if the preterm infant shows an increase in oxygen demand, deterioration of ventilation parameters, development of mixed or metabolic acidosis, and the appearance of a systolic murmur [1, 30, 49, 65, 69].

According to the results of the dissertation study by S.O. Efremov (2007) defined the criteria of haemodynamically significant PDA [10]: left-right direction of blood flow through PDA (bidirectional flow occurs in newborns with PDA and high pulmonary hypertension) in combination with LV enlargement (the end diastolic volume >50 ml/m^2), and LA/aortic root ratio >1.2 LV/aortic root ratio > 1.92.

H. Evans[2] et al. (2004), P.J. McNamara[3] et al. identified a number of other criteria of significant PDA, namely [16, 51, 73]:

1) left atrial (LA) dilation (LA/aortic root ratio equal to or greater than 1.4);
2) diastolic turbulent blood flow in the left pulmonary artery when the internal diameter of the duct is greater than 1.5 mm;
3) reverse diastolic blood flow in the descending aorta and superior mesenteric artery;
4) left ventricular (LV) enlargement, increased mean and diastolic blood flow in the pulmonary artery and inverse ratio of peak blood flow velocity during systole and diastole in the left heart, indicating pulmonary hyperperfusion.

Doppler imaging with colour flow imaging makes the study more accurate, detects retrograde blood flow in the descending aorta, and helps to exclude associated congenital heart defects, such as atrial and interventricular septal defects [11, 18, 26, 34, 53, 60]. It is also used to more accurately determine the strength of shunt blood flow, pulmonary hypertension (LA pressure) [34]. It is important to distinguish PDA from the aortopulmonary window, which may be located in the distal part of the ascending aorta [7, 11, 25].

Multispiral computer tomography. The procedure is performed according to the standard research protocol (including selective angiography mode), however, when studying the obtained data (if PDA is suspected), it is recommended to analyse it on the basis of oblique MIP reformations, plotted along the axes of the ascending aorta and descending aorta (thoracic aorta), three-dimensional reconstructions [15, 40, 42]. The specificity of MSCT in terms of PDA detection, according to some researchers, reaches 100% [15]. Using the method of spiral CT of thoracic organs, we obtain an accurate anatomical picture of its structures (shape, size, spatial position, syntopy and skeletotopy), as well as clear morphometric data, which allows to perform conservative treatment and determine indications for surgical treatment in a balanced way. It should not be forgotten that the method is associated with radiation and contrast loads, which requires from the doctor a more differentiated approach to the use of the method.

The perfusion index in PDA is determined on the basis of pulse wave strength calculation of the preductal index (on the upper limbs) and postductal index (on the lower limbs), which is always higher due to hypervolemia [48, 59, 71]. Under normal conditions, the blood flow in the postductal aorta is unidirectional, but in the presence of a functioning shunt, the blood flow during diastole is directed into the duct, as a result of which Doppler imaging shows first an isolinear and then retrograde blood flow [18, 26, 60, 68, 69]. Retrograde flow is associated with the ratio of pulmonary to systemic blood flow, with pulmonary blood flow 60% greater than systemic blood flow [11].

The near-infrared spectroscopy (NIRS) is based on three biophysical indices — the ability of tissues to transmit

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[3] McNamara PJ, Sehgal A. Toward rational manage- ment of the patent ductus arteriosus: the need for disease stag- ing. Arch. Dis. Child Fetal Neonatal Ed. 2007; 92: 424–427

Table 4

Echocardiographic signs of PDA in preterm neonates (World Health Organisation protocol), 2014[1]

EchoCG findings	None PDA	Minor PDA	Moderate PDA	Large PDA
Signs of PDA				
Minimum diameter of PDA, mm	0	< 1,5	1,5–3	> 3
PDA flow velocity V_{max}, cm/sec	0	> 2	1,5–2	< 1,5
PA antegrade diastolic blood flow, cm/sec	0	> 30	30–50	> 50
Pulmonary hypercirculation				
Left atrium/Aortic root diameter ratio	1,1	1,1–1,4	1,4–1,6	> 1,6
Left Ventricle/Aortic Root Diameter Ratio	1,9 ± 0,3	–	2,2 ± 0,4	2,27 ± 0,27
E-wave / A-wave ratio	< 1	< 1	1–1,5	> 1,5
Isovolaemic relaxation time, ms	< 55	46–54	36–45	< 35
LV stroke volume index	0,34 ± 0,09	–	0,26 ± 0,03	0,24 ± 0,07
Systemic hypoperfusion				
Retrograde diastolic blood flow in aorta, % of antegrade blood flow	10	< 30	30–50	> 50
Aortic shock ejection, ml/kg	≤ 2,25			≥ 2,34
LV cardiac output, ml/kg per minute	190–310			> 310
Ratio of LV ejection to blood flow in the VCS	2,4 ± 0,3			4,5 ± 0,6

Note: VCS — vena cava superior; CA, PA — pulmonary artery; LV — left ventricle; LA — left atrium.
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light in the infrared range, the absorption of light radiation by chromophores and the dependence of the degree of light absorption by chromophores on the level of blood oxygen saturation [9, 24]. Calculation of the index (oxyhaemoglobin level multiplied by the scattering constant, divided by the total amount of haemoglobin multiplied by the scattering constant and multiplied by 100%) is performed to determine regional oxygenation [29, 47, 67]. Basically, tissue (peripheral), visceral (splanchnotic) and cerebral oxygenation (CO) are distinguished [28, 29, 59]. In preterm neonates, the level of CO is 79 ± 4.06 % in the left hemisphere and 84.89 ± 5.1 % in the right hemisphere, while in premature neonates it ranges from 55 to 85% (mean value = 67 ± 8 %) [28, 36, 59, 61, 67].

It has been found that in various CHD, the level of CO is quite low [28]. The CO level in PDA is one of the diagnostic search methods, but when we observe a haemodynamically significant duct, determination of the CO level becomes especially important as a diagnostic criterion to determine further treatment tactics [36, 47, 52]. Increased blood shunting in haemodynamically significant PDA leads to systemic hypoperfusion, which is also determined by analysing the level of CO [28].

LABORATORY DIAGNOSTICS

In addition to the above studies, biochemical markers have received much attention in the diagnosis of PDA

Table 5

NT-proBNP concentrations at different periods of a child's life [62]

Age	NT-proBNP concentration in plasma, median value (pg/ml)
The 1st day after birth	3183
The 14th day after birth	2210
1 year after birth	141
2 years after birth	126
6 years after birth	70
14 years after birth	52

[43, 44, 62]. It was shown that the level of plasma B-type natriuretic peptide (NT-proBNP) (Table 5) and cardiac troponin T (cTnT) can be used as biomarkers indicating the presence of PDA and allowing to determine the treatment tactics [8, 27, 37, 43]. Studies by S. Budd et al. showed that if the concentration of the level of N-terminal fragment of cerebral natriuretic propeptide (NT-proBNP) exceeds 5900 pg/ml (reference interval — from 0 to 125 pg/ml), it is possible to diagnose haemodynamically significant PDA in a premature newborn [37].

Determination of NT-proBNP content in PDA contributes to adequate assessment of functional disorders in the heart muscle, as well as the effectiveness of conservative

and surgical treatment [8, 28, 37, 44, 45, 74]. Increased NT-proBNP level reflects the degree of pressure changes in the cardiac cavities and is an independent prognostic factor of mortality in premature neonates with PDA [28, 43, 44, 75]. It is important that due to the influence on peripheral pulses NT-proBNP is involved in water-electrolyte metabolism and displays the degree of pressure transformation in cardiac cavities [28, 43, 55, 60].

CONCLUSION

Thus, we have attempted to systematise the available data on classifications of the open ductus arteriosus, and described the main diagnostic tests required to assess the condition of premature and preterm neonates with a functioning ductus arteriosus for the subsequent choice of their treatment.

ADDITIONAL INFORMATION

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