



<https://doi.org/10.29001/2073-8552-2024-39-4-162-170>  
УДК 616.125.6-089.844-053.2:616.125.2-07

# Left atrial shape and function after endovascular and surgery atrial septal defects correction in children

Alexander A. Sokolov, Viktor I. Varvarenko, Oleg A. Egunov, Andrey V. Smorgon

Cardiology Research Institute, Tomsk National Research Medical Center of the Russian Academy of Sciences (Cardiology Research Institute, Tomsk NRMC),  
111a, Kievskaya str., Tomsk, 634012, Russian Federation

## Abstract

**Introduction.** Atrial septal defects (ASD) are one of the most common congenital heart defects, accounting for about 10–15% of all congenital heart defects. Currently, endovascular treatments for ASD are considered the method of choice in most patients with secondary ASD. ASD occluders, after fixation on the atrial septum, close the shunt, eliminating the intercameral communication. However, these devices increase the stiffness of the septum, limit its mobility and presumably disrupt the mechanical function of the left atrium (LA).

**Aim:** To study the effect of surgical and endovascular correction on the size, shape and function of the left atrium and left ventricle in children.

**Material and Methods.** We retrospectively studied sequential echocardiograms of patients who underwent endovascular and surgical treatment of ASD in the period from 2006 to 2016 at the cardiology center in Tomsk, Russia. 756 patients with ASD were examined, 564 of them before and after endovascular closure and 192 after surgical correction. The duration of follow-up ranged from 1 day to 10 years, with an average of 3.6 years in the group receiving hardware treatment and 4.2 years in the group undergoing surgery. The control group consisted of 3393 healthy patients of the same age group. Echocardiographic images were obtained using an iE33 ultrasound scanner, Philips Ultrasound. Standard echo indicators of heart sizes and volumes were evaluated, including taking into account the body surface area and deviations of the studied parameters as a percentage of the predicted norm.

**Results.** 15 days after ASD closure, both groups showed a significant increase in the volume of the left ventricle and a decrease in the right chambers. During follow-up in the group receiving the devices, left atrial shape index (LASI) was significantly reduced, while in patients undergoing surgery, there was no significant increase in LASI. A change in the shape of the left atrium and a decrease in its volume were combined with a change in the effort in the left atrium (LAEF), determined by the Manning method.

**Discussion.** The study showed that both endovascular and surgical methods are effective in correcting ASD and lead to favorable results with respect to left atrial function. However, there is still uncertainty about the long-term effects after implanted devices used in endovascular procedures. Although these devices offer a minimally invasive solution, there is a problem associated with increased stiffness and limited mobility of the partition. Such changes can potentially alter the normal dynamics of atrial filling and contraction, affecting the overall function of the left atrium over time.

**Conclusion.** Endovascular correction of ASD in 35% of children was accompanied by a change in the shape of the left atrium a decrease in sphericity and an increase in ellipsoid. Changes in the shape of the left atrium persisted in 22% of patients after transcatheter correction in the long term. These changes were not accompanied by any disorders of contractility and volume of the heart chambers. In the group of children after surgical correction of ASD, the contractility and volume of the heart chambers did not significantly differ from those in the device group.

<b>Keywords:</b>	atrial septal defects (ASD); left atrium; right atrium; left atrial indexed volume; left atrial shape index; QP/QS.
<b>Funding:</b>	the study was carried out without financial support from grants, public, non-profit, commercial organizations and structures.
<b>Compliance with ethical standards:</b>	an informed consent was obtained from all patients.
<b>For citation:</b>	Sokolov A.A., Varvarenko V.I., Egunov O.A., Smorgon A.V. Left atrial shape and function after endovascular and surgery atrial septal defects correction in children. <i>Siberian Journal of Clinical and Experimental Medicine</i> . 2024;39(4):162–170. <a href="https://doi.org/10.29001/2073-8552-2024-39-4-162-170">https://doi.org/10.29001/2073-8552-2024-39-4-162-170</a> .

✉ Сморгон Андрей Владимирович, [sav@cardio-tomsk.ru](mailto:sav@cardio-tomsk.ru).

# Форма и функция левого предсердия после хирургической и эндоваскулярной коррекции дефекта межпредсердной перегородки у детей

А.А. Соколов, В.И. Варваренко, О.А. Егунов, А.В. Сморгон

Научно-исследовательский институт кардиологии, Томский национальный исследовательский медицинский центр Российской академии наук (НИИ кардиологии Томского НИМЦ),  
634012, Российская Федерация, Томск, ул. Киевская, 111а

## Аннотация

**Введение.** Дефекты межпредсердной перегородки (ДМПП) являются одним из наиболее распространенных врожденных пороков сердца. На их долю приходится около 10–15% всех врожденных пороков сердца. В настоящее время эндоваскулярные методы лечения ДМПП считаются методом выбора у большинства пациентов с вторичным ДМПП. Окклюдеры ДМПП после фиксации на межпредсердной перегородке закрывают шунт, устраняя межкамерное сообщение. Однако эти устройства повышают жесткость перегородки, ограничивают ее подвижность и, предположительно, нарушают механическую функцию левого предсердия.

**Цель:** изучение влияния хирургической и эндоваскулярной коррекции на размер, форму и функцию левого предсердия и левого желудочка у детей.

**Материал и методы.** Ретроспективно изучили последовательные эхокардиограммы пациентов, прошедших эндоваскулярное и хирургическое лечение ДМПП в период с 2006 по 2016 гг. в НИИ кардиологии Томского НИМЦ (Томск, Россия). Были обследованы 756 пациентов с ДМПП, из них 564 – до и после закрытия эндоваскулярным методом, 192 – после хирургической коррекции. Продолжительность наблюдения составляла от 1 дня до 10 лет, в среднем 3,6 года в группе, получавшей аппаратное лечение; 4,2 года – в группе, подвергшейся хирургическому вмешательству. В контрольную группу вошли 3 393 здоровых пациента той же возрастной группы. Эхокардиографические изображения были получены с использованием ультразвукового сканера iE33, Philips Ultrasound (Holland). Были оценены стандартные эхокардиографические показатели размеров и объемов камер, в том числе с учетом площади поверхности тела и отклонений исследуемых параметров в процентах от прогнозируемой нормы.

**Результаты.** Через 1–5 дней после закрытия ДМПП в обеих группах регистрировалось значительное увеличение объема левого желудочка и уменьшение правых камер. Во время наблюдения в группе с применением устройства индекс жесткости левого предсердия (LASI) был значительно снижен, в то время как у пациентов, перенесших операцию, существенного увеличения LASI не отмечалось. Изменение формы левого предсердия и уменьшение его объема сочеталось с изменением усилия в левом предсердии (LAEF), определяемого по методу Маннига.

**Обсуждение.** Исследование показало, что как эндоваскулярные, так и хирургические методы эффективны в отношении коррекции ДМПП. Они приводят к благоприятным результатам в отношении функции левого предсердия. Однако по-прежнему существует неясность касательно долгосрочных последствий применения имплантированных устройств, используемых при эндоваскулярных процедурах. Хотя эти устройства предлагают минимально инвазивное решение, существует проблема, связанная с повышенной жесткостью и ограничением подвижности перегородки. Такие изменения потенциально могут изменить нормальную динамику наполнения и сокращения предсердий, влияя на общую функцию левого предсердия с течением времени.

**Заключение.** Эндоваскулярная коррекция ДМПП у 35% детей сопровождалась изменениями формы левого предсердия (уменьшением сферичности и увеличением эллипсоидности), которые сохранялись у 22% пациентов после транскатетерной коррекции в отдаленные сроки. Эти изменения не сопровождалось какими-либо нарушениями сократительной способности и объема камер сердца. В группе детей после хирургической коррекции ДМПП сократительная способность и объем камер сердца достоверно не отличались от таковых в группе устройств.

<b>Ключевые слова:</b>	левое предсердие; дефекты межпредсердной перегородки; окклюдер; индекс левого предсердия; QP / QS.
<b>Финансирование:</b>	исследование выполнено без финансовой поддержки грантов, общественных, некоммерческих, коммерческих организаций и структур.
<b>Соответствие принципам этики:</b>	информированное согласие было получено от каждого пациента.
<b>Для цитирования:</b>	Соколов А.А., Варваренко В.И., Егунов О.А., Сморгон А.В. Форма и функция левого предсердия после хирургической и эндоваскулярной коррекции дефекта межпредсердной перегородки у детей. <i>Сибирский журнал клинической и экспериментальной медицины</i> . 2024;39(4):162–170. <a href="https://doi.org/10.29001/2073-8552-2024-39-4-162-170">https://doi.org/10.29001/2073-8552-2024-39-4-162-170</a> .

## Introduction

Atrial septal defects (ASD) are one of the most common congenital heart defects, accounting for about 10–15% of all congenital heart disease (CHD). Using the endovascular technology ASD closure is currently accepted as the treatment of choice in most patients with secundum ASD. It has shown good efficacy as well as lower complication rates compared to surgery. However, in some patients who have contraindications for using the endovascular method, surgical treatment is still used. [1].

ASD occluder devices, after fixation on the interatrial septum, close the shunt, by eliminating the interchamber communication. However, these devices increase the rigidity of the septum, limit its mobility, and presumably disrupt the mechanical function of the left atrium (LA). There are some data that LA shape correlates with atrial fibrillation (AF) and influences outcomes. Multiple markers of LA remodeling, including LA shape, were correlated with outcomes in atrial fibrillation (AF). Catheter ablation is an important treatment of AF, but better tools are needed to

determine which patients will benefit. This study evaluated particle-based modeling to quantitatively assess LA shape, and determine to what degree it predicts AF recurrence after catheter ablation [2].

The aim of this study was to assess the effect of surgical and endovascular correction on the size, shape and function of the left atrium and left ventricle in children

## Subjects and methods

### Subjects

We retrospectively studied consecutive echocardiograms from subjects who received endovascular and surgery ASD closure between 2006 and 2016 at the Cardiac Center in Tomsk Russia. 756 patients with ASD were examined, 564 of them before and after endovascular device closure and 192 after surgical correction. Follow-up duration was from 1 day to 10 years (yrs), mean 3.6 yrs for the device group and 4.2 yrs for the surgery group, 3393 healthy patients in the same age group, consisted of the controls. The control group was further stratified by age.

Table 1. Clinical and age characteristics in patient groups

Таблица 1. Клинические и возрастные характеристики групп пациентов

Follow-up / Контрольная точка	Total / Всего (n = 756)					
	Device / Группа пациентов, которым выполнялась эндоваскулярная коррекция ДМПП			Surgery / Группа пациентов, которым выполнялась хирургическая коррекция ДМПП		
	n	Age / Возраст		n	Age / Возраст	
		Mean	Median		Mean	Median
1–7 day / 1–7 дней	564	6.87	5.00	192	6.54	2.09
6 months / 6 мес.	154	6.91	6.00	117	5.05	2.50
1 year / 1 год	128	7.14	6.00	87	5.23	3.020
> 2 years (2.1–10 years)/ После 2 лет	175	8.87	8.00	192	9.11	7.00

Note: the group of surgical patients included patients with contraindications for endovascular correction, in most cases because of a small or deficient rim of the defect.

Примечание: в группу хирургических пациентов вошли пациенты с противопоказаниями к эндоваскулярной коррекции, в большинстве случаев из-за недостаточного края дефекта.

## Methods

Echocardiographic images were obtained using commercially available ultrasound equipment (iE33, Philips Ultrasound). Standard echo measures of chamber sizes and volumes were evaluated, including those indexed to body surface area and deviations of the studied parameters as a percentage of the predicted norm. Left ventricular filling pressure was calculated by the relation of E/e waves [3, 4]. Doppler echocardiographic evaluation of mitral inflow velocities and time duration was used in the assessment of left ventricular diastolic function. E/A ratio transmitral flow and mean septal and lateral mitral annular tissue doppler diastolic velocities and peaks duration was evaluated. The duration of diastole can be defined as one of the markers diastolic function [5]. A high systolic/diastolic ratio was associated with increased unfavorable events [6]. To reduce the effect of heart rate, diastole duration as a percentage of the duration of the R-R interval was used. Diastolic time expressed as percent of cardiac cycle (% D) calculated by the formula:  $D\% = ((R-R)-ET)/RR \times 100$ , where D% – diastole duration, ET – ejection time. Diastolic

interval measured from transmitral flow was named FD (flow diastole), another from used tissue Doppler-MD (mechanical diastole).

Left atrial shape index (LASI) was calculated as the ratio of half-sum anteroposterior and transverse size to vertical, . (Fig. 1).

An increase in the index close to 100 was regarded as an increase in sphericity, a decrease in ellipsoid LAVi-left atrial indexed volume and RAVi-right indexed atrial volume was calculated as volume-BSA relation. Atrial volumes were calculated by standard methods by using 4 and 2 chamber projections. Atrial index (Ai)- define as divided RA/LA volumes [7].

Continuous variables are presented as mean and standard deviation,  $M \pm SD$ . Categorical data are represented by frequencies and percentages. Clinical and procedural variables were compared with t-test for independent groups and paired t-test in the case of continuous data. Independent samples of categorical data were compared with Pearson  $\chi^2$ -test. Critical level of statistical significance was 0.05.



Fig. 1. Measurement of the size of the left atrium; H – height, w1 – anterior-posterior size, W2 – width

Рис. 1. Измерение размера левого предсердия; H – высота, w1 – переднезадний размер, W2 – ширина

## Results

Before correction, patients in the surgical group had statistically higher  $Q_p \setminus Q_s$  than the device group,  $2.97 \pm 0.47$  and  $2.10 \pm 0.64$ ,  $p = 0.01$ , the indexed volume of the right atrium in the surgical group was larger (Table 2). During follow-up in the device group, LASI was statistically significantly decreased, in surgery patients there was no statistically significant increase in LASI (Fig. 2).

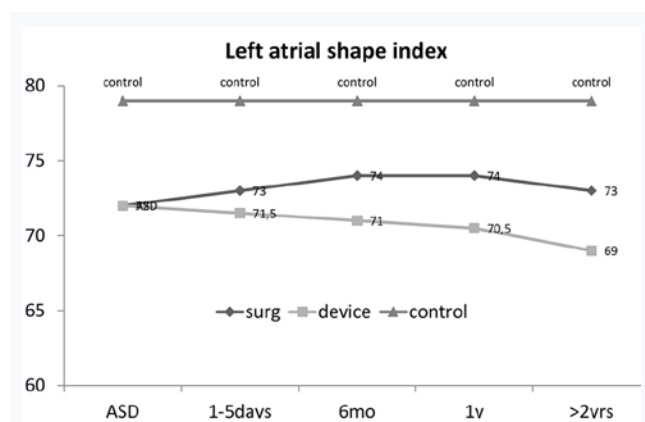


Fig. 2. The absolute values of the LASI during follow-up

Рис. 2. Абсолютные значения LASI в динамике

LASI in the control group was evaluated by a frequency table. LASI < 65 had 5% we defined it value as “cut of point” between normal and abnormal (Fig. 3). ASD patients had LASI less than 65 in 10.6% of all cases (Fig. 4). After closing the ASD (surgery and endovascular), percentages patients who had abnormal LASI (< 65) increased in both groups predominantly in the device group (Fig. 4).

LASI in patients before intervention and surgery was statistically different from the control group and was not difference in device and surgery groups. It was decreased from the first day to years after device implantation. But, there was no significant difference between surgery and control group at the late points, but the device group had more decreased LASI (Fig. 2)

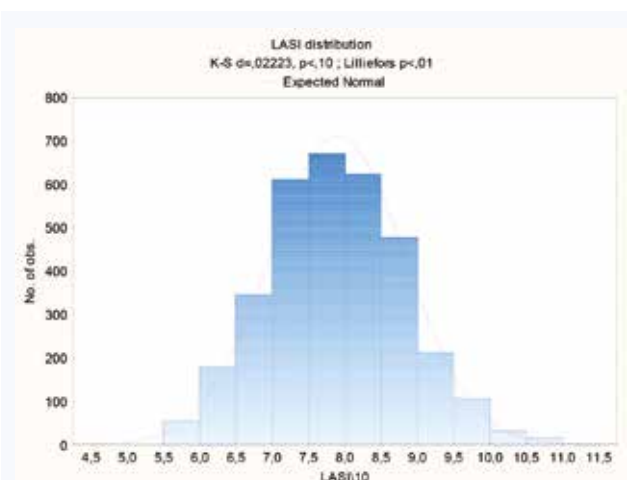


Fig. 3. LASI control group distribution

Рис. 3. Распределение контрольной группы по LASI

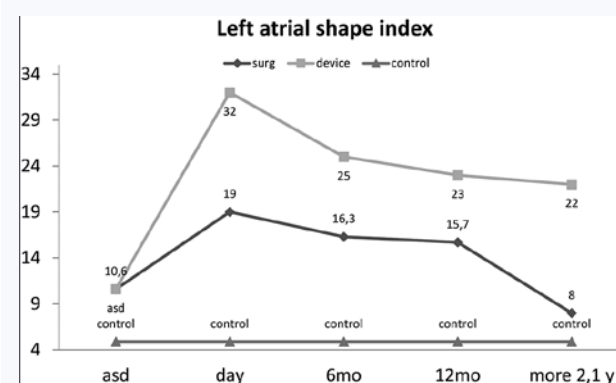


Fig. 4. Percentage of abnormal (less 65) LASI at follow up time

Рис. 4. Процент патологических (менее 65) LASI в динамике на контрольных точках наблюдения

The number of patients with an abnormal shape of the left atrium in the surgical group decreased during the observation period, in the device group – was stable from 6 month until long-term follow-up (Fig. 4). We could not

find any difference between surgery and device groups in respect to chambers volumes, their contractility, right ventricular systolic pressure at all follow up points. We then evaluated the possible significance of changing LASI

to chamber volumes and function. All device patients were divided into a “reshaping” group (RS) – LASI  $\leq 65$  any point after implantation, and a “saved” shape group that had LASI  $> 65$  (Table 4).

**Table 2.** Chamber volumes in ASD patients before and after surgery and device correction (1–5 days,  $M \pm SD$ )

**Таблица 2.** Объемы камер у пациентов с дефектом межпредсердной перегородки до и после хирургической и эндоваскулярной коррекции (1–5 дней,  $M \pm SD$ )

Parameter Показатели	Control Контроль-ная группа ( $n = 3000$ )	Surgery / Группы пациентов, которым выполнялась хирургическая коррекция ДМПП ( $n = 192$ )		Device / Группы пациентов, которым выполнялась эндоваскулярная коррекция ДМПП ( $n = 564$ )	
		Before operation До операции	After operation После операции	Before operation До операции	After operation После операции
EDVi, ml/m <sup>2</sup> КДИ	43.2 $\pm$ 7.7	44.2 $\pm$ 7.5	39.2 $\pm$ 7.0 <sup>^</sup>	43.3 $\pm$ 6.4	44.9 $\pm$ 7.2
LAI, ml/m <sup>2</sup> ИОЛП	17.4 $\pm$ 4.3	21.5 $\pm$ 6.1	19.1 $\pm$ 6.0	19.9 $\pm$ 6.1	18.4 $\pm$ 4.5 <sup>^</sup>
RAi, ml/m <sup>2</sup> ИОПП	19.3 $\pm$ 5.3	38.9 $\pm$ 6.1*	21.8 $\pm$ 8.2 <sup>^</sup>	30.8 $\pm$ 6.1*	21.9 $\pm$ 6.0 <sup>^</sup>
Ai	1.13 $\pm$ 0.25	1.68 $\pm$ 0.61*	1.16 $\pm$ 0.29 <sup>^</sup>	1.68 $\pm$ 0.62*	1.22 $\pm$ 0.33 <sup>^</sup>
EF, % ФВ	70.6 $\pm$ 5.1	69.1 $\pm$ 5.7	69.5 $\pm$ 6.2	69.2 $\pm$ 6.2	70.5 $\pm$ 5.4 <sup>^</sup>
RVSP, mm Hg СДПЖ	22.2 $\pm$ 2.7	26.2 $\pm$ 5.4*	22.4 $\pm$ 4.3 <sup>^</sup>	25.2 $\pm$ 4.9*	22.4 $\pm$ 3.1 <sup>^</sup>
DM, RR%	59.5 $\pm$ 5.0	58.6 $\pm$ 4.9	64.3 $\pm$ 4.2*	58.4 $\pm$ 1.6	59.2 $\pm$ 1.8
DF, RR%	53.0 $\pm$ 9.6	49.4 $\pm$ 6.9	50.2 $\pm$ 6.0	49.8 $\pm$ 7.8	45.9 $\pm$ 5.1 <sup>^</sup>
E/A	1.96 $\pm$ 0.51	1.79 $\pm$ 0.48	1.78 $\pm$ 0.046	1.81 $\pm$ 0.47	1.84 $\pm$ 0.49

Note: EDVi – EDVi – left ventricular indexed end diastolic volume, LAi – left atrial volume (indexed BSA), Rai – right atrial volume (indexed BSA), Ai – atrial volume relation index (Rai/LAi), EF – LV ejection fraction, RVSP – right ventricular systolic pressure. DM – mechanical diastole duration % R-R (by tissue Doppler), DF – flow diastole duration (by transmitral flow Doppler, % R-R), <sup>^</sup> – marked significance difference with preoperative values, \* – marked significance difference with control.

Примечание: КДИ – индексированный конечный диастолический объем левого желудочка, ИОЛП – объем левого предсердия (индексированный BSA), ИОПП – объем правого предсердия (индексированный BSA), Ai – индекс соотношения объема предсердий (Rai/LAi), ФВ – фракция выброса левого желудочка, СДПЖ – систолическое давление в правом желудочке. DM – продолжительность механической диастолы, % от нормы (по данным тканевой доплерографии), DF – продолжительность диастолы потока (по данным трансмитральной доплерографии, % от нормы) <sup>^</sup> – отмечена разница в значимости с дооперационными показателями, \* – отмечена разница в значимости с контролем.

**Table 3.** Chamber volumes in the device group with normal and abnormal LA shape ( $M \pm SD$ )

**Таблица 3.** Объемы камер в группе устройств с нормальной и патологической формой левого предсердия ( $M \pm SD$ )

Parameter Показатели	Control Контрольная группа ( $n = 3383$ )	Early follow-up Первая контрольная точка		Late follow up Последняя контрольная точка	
		Saved LASI Форма ЛП не изменена	Reshape Патологическая форма ЛП	Saved LASI Форма ЛП не изменена	Reshape Патологическая форма ЛП
EDVi, ml/m <sup>2</sup> КДИ	45.1 $\pm$ 5.9	45.2 $\pm$ 7.5	44.5 $\pm$ 6.8	48.7 $\pm$ 6.2	51.3 $\pm$ 9.2
LAVi, ml/m <sup>2</sup> ИОЛП	18.0 $\pm$ 5.2	17.7 $\pm$ 9.1	19.0 $\pm$ 4.1	20.1 $\pm$ 4.9	18.1 $\pm$ 5.5 <sup>^</sup>
RAVi, ml/m <sup>2</sup> ИОПП	20.1 $\pm$ 5.8	22 $\pm$ 10.1	21.6 $\pm$ 5.5	22 $\pm$ 5.5	19.2 $\pm$ 6.1
Ai	1.14 $\pm$ 0.21	1.27 $\pm$ 0.35	1.17 $\pm$ 0.31	1.16 $\pm$ 0.3	1.07 $\pm$ 0.3 <sup>^</sup>
EF, % ФВ	70.8 $\pm$ 5.3	70.6 $\pm$ 5.6	70.3 $\pm$ 5.1	69.6 $\pm$ 6.0	68.3 $\pm$ 5.2
RVSP, mm Hg СДПЖ	22.2 $\pm$ 2.8	22.7 $\pm$ 3.0	22.1 $\pm$ 2.9	22.4 $\pm$ 3.8	23.8 $\pm$ 3.6
Duration MD, RR% Продолжительность MD	56.4 $\pm$ 6.6	60.4 $\pm$ 6.1	64.7 $\pm$ 5.0*	59.1 $\pm$ 5.1	60.6 $\pm$ 5.1
Duration FD, RR% Продолжительность FD	54.5 $\pm$ 6.3	51.3 $\pm$ 9.1	55.8 $\pm$ 7.2	48.5 $\pm$ 5.5	51.4 $\pm$ 6.7
E/A	1.99 $\pm$ 0.52	1.82 $\pm$ 0.4	1.86 $\pm$ 0.6	1.89 $\pm$ 0.4	1.71 $\pm$ 0.4

Note: <sup>^</sup> – marked significance difference with preoperative values, \* – marked significance difference with control.

Примечание: \* – отмечена существенная разница с контролем, <sup>^</sup> – отмечена существенная разница с контролем.



**Table 4.** Chamber volumes in ASD patient before and after surgery and transcatheter correction (2–10 years,  $M \pm SD$ )**Таблица 4.** Объем камер у пациентов с дефектом межпредсердной перегородки до и после операции и эндоваскулярной коррекции (2–10 лет,  $M \pm SD$ )

Parameter Показатели	Control Контроль-ная группа ( $n = 2700$ )	Surgery / Группа пациентов, которым выполнялась хирургическая коррекция		Device / Группа пациентов, которым выполнялась эндоваскулярная коррекция	
		Before operation До операции	After operation После операции	Before operation До операции	After operation После операции
EDVi, ml/m <sup>2</sup> КДИ	46.7 $\pm$ 7.1	44.2 $\pm$ 7.5	49 $\pm$ 9.1 <sup>^</sup>	44.3 $\pm$ 6.4	49.3 $\pm$ 7.2 <sup>^</sup>
LAVi, ml/m <sup>2</sup> ИОЛП	18.3 $\pm$ 4.5	21.5 $\pm$ 6.1	20.5 $\pm$ 7.0	19.9 $\pm$ 6.1	18.4 $\pm$ 4.5
RAVi, ml/m <sup>2</sup> ИОПП	20.7 $\pm$ 5.1	38.9 $\pm$ 6.1*	20.9 $\pm$ 8.4 <sup>^</sup>	30.8 $\pm$ 6.1*	21.9 $\pm$ 6.0 <sup>^</sup>
Ai	1.15 $\pm$ 0.2	1.68 $\pm$ 0.61* <sup>^</sup>	1.04 $\pm$ 0.3	1.68 $\pm$ 0.62*	1.22 $\pm$ 0.33 <sup>^</sup>
EF, % ФВ	70.1 $\pm$ 4.9	69.1 $\pm$ 5.7	70.7 $\pm$ 5.0	69.2 $\pm$ 6.2	70.5 $\pm$ 5.4 <sup>^</sup>
RVSP, mm Hg СДПЖ	22.2 $\pm$ 2.9	26.2 $\pm$ 5.4*	23.5 $\pm$ 3.0 <sup>^</sup>	25.2 $\pm$ 4.9*	22.4 $\pm$ 3.1 <sup>^</sup>
DM, RR%	60.01 $\pm$ 5.3	58.6 $\pm$ 4.9	64.7 $\pm$ 5.0 <sup>^</sup>	58.4 $\pm$ 1.6	60.6 $\pm$ 5.1
DF, RR%	54.3 $\pm$ 7.2	49.4 $\pm$ 6.9	55.8 $\pm$ 7.2 <sup>^</sup>	49.8 $\pm$ 7.8	51.4 $\pm$ 6.7
E/A	2.0 $\pm$ 0.5	1.79 $\pm$ 0.48	2.16 $\pm$ 0.63 <sup>^</sup>	1.81 $\pm$ 0.47	1.92 $\pm$ 0.5

Note: EDVi – left ventricular indexed end diastolic volume, Lai – left atrial volume (indexed BSA), Rai – right atrial volume (indexed BSA), Ai – atrial volume relation index (Rai/Lai), EF – LV ejection fraction, RVSP – right ventricular systolic pressure. DM – mechanical diastole duration % R-R (by tissue Doppler) DF – flow diastole duration (by transmitral flow Doppler, % R-R), <sup>^</sup> – marked significance difference with preoperative values, \* – marked significance difference with control.

Примечание: КДИ – индексированный конечный диастолический объем левого желудочка, ИОЛП – объем левого предсердия (индексированный BSA), ИОПП – объем правого предсердия (индексированный BSA), Ai – индекс соотношения объема предсердий (Rai/Lai), ФВ – фракция выброса левого желудочка, СДПЖ – систолическое давление в правом желудочке. DM – продолжительность механической диастолы, % R-R (по данным тканевой доплерографии) DF – продолжительность диастолы потока (по данным трансмитральной доплерографии, % R-R), <sup>^</sup> – выраженная разница в значимости с дооперационными значениями, \* – выраженная разница в значимости с контролем.

## LA and RA Volumes

At 1–5 days after ASD closure there were statistically significant increasing LV volume and decreased right chambers in both groups.

Left ventricular volume in short-term after ASD closure were not changed in both groups, but left atrial volume statistically significantly decreased in the device group. There were decreasing right ventricular systolic pressure, and right atrial volume with unchanging left ventricular ejection fraction. We found that in 32% of patients in the device group who had LA reshaping that their LA volume reduction was more evident ( $LAi = 17.0 \pm 4.4$  ml/m<sup>2</sup>).

LA reshape and volume reduction was combined changing the left atrial force (LAEF) determined by Mannig method [8, 9]. The LAEF in the device group with LA reshape was maximal after few days (1–5days, mean  $3.4 \pm 1.3$  and years (2–10 mean  $3.6 \pm 3.5$  years (Fig. 3).

Other Echo-parameters in these patients were not different with the surgery group and patients who had “saved” left atrial shape. Only mechanical diastole in reshape subgroup early follow-up was longer with control (Table 3).

In the early postoperative time (1–5 days) after the closure of the defects in patients with surgical and endovascular groups, the volume of the right atrium significantly decreased, the atrial index (see Table 2). At the same time, the diastolic indexed volume of the left ventricle in the endovascular group-increased, while in the surgical group it decreased in comparison with the preoperative period. The RV systolic pressure in both groups decreased significantly, while the contractility of left ventricle remained unchanged. The patients in device group with a registered violation of the LASI at the time of implantation of the device was significantly younger

than patients with a preserved LASI  $2.06 \pm 0.47$  and  $2.98 \pm 0.64$  yrs,  $p = 0.02$ . It could be the hypothetic reason abnormal left atrial shape in early follow-up.

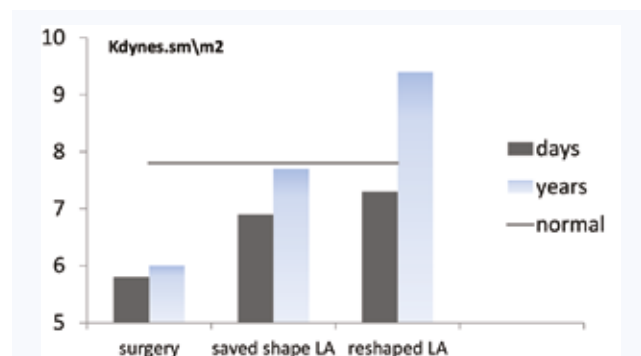


Fig. 5. Left atrial ejection force in ASD patients after surgery and device closing

Рис. 5. Сила выброса левого предсердия у пациентов с дефектом межпредсердной перегородки после операции и эндоваскулярной коррекции в динамике относительно здоровых пациентов

## Long-term postoperative period:

The groups of surgical and endovascular correction were compared in the long-term from 2.1 to 10 years. The age of patients in the groups was the same  $9.58 \pm 6.9$  (surgery) and  $8.96 \pm 4.5$  years (device). Mean follow up time in the surgery group was 4.3 years, in device group – 3.6yrs.

The left atrial shape index was lower in the device group  $74.0 \pm 0.3$  and  $70.0 \pm 2$  (surgery). A LASI < 65 (significantly different from the norm) in the device group was found in 22.5%, of the device group, 8.0% in the surgical group and

16.6%, in the complete study cohort. The indexed diastolic volumes of LV in the groups did not differ (surgery  $49.9 \pm 10.8$  and devices  $49.3 \pm 9.6$  ml / m<sup>2</sup>,  $p = 0.61$ ). Contractility was the same EF:  $70.7 \pm 5$  and  $70.5 \pm 5$ ,  $p = 0.99$  (Table 4).

The LAEF (left atrial contraction force) was statistically significantly lower in the surgery group than in the endovascular correction group (Fig. 6). In turn, the contraction force of the left atrium in the endovascular group did not differ with the control group.

It should be noted that in patients that lost form of the left atrium after correction (34.4% of total), the strength of

the contraction of the left atrium was higher than the group average and amounted to  $9.51 \pm 4.7$  kdynes \ cm \ m<sup>2</sup>, while in those with impaired left form atrial after surgery, the strength of the contraction of the LA was not significantly different from the entire surgery group ( $6.5 \pm 4.4$  kdynes \ cm \ m<sup>2</sup>) and the control group  $6.6 \pm 4.8$  kdynes / cm V m<sup>2</sup>.

It can be assumed that an increase in the force of LA contraction causing LV filling, may adversely affect further. Some researchers find an increase in left atrial contraction force as one of the mechanisms of left ventricular hypertrophy in hypertension [10, 11].

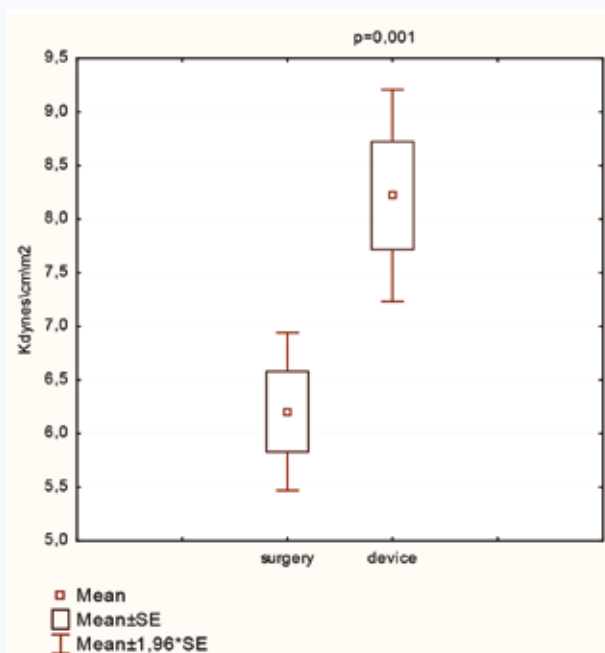


Fig. 6. Left atrial force in surgery and device groups at long-term follow-up

Рис. 6. Сила левого предсердия в группах с хирургической и эндоваскулярной коррекцией при длительном наблюдении

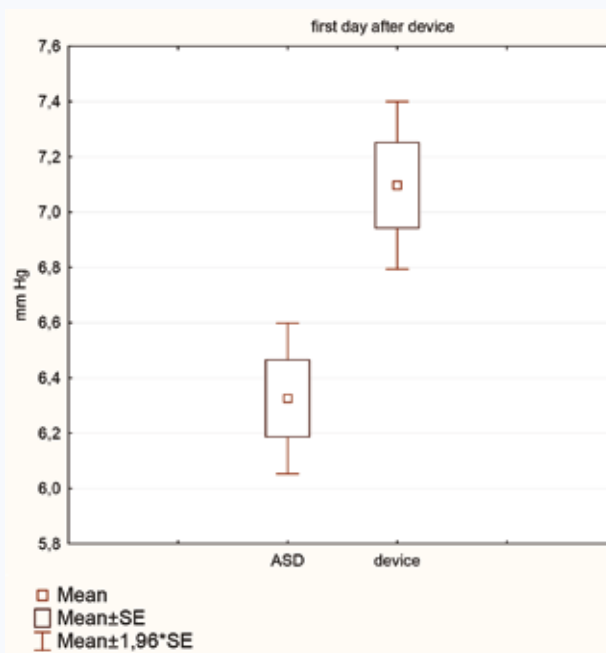


Fig. 7. LV filling pressure in device group at early follow-up

Рис. 7. Давление наполнения левого желудочка в группе с эндоваскулярной коррекцией при раннем наблюдении

It has been shown that a change in the shape of the left atrium in patients with paroxysmal arrhythmias is associated with frequent relapses after treatment [12].

The possibly unfavorable value of the change in the shape of the left atrium may be indicated by the tendency to increase the filling pressure of the left ventricle ( $5.87 \pm 1.4$ -control and  $6.5 \pm 1.5$  mm Hg) in the device group for 1-5 days. The pressure of LV filling remaining within normal values after endovascular closure of the defects significantly increased within 1–5 days after the procedure (Fig. 7.). In patients with post-endovascular correction, a small negative, but statistically significant correlation between the LASI and filling pressure was recorded,  $r = -0.39$ ,  $p = 0.001$ .

It can be assumed that the first mechanisms of remodeling of the left atrium, in particular, changes in its shape and force of contraction will turn out to be unfavorable factors in the future. Similar changes were found in adult patients with interatrial adiposity. These remodeling processes were associated with increased LA global kinetic energy and ejection force at the hemodynamic level, which may serve to compensate for those functional deteriorations of LA in asymptomatic population [13].

It is hoped that these changes are not significant for the lives of our young patients. In the long term after surgical and endovascular treatment of ASD, the main hemodynamic parameters and indexed chamber volumes did not abnormal (Table 4).

## Discussion

The main attention in the manuscript is paid to the results of two methods of treatment of atrial septal defects (ASD) in children — endovascular and surgical correction — and their effect on the shape, size and function of the left atrium (LA). ASD are common congenital heart defects, and understanding the effects of treatment on the structure and function of the heart is crucial to optimize treatment strategies and improve the results of correction of the defect in patients.

The study showed that both endovascular and surgical methods are effective in correcting ASD and lead to favorable results with respect to left atrial function. However, there is still uncertainty about the long-term effects after implanted devices used in endovascular procedures. Although these devices offer a minimally invasive solution, there is a problem associated with increased stiffness and limited mobility of

the partition. Such changes can potentially alter the normal dynamics of atrial filling and contraction, affecting the overall function of the left atrium over time.

The retrospective nature of the study allows for a valuable comparison between surgical and endovascular interventions. Since the average follow-up period in different groups was 3.6 years in the endovascular group and 4.2 years in the surgical group, the results indicate that although both correction methods give satisfactory short- and medium-term results in LA remodeling, further studies are needed to assess potential long-term consequences, especially when using endovascular methods.

The results of the study confirm the importance of an individual approach to the treatment of pediatric patients with ASD, taking into account the possible trade-offs between immediate recovery and the long-term state of the cardiovascular system. Understanding how each intervention affects the structure of the heart over time can serve as a basis for clinical decision-making. The results obtained emphasize the need for clinicians to conduct echocardiographic monitoring not only directly in the postoperative period, which allows timely detection of any mechanical complications or changes in the work of the heart and take measures to eliminate them.

Retrospective analysis has its limitations, including possible errors in the selection of patients and differences

in surgical technique that may affect the results. In addition, the presence of factors such as pre-existing heart function or the age of patients at the time of intervention requires careful interpretation of the results. Future prospective studies with a large sample size and controlled variables will increase the generalizability of the results and provide more reliable data on the long-term results of both surgical and endovascular treatment [14].

## Conclusion

Endovascular correction of ASD in 35% of children was accompanied by a change in the shape of the left atrium - a decrease in sphericity and an increase in ellipsoidity. Changes in the shape of the left atrium persisted in 22% after transcatheter correction in the long term. Changes in the shape of the left atrium at early follow-up were more often observed in the device closure group in children of a younger age. The change in the shape of the left atrium after the placement of devices was accompanied by activation of the mechanical function of the atrium and an increase in the filling pressure of the left ventricle. These changes were not accompanied by any disturbance in the contractility and volume of the heart chambers. In the group of children after surgical correction of ASD, the contractility and volume of the heart chambers did not statistically significantly differ from those in the device group.

## Reference / Литература

- Jung S.Y., Choi J.Y. Transcatheter closure of atrial septal defect: principles and available devices. *J. Thorac. Dis.* 2018;10(Suppl\_24):S2909–S2922. DOI: 10.21037/jtd.2018.02.19.
- Bisbal F., Guiu E., Cabanas P., Calvo N., Berrueto A., Tolosana J.M. Reversal of spherical remodeling of the left atrium after pulmonary vein isolation: incidence and predictors. *Europace.* 2014;16(6):840–847. DOI: 10.1093/europace/eut385.
- Nagueh S.F. Non-invasive assessment of left ventricular filling pressure. *Eur. J. Heart Fail.* 2018;20(1):38–48. DOI: 10.1002/ejhf.971.
- Andersen O.S., Smiseth O.A., Dokainish H., Abudiyab M.M., Schutt R.C., Kumar A. et al. Estimating left ventricular filling pressure by echocardiography. *J. Am. Coll. Cardiol.* 2017;69(15):1937–1948. DOI: 10.1016/j.jacc.2017.01.058.
- Chung C.S., Karamanoglu M., Kovács S.J. Duration of diastole and its phases as a function of heart rate during supine bicycle exercise. *Am. J. Physiol. Heart Circ. Physiol.* 2004;287(5):H2003–H2008. DOI: 10.1152/ajpheart.00404.2004.
- Mondal T., Slorach C., Manliot C., Hui W., Kantor P.F., McCrindle B.W. et al. Prognostic implications of the systolic to diastolic duration ratio in children with idiopathic or familial dilated cardiomyopathy. *Circ. Cardiovasc. Imaging.* 2014;7(5):773–780. DOI: 10.1161/CIRCIMAGING.114.002120.
- Pritchett A.M., Jacobsen S.J., Mahoney D.W., Rodeheffer R.J., Bailey K.R., Redfield M.M. Left atrial volume as an index of left atrial size: a population-based study. *J. Am. Coll. Cardiol.* 2003;41(6):1036–1043. DOI: 10.1016/s0735-1097(02)02981-9.
- Triposkiadis F., Harbas C., Sitafidis G., Skoularigis J., Demopoulos V., Kelepeshis G. Echocardiographic assessment of left atrial ejection force and kinetic energy in chronic heart failure. *Int. J. Cardiovasc. Imaging.* 2008;24(1):15–22. DOI: 10.1007/s10554-007-9219-7.
- Manning W.J., Silverman D.I., Katz S.E., Douglas P.S. Atrial ejection force: a noninvasive assessment of atrial systolic function. *J. Am. Coll. Cardiol.* 1993;22(1):221–225. DOI: 10.1016/0735-1097(93)90838-r.
- Chinali M., de Simone G., Liu J.E. et al. Left atrial systolic force and cardiac markers of preclinical disease in hypertensive patients: the Hypertension Genetic Epidemiology Network (HyperGEN) Study. *Am. J. Hypertens.* 2005;18(7):899–905. DOI: 10.1016/j.amjhyper.2005.01.005.
- Mazzzone C., Cioffi G., Faganello G., Faggiano P., Candido R., Cherubini A. et al. Analysis of left atrial performance in patients with type 2 diabetes mellitus without overt cardiac disease and inducible ischemia: high prevalence of increased systolic force related to enhanced left ventricular systolic longitudinal function. *Echocardiography.* 2015;32(2):221–228. DOI: 10.1111/echo.12639.
- Bisbal F., Guiu E., Calvo N., Marin D., Berrueto A., Arbelo E. et al. Left atrial sphericity: A new method to assess atrial remodeling. Impact on the outcome of atrial fibrillation ablation. *J. Cardiovasc. Electrophysiol.* 2013;24(7):752–759. DOI: 10.1111/jce.12116.
- Lai Y.H., Yun C.H., Su C.H., Yang F.S., Yeh H.I., Hou C.J. et al. Excessive interatrial adiposity is associated with left atrial remodeling, augmented contractile performance in asymptomatic population. *Echo. Res. Pract.* 2016;3(1):5–15. DOI: 10.1530/ERP-15-0031.
- Соколов А.А., Егунов О.А., Сморгон А.В., Кожанов Р.С. Оценка интракардиальной гемодинамики у детей до 1 года с дефектом межпредсердной перегородки. *Медицинская визуализация.* 2024;28(3):99–105. DOI: 10.24835/1607-0763-1448.

Sokolov A.A., Egunov O.A., Smorgon A.V., Kozhanov R.S. Evaluation of intracardial hemodynamics in children under 1 year of age with atrial septal defect. *Medical Visualization.* 2024;28(3):99–105. (In Russ.). DOI: 10.24835/1607-0763-1448.

## Информация о вкладе авторов

Соколов А.А., Сморгон А.В. – проведение пациентам эхокардиографического исследования, формирование выборки, написание первой версии рукописи.

Варваренко В.И., Егунов О.А. – хирургическая и эндоваскулярная коррекция дефекта межпредсердной перегородки, доработка рукописи.

Все авторы дали окончательное согласие на подачу рукописи и согласились нести ответственность за все аспекты работы, ручаясь за их точность и безупречность.

## Information on author contributions

Sokolov A.A., Smorgon A.V. – conducting echocardiography on patients, sampling, writing the first version of the manuscript

Varvarenko V.I., Egunov O.A. – surgical and endovascular ASD correction, manuscript revision.

All authors gave their final consent to the submission of the manuscript and agreed to be responsible for all aspects of the work, vouching for their accuracy and flawlessness.



**Конфликт интересов:** конфликт интересов отсутствует.

**Conflict of interest:** there is no conflict of interest.

## Сведения об авторах

**Соколов Александр Анатольевич**, д-р мед. наук, профессор, заведующий лабораторией ультразвуковых и функциональных методов исследования, НИИ кардиологии Томского НИМЦ, Томск, <https://orcid.org/0000-0003-0513-9012>.

E-mail: [asa@cardio-tomsk.ru](mailto:asa@cardio-tomsk.ru).

**Варваренко Виктор Иванович**, канд. мед. наук, врач по рентгенэндоваскулярным диагностике и лечению, отделение рентгенохирургических методов диагностики и лечения, НИИ кардиологии Томского НИМЦ, Томск, <https://orcid.org/0000-0003-0513-9015>.

E-mail: [vvi@cardio-tomsk.ru](mailto:vvi@cardio-tomsk.ru).

**Егунов Олег Анатольевич**, канд. мед. наук, врач сердечно-сосудистый хирург, кардиохирургическое отделение № 2, НИИ кардиологии Томского НИМЦ, Томск, <https://orcid.org/0000-0003-4023-455X>.

E-mail: [eo@cardio-tomsk.ru](mailto:eo@cardio-tomsk.ru).

**Сморгон Андрей Владимирович**, младший научный сотрудник, лаборатория ультразвуковых и функциональных методов исследования, НИИ кардиологии Томского НИМЦ, Томск, <https://orcid.org/0000-0002-6531-7223>.

E-mail: [sav@cardio-tomsk.ru](mailto:sav@cardio-tomsk.ru).

Поступила 29.08.2024;  
рецензия получена 28.11.2024;  
принята к публикации 02.12.2024.

## Information about the authors

**Alexander A. Sokolov**, Dr. Sci. (Med.), Professor, Head of the Department of Functional and Laboratory Diagnostics, Cardiology Research Institute, Tomsk NRMC, Tomsk, Russia, <https://orcid.org/0000-0003-0513-9012>.

E-mail: [asa@cardio-tomsk.ru](mailto:asa@cardio-tomsk.ru).

**Viktor I. Varvarenko** Cand. Sci. (Med.), Interventional Cardiologist Cardiology Research Institute, Cardiology Research Institute, Tomsk NRMC, Tomsk, Russia, <https://orcid.org/0000-0003-0513-9015>.

E-mail: [vvi@cardio-tomsk.ru](mailto:vvi@cardio-tomsk.ru).

**Oleg A. Egunov** Cand. Sci. (Med.), Cardiac Surgeon, Cardiology Research Institute, Tomsk NRMC, Tomsk, Russia, <https://orcid.org/0000-0003-4023-455X>.

E-mail: [eo@cardio-tomsk.ru](mailto:eo@cardio-tomsk.ru).

**Andrey V. Smorgon**, Junior Research Scientist, Laboratory of Ultrasound and Functional Research Methods, Cardiology Research Institute, Tomsk NRMC, Tomsk, Russia, <https://orcid.org/0000-0002-6531-7223>.

E-mail: [sav@cardio-tomsk.ru](mailto:sav@cardio-tomsk.ru).

Received 29.08.2024;  
review received 28.11.2024;  
accepted for publication 02.12.2024.