

# Closure of perimembranous ventricular septal defect using transcatheter technique versus surgical repair

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## Abstract

**Introduction:** Since the Membr VSD occluders have been introduced into clinical practice it is now possible to compare two treatment methods – surgical and percutaneous.

**Aim:** Assessment of the effectiveness and risk of complications in patients with perimembranous ventricular septal defect (VSD) treated with the Membr VSD occluder or surgery as well as comparison of postprocedural left and right ventricular systolic functions.

**Methods:** The study involved 11 children with perimembranous VSD treated with occluder implantation (Group A) and 12 children with surgical repair (Group C). Groups A and C differed slightly in terms of age of patients ( $p=0.026$ ), but had similar mean weights ( $p=0.103$ ), pulmonary to systemic flow ratios ( $Q_p: Q_s$ ,  $p=0.929$ ) and follow-up duration after the procedure. No significant differences were observed between the two groups in terms of left ventricular systolic function [left ventricular ejection fraction (EF,  $p=0.567$ )], diastolic function [mitral flow E/A ratio ( $p=0.975$ )], E deceleration time (DCT,  $p=0.346$ ), isovolumetric relaxation time (IVRT,  $p=0.606$ ), heart rate (HR,  $p=0.133$ ) or left ventricular diameter (LV) on transthoracic echocardiography (TTE) before VSD closure. TEE was performed in all patients and the parameters listed above were measured within 3 to 16 months after the procedure, and then intra- and inter-group comparisons were carried out. Additionally, mitral (MV), tricuspid (TV) and aortic (Ao) valve regurgitations were evaluated.

**Results:** Tricuspid (TR,  $p=0.028$ ) and mitral regurgitation (MR,  $p=0.043$ ) decreased significantly after closure of VSD with the Membr VSDO. MR was significantly smaller in patients treated with the occluder than in those after surgery ( $p=0.026$ ). Postprocedural TR ( $p=0.486$ ) and aortic regurgitation (AR, 0.607) did not differ significantly between the two groups. Left ventricular EF was significantly ( $p=0.004$ ) lower and HR higher (0.043) after surgery than in children treated with the occluder. No significant differences of the diastolic function were found – E/A ( $p=0.88$ ), DCT ( $p=0.413$ ), IVRT ( $p=0.09$ ).

**Conclusions:** 1. During the mid-term follow-up left ventricular EF was higher in patients after Membr VSDO occluder implantation than after surgical repair of VSD. 2. Higher EF and reduction of the left ventricular diameter after closure of perimembranous VSD with the occluder may result in a lower incidence and degree of MR in comparison to the subjects operated on.

**Key words:** Amplatzer Membr VSDO, perimembranous ventricular septal defect

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## Introduction

For over 40 years surgical complications have inspired investigations aimed at the development of non-surgical techniques of congenital heart disease treatment, including ventricular septal defects (VSD). Cardiac surgery has been reported to be followed by postcardiotomy syndrome, adhesions of the pericardium

and the cardiac muscle, residual shunts, early- and late-onset arrhythmias, and deaths in single cases [1-5].

Animal studies have shown that postsurgical lesions within myocardial surroundings result in decreased right ventricular systolic function [6]. The influence of adhesions on systolic and diastolic performance of the left ventricle remains unknown.

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VSD were initially closed with occluders similar to those used for atrial septal defect management. In the long-term follow-up residual shunts, damage to the aortic and atrio-ventricular valves were reported [7-9]. The modified MuscVSDO Amplatzer Membranous VSD Occluder (Membr VSDO) has permitted closure of perimembranous defects since the first use in 1999 [10-13].

As non-surgical methods of VSD closure became available in clinical practice, studies comparing the results of the treatment using such devices with surgical VSD closure were started. We are not aware of any published data on the systolic and diastolic function of the left ventricle in children after VSD closure with the Amplatzer occluder.

Left ventricular systolic and diastolic dysfunction in patients with congenital heart disease before and after surgery is a common problem [14-16]. It depends on many factors: relaxation, contractility, compliance and filling of the atria and ventricles [17-23]. The major parameter used for the evaluation of left ventricular systolic function on transthoracic echocardiography (TTE) is the ejection fraction (EF), and, for diastolic function, the Doppler velocity spectrum of blood flow from the left atrium into the left ventricle. Among a number of parameters describing the inflow curve the most important for the evaluation of diastolic function are isovolumetric relaxation time (IVRT), mitral E and A velocities, E deceleration time (DCT), and E/A ratio [22].

### Aim of the study

1. To evaluate the effectiveness of treatment and early- and mid-onset risk of complications after VSD closure with Membr VSDO or surgery.
2. To determine whether systolic and diastolic function of the left ventricle differ with regard to the perimembranous VSD closure method used.

### Methods

#### Patients

From 16.06.2003 to June 2004, perimembranous VSD was closed using the Amplatzer Membr VSDO (Membr VSDO) in 11 children (Group A), and from January 2003 to January 2004 surgical closure was performed in 12 children (Group C).

Children over 5 kg body weight were selected for VSD closure with the occluder based on TTE performed using the Hewlett-Packard Image-Point system. The final qualifying decisions were guided by transoesophageal echocardiography (TEE) performed during cardiac catheterization.

All children referred to the VSD closure procedure had a haemodynamically significant increased left

ventricular end-diastolic diameter (LVEDD), and pulmonary to systemic flow ratio ( $Q_p: Q_s$ )  $>1.5$  as assessed by TTE.

Children treated surgically had similar weight at the time of the operation and similar postprocedural follow-up to children treated with the Membr VSDO.

The characteristics of the two groups are shown in Table I. Children operated on were a little younger; however, they did not differ with respect to body weight, defect size or VSD flow rate. The technique of perimembranous VSD closure using the Membr VSDO was detailed in our previous paper [24].

### Echocardiographic assessment

Systolic and diastolic function was evaluated with echocardiography before and after closure of the defect in long-term follow-up; a Hewlett-Packard system with a 3.5-2.5 MHz probe was used and standard views recorded (Table I). The mean value from five measurements was used for the analysis of each parameter. LVEDD, right ventricular end-diastolic diameter (RVEDD), and ejection fraction (EF) were measured from the M-mode parasternal long axis view, with measurement lines placed just behind the mitral valve leaflets. Mitral flow was measured from the apical four-chamber view after placing the Doppler gate between the mitral valve leaflets at the level of the valvular annulus. The following parameters were measured: E and A flow velocities, isovolumetric relaxation time (IVRT), deceleration time (DCT) and E to A velocity ratio. ECG was monitored during echocardiography. Children from both groups were examined in the same conditions.

LVEDD and RVEDD were expressed as the percentage of the mean weight-matched reference values [18]. The E/A ratio was calculated as the mitral valve inflow E- to A-wave flow velocity quotient.

The analysis involved a comparison of the values measured before and after closure of the defects as well as between the groups of children treated with the Membr VSD occluder and surgery. The study investigated the efficacy of both methods, the rate of complications, residual shunts, and prevalence of arrhythmias, based on 12-lead ECG.

### Statistical analysis

The normal distribution of variables was tested using the Shapiro-Wilk test; significant outcomes were analysed with the applicable nonparametric tests. Variables following the normal distribution were expressed as a median and a range. The Mann-Whitney U test was used for independent variables and the

**Table I.** Preprocedural characteristics of children with perimembranous VSD treated with the MembrVSDO (Group A) or surgery (Group C) and comparisons between the groups

Variables	Group A, n=11			Group C, n=12			P value
	Median	Range	Mean±SD	Median	Range	Mean±SD	
Age [years]	6.5	0.8-18	7.78±6.4	1.5	0.6-7	2.58±2.26	0.026*
Weight [kg]	23	9.4-72	28.12±21.25	17.5	6.8-34	16.68±8.44	0.103
Height [cm]	122	69-172	118.22±32.91	99.5	67-143	99.9±25.35	0.133
LVEDD	1.12	1-1.32	1.13±0.077	1.23	0.99-1.5	1.23±0.15	0.090
Qp: Qs	1.56	1.5-2.3	1.71±0.28	1.65	1.2-2.5	1.74±0.39	0.927
ØVSD/vAo	0.36	0.27-0.58	0.389±0.10	0.3	0.2-0.55	0.32±0.09	0.133
PG VSD [mmHg]	78	56-95	75.9±10.6	67.1	59.9-84	69.67±7.32	0.079
Follow-up duration [months]	10	3-15	10.36±4.34	11.5	7-16	11.5±2.77	0.486

LVEDD/mean – left ventricular end-diastolic diameter on TTE to mean reference body weight ratio; Qp: Qs – pulmonary to systemic flow ratio; ØVSD TTE/ vAo – defect size to aortic valve diameter ratio; PG VSD – VSD pressure gradient. VSD – ventricular septal defect

Wilcoxon test for dependent variables. The chi square test (independent data) and the McNemara test (dependent data) were used for dichotomous variables. The results were found significant when the p value was less than 0.05.

## Results

Closure of VSD with the Membr VSDO was successfully performed in all patients. Sizes of defects and occluders are listed in Table II.

In two children the totally decompressed occluder was withdrawn to the long sheath and after changing its position and angiographic view; both discs were correctly decompressed over the intraventricular septum under TEE guidance. In the first child the incorrect occluder placement was suggested by disc ballottement, which indicated decompression of both discs in the left ventricle; in the other child, the angiography provided the proof.

In one child, a residual shunt of about 1 mm diameter originating from one of three intraventricular septum aneurysm openings was observed.

Closure of the VSD with the Membr VSDO was found to be associated with tricuspid regurgitation

magnitude reduction in 9 children. In only a single case of the youngest child did the tricuspid regurgitation increase from first to second degree. In this child the 4.5 mm defect was closed with the 6mm occluder. The differences between the number of patients with TR and degree of TR before and after occluder insertion were statistically significant ( $p=0.028$ ). None of the children was observed to develop mitral regurgitation, unless present before the procedure. The occurrence of MR decreased significantly after occluder placement ( $p=0.043$ ). Before the procedure, first-degree aortic regurgitation was observed in three children, whereas it was found in two more children after the procedure; however, these differences were not significant ( $p=0.592$ , Table III).

None of the children treated with the Membr VSDO presented new significant arrhythmias such as complete heart block, left bundle branch block, or bradycardia during or after occluder implantation. Complete left bundle branch block (LBBB) was seen in two children, and incomplete LBBB was noted also in two children before and after VSD closure. Right bundle branch block (RBBB), which was not observed at baseline, occurred post procedure in two children in Group A (transient

**Table II.** Defect diameter measured with TTE, TEE and angiography and Membr VSDOs sizes

ØVSD	Group A, n=11			Group C, n=12		
	Median	Range	Mean±SD	Median	Range	Mean±SD.
TTE	6.0	5.0-9.0	6.22±1.09	5.0	3.5-6.0	4.79±0.94
TEE	5.7	4.0-11.0	6.17±2.39			
Angio	5.7	4.0-10.0	5.91±2.22			
MVSDO	6.0	4.0-10.0	6.36±2.15			

**Abbreviations:** A – group treated with Membr VSDO; C – group treated with surgery; ØVSD – defect diameter, MVSDO – occluder diameter, TTE – transthoracic echocardiography, TEE – transoesophageal echocardiography, Angio – angiography

**Table III.** Valve regurgitations in children with VSD prior to and after VSD closure with the Membr VSDO (Group A) and with surgery (Group C)

Valve		Group A, n-11		Prior vs afterA	Group C, n-12		Prior vs afterC	A vs C prior	A vs C after
		Prior	After	p	Prior	After	p	p	
TR	Median	2	1	0.028*	1.5	1	0.109	0.880	0.486
	Range	0-3	0-2		0-3	1-1.5			
	Mean±SD	1.59±0.97	0.95±0.56		1.62±0.93	1.25±0.22			
AR	Median	0		0.593	0	1	0.067	0.651	0.607
	Range	0-1	0-1		0-1	0-1			
	Mean±SD	0.36±0.50	0.45±0.52		0.25±0.45	0.58±0.51			
MR	Median	1		0.043*	1	1	1.0	0.786	0.026*
	Range	0-1.5	0-1		0-1	1-1			
	Mean±SD	0.95±0.35	0.45±0.52		0.91±0.28	1.0±0.00			

**Abbreviations:** TR – tricuspid regurgitation, AR – aortic regurgitation, MR – mitral regurgitation

complete RBBB in one child and incomplete RBBB in another one). One boy had a wandering supraventricular pacemaker before and after the closure of VSD. Differences in incidence of arrhythmia and conduction disturbances before and after VSD closure did not reach statistical significance ( $p=0.723$ ).

Complete RBBB after surgery was found in four children, and incomplete RBBB in one. In one child, incomplete RBBB at baseline turned into a complete block after surgery. There was no difference in the occurrence of arrhythmias before or after surgical closure of VSD. Residual VSD in the inferior aspect of the patch was found in one child after surgery.

Second- and third-degree tricuspid regurgitations reported before surgery were reduced to first degree in five children, and to first/second degree in one subject. However, in two children an increase from first to second degree was observed. These differences were not significant. First-degree aortic regurgitation was diagnosed in three children before surgery and, additionally, occurred in four in the postsurgical period; however, this difference was not significant ( $p=0.067$ ). First-degree mitral regurgitation before surgery was seen in 11 children and developed in another child after surgery.

Mitral regurgitation was significantly less severe in patients treated with the Membr VSDO than after surgical closure; the difference in tricuspid and aortic regurgitations did not reach statistical significance. The comparison of valvular regurgitations in pre- and post-treatment periods between the two groups, treated with the occluder or surgery, is shown in Table III.

The comparison of echocardiographic parameters of left ventricular systolic and diastolic function, and left

ventricular diameter prior to and after VSD closure is detailed in Table IV. Left ventricular diameter and heart rate decreased significantly after VSD closure with the Membr VSDO. The ejection fraction was significantly higher following VSD closure. Systolic function remained unchanged after occluder implantation. No significant difference was found with respect to DCT and IVRT before or after VSD closure with the Membr VSDO.

Surgical treatment resulted in a significantly lower left ventricular diameter and heart rate. The echocardiographic parameters of left ventricular systolic and diastolic function did not differ between the pre- and post-operative periods.

The comparison of left ventricular systolic and diastolic function and left ventricular diameter between groups A and C prior to and after the procedures is shown in Table V.

In children with surgical closure of VSD, left ventricular ejection fraction was significantly lower and heart rate was increased in comparison to children treated with Membr VSDO implantation.

## Discussion

Rapid development of non-surgical treatments of many congenital heart diseases, including VSD, started in 1995, when Masura performed intra-atrial septal defect closure with the Amplatzer occluder. Modification of Musc VSDO allowed closure of perimembranous VSDs as well [12]. There have also been reports on the use of muscular occluders to close such defects [25].

Hijazi et al. were first to report a successful closure of perimembranous defects with the Membr MVSD occluder in six patients [13]. Since then several other authors have reported very good results of perimembranous VSD

**Table IV.** Comparison of echocardiographic indices of left ventricular systolic and diastolic function and diameter prior to and after Membr VSDO placement (Group A) and prior to and after surgery (Group C)

variables	Group A				Group C						
	Prior, n=11		After, n=11		Prior, n=12		After, n=12				
	median	Range	Mean±SD	p	Median	Range	Mean	p			
LVEDDI	1.12	1-1.32	1.1±0.07	0.004*	1.23	0.99-1.5	1.23±0.1	1.03	0.93-1.22	1.05±0.0	0.006*
EF [%]	70	57-80	70.63±5.5	0.048*	76	70-95	72.25±6.3	67.5	59.0-80.0	68.66±5.9	0.286
E [m/sec.]	0.94	0.8-1.39	1.01±0.2	0.284	0.91	0.66-1.37	0.93±0.1	0.92	0.78-1.14	0.93±0.1	0.146
A [m/sec.]	0.68	0.4-1.18	0.7±0.26	0.722	0.56	0.41-1.13	0.65±0.24	0.63	0.37-1.1	0.67±0.2	0.388
E/A	1.56	0.98-2.25	1.53±0.40	0.858	1.57	1.08-2.19	1.54±0.41	1.47	0.97-2.32	1.48±0.3	0.637
DCT [ms]	170	130-240	174.18±41.3	0.168	160	110-220	160.4±38.5	180	110-220	174.5±40.5	0.224
IVRT [ms]	50	40-70	53.63±12.0	0.308	45	40-65	49.54±9.6	40	30-5	42±9.6	0.123
HR [min <sup>-1</sup> ]	86	62-150	95.36±28.5	0.020*	82	56-119	83.6±18.1	101	58-120	98.58±16.6	0.050*

**Abbreviations:** LVEDD/mean. ref. – left ventricular end-diastolic diameter on TTE to mean reference body weight ratio; RVEDD/mean. ref. – right ventricular end-diastolic diameter on TTE to mean reference body weight ratio; EF – ejection fraction; E – mitral E flow velocity; A – mitral A flow velocity; E/A – E to A ratio; DCT – E deceleration time; IVRT – isovolumetric relaxation time; HR – heart rate

**Table V.** The comparison of left ventricular systolic and diastolic function and left ventricular diameter between groups A and C before and after the procedures

Variables	A vs CPrior	A vs CAfter
	p	p
LVEDDI	0.090	0.068
EF [%]	0.565	0.004*
MV:E [m/sec.]	0.739	0.927
MV:A [m/sec.]	0.346	0.525
E/A	0.975	0.880
DCT [ms]	0.346	0.413
IVRT [ms]	0.606	0.090
HR (min <sup>-1</sup> )	0.133	0.043*

**Abbreviations:** LVEDD/mean. ref. – left ventricular end-diastolic diameter on TTE to mean reference body weight ratio; RVEDD/mean. ref. – right ventricular end-diastolic diameter on TTE to mean reference body weight ratio; EF – ejection fraction; E – mitral E flow velocity; A – mitral A flow velocity; E/A – E to A ratio; DCT – E deceleration time, IVRT – isovolumetric relaxation time; HR – heart rate

treatment with the use of these occluders. Over several years of clinical use, the occluder delivery system has been improved, significantly facilitating correct placement of the device on the intraventricular septum [24, 26].

All our patients referred for such a procedure after TTE underwent successful VSD closure with the Membr VSDO.

The size of the occluder was matched to the VSD diameter measured on TEE and angiography. We made an effort to use an occluder size equal to or at most 1 mm larger than the diastolic VSD diameter. Occluders are sized at 2 mm intervals, and sometimes correct matching of the occluder size is difficult. Others have reported implanting occluders 1-2 mm larger than diastolic opening size [13, 28]. We noted that occluding the VSD flow reduced the defect size. Occasionally, inserting the sheath into the defect blocked the blood shunt through the VSD. The diameter of 9F sheath is about 3mm, and the smallest 4 mm occluder is introduced via the 7F system. In the youngest child only with a 4.5 mm defect measured on TEE and 4.7 mm on angiography, a 6 mm occluder was inserted, which was over 1 mm larger than the opening size. This child developed tricuspid regurgitation, absent before the procedure. We speculate that the new TR may have resulted from excessive occluder size or trapping of the tricuspid valve tendinous chordae during decompression of the disc in the right ventricle.

Very close proximity of the defect with the valve within the inflow tract (Kirklin type III) of the membranous intraventricular septum is no contraindication to transcatheter VSD closure, which was also mentioned in our previous study [24]. A significant reduction of tricuspid regurgitation was found after placement of the Membr

VSDO. Protruding the occluder under the tricuspid valve was described by Pedra et al. [27]. They used to close defects the occluders up to 2 mm larger than the diameter of the defect measured at diastole. Definite assessment of the appropriateness of occluder size matching the defect diameter requires further studies involving a larger number of patients.

Mitral regurgitation was found significantly more often after surgery than after VSD closure with the occluder. This may result from worse postsurgical left ventricular systolic function. The two groups did not differ in terms of aortic regurgitation prior to and after the procedures. Aortic valve competence also depends on the procedure's course. Careful catheter manipulation and proficient placement of the occluder determine its correct function.

Selection of occluder size may also affect the rate of other complications, such as arrhythmias. If the occluder is oversized it may cause compression of the cardiac conductive pathways. Complete heart block was not found in any child. Such complications have been reported; there are also data available on an episode of atrioventricular block after insertion of the long sheath into the defect. It is contraindicated to continue the procedure if such complications occur [27]. In our studies no significant differences between the children operated on and those treated with the occluder were observed with respect to arrhythmias.

Insertion of the intraventricular septum occluder under TEE and angiographic guidance enables its correct placement at a safe distance from the valves. Van der Velde reported as early as in 1994 the essential role of TEE in the assessment of valve function during VSD closure with other occluders as well [28]. Current occluder design allows its withdrawal into the sheath and changing its location, which took place in two of our patients. It is also possible to exchange the occluding device with a smaller or larger one; however, this is a costly solution. A residual shunt in the periooccluder area remained in one child. This child was found to have a membranous intraventricular septum aneurysm with three openings. The patient was treated with an occluder sized smaller than the aneurysm entry diameter (a larger occluder was unavailable at the time). Pedra et al. also emphasized that selection of occluder size is more difficult in such patients [27]. In one case the opening was not entirely covered by the occluder. It seems, however, that referring such children to these procedures is not impossible.

A residual VSD shunt was found in one of the children operated on. Data exist on the reduction of residual shunts in the PeriPatch region after surgery and on haemolysis in children with residual periooccluder or

PeriPatch shunts [1, 29]. The above-mentioned complication was not observed in our patients.

The left ventricular ejection fraction significantly improved in children treated with the occluder. Children who underwent surgery were not observed to have a significant change of left ventricular ejection fraction, which even slightly decreased after surgery. We suppose that the extracorporeal circulation and/or adhesions may compromise the left ventricular function additionally to volume overload prior to surgery. The reduced heart rate in children after VSD closure with the occluder may also confirm the better exercise capacity of children not undergoing surgery.

Diastolic function impairment was observed in all children prior to closure of the defects. These changes may result from left ventricular hypertrophy and increased stiffness [18, 19, 22]. Indices of diastolic function tended to normalize in children after occluder placement rather than in those after surgery; however, the statistical significance of such a trend was not found.

## Conclusions

1. During mid-term follow-up, left ventricular systolic function was better in patients with perimembranous VSD closure using Membr VSDO than after surgical repair.
2. Improvement of left ventricular systolic function and diameter after treatment of perimembranous VSD with the occluder may result in a lower incidence and degree of mitral regurgitation than in subjects treated with surgery.

## References

1. Bol-Raap G, Weerheim J, Kappetein AP, et al. Follow-up after surgical closure of congenital ventricular septal defects. *Eur J Cardiothorac Surg* 2003; 24: 511-15.
2. Serraf A, Lacour-Gayet F, Bruniaux J, et al. Surgical management of isolated multiple ventricular septal defects. *J Thorac Cardiovasc Surg* 1992; 103: 437-43.
3. Kitagawa T, Durham LA, Mosca RS, et al. Techniques and results in the management of multiple ventricular septal defects. *J Thorac Cardiovasc Surg* 1998; 115: 848-56.
4. Kidd L, Driscoll DJ, Gersony WM, et al. Second natural history study of congenital heart defects. Results of treatment of patients with ventricular septal defects. *Circulation* 1993; 879 (suppl I): I-38-I-51.
5. Konertz WF, Kostelka M, Mohr FW, et al. Reducing the incidence and severity of pericardial adhesions with a sprayable polymeric matrix. *Thorac Surg* 2003; 76 (4): 1270-4.
6. Bailey LL, Ze-jian L, Schulz E, et al. A cause of right ventricular dysfunction after cardiac operations. *J Thorac Cardiovasc Surg* 1984; 87: 539-42.
7. Lock JE, Block PC, Mc Kay RG. Transcatheter closure of ventricular septal defects. *Circulation* 1988; 78: 361-8.

8. Rigby ML, Redington AN. Primary transcatheter umbrella closure of perimembranous ventricular septal defects. *Br Heart J* 1994; 72: 368-71.
9. Karla G, Verma PK, Dhall A, et al. Transcatheter device closure of ventricular septal defects: Immediate results and intermediate – term follow-up. *Am Heart J* 1999; 138: 339-44.
10. Tofig M, Patel RG, Walsh KP. Transcatheter closure of a mid-muscular ventricular septal defect with Amplatzer VSD occluder device. *Heart* 1999; 81: 438-40.
11. Thanopoulos BD, Tsaousis GS, Kontadopoulou GN. Transcatheter closure of muscular ventricular septal defects with the Amplatzer ventricular septal defect occluder: initial applications in children. *J Am Coll Cardiol* 1999; 33: 1395-9.
12. Amin Z, Xiapong G, Berry JM, et al. Periventricular closure of ventricular septal defects without cardiopulmonary bypass. *Ann Thorac Surg* 1999; 68: 149-54.
13. Hijazi ZM, Hakim F, Haweleh AA, et al. Catheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: initial clinical experience. *Cathet Cardiovasc Interv* 2002; 56: 508-15.
14. Tede N, Child JS. Diastolic Dysfunction in patients with congenital heart disease. *Cardiol Clin* 2000; 18: 491-9.
15. Bonow RO, Borer JS, Rosing DR, et al. Left ventricular functional reserve in adult patients with atrial septal defect; pre- and postoperative studies. *Circulation* 1981; 63: 1315-22.
16. Siwińska A, Górzna-Kamińska H, Mroziński B, et al. Funkcja skurczowa i rozkurczowa lewej komory u młodzieży i dorosłych po operacji ubytku w przegrodzie międzykomorowej. *Kardiologia Pol* 2001; 55: 1-82.
17. Federmann M, Hess OM. Differentiation between systolic and diastolic dysfunction. *Eur Heart J* 1994; 15 (Suppl. D): 2-6.
18. Feigenbaum H. Echocardiography. Fifth edition. *Lea 7 Febiger* 1994.
19. Rakowski H, Appleton C, Chan KL, et al. Canadian consensus recommendations for the measurement and reporting of diastolic dysfunction by echocardiography. *J Am Soc Echocardiogr* 1996; 9: 736-60.
20. Thomas JD, Weyman AE. Echocardiographic Doppler evaluation of left ventricular diastolic function. Physics and physiology. *Circulation* 1991; 84 (3): 977-90.
21. Nishimura RA, Tajik J. Evaluation of diastolic filling of left ventricle in health and disease: Doppler echocardiography is the clinicians Rosetta's stone. *J Am Coll Cardiol* 1997; 30: 8-18.
22. Brakstor W, Krzyżanowski W, Kuch M. Echokardiografia dopplerowska podstawową metodą oceny czynności rozkurczowej lewej komory. *Ultrason Pol* 1996; 6: 9-18.
23. Caruana L, Ptrie MC, Davie AP, et al. Do patients with suspected heart failure and preserved left ventricular systolic function suffer from "diastolic heart failure" or from misdiagnosis? A prospective descriptive study. *BMJ* 2000; 321: 215-19.
24. Pawelec-Wojtalik M, Masura J, Siwińska A, et al. Zamykanie okołobłoniastego ubytku przegrody międzykomorowej przy pomocy zatyczek Amplatzer. Wyniki wczesne. *Kardiologia Pol* 2004; 61: 31-40.
25. Szkutnik M, Białkowski J, Baranowski J, et al. Nieoperacyjne zamykanie okołobłoniastych ubytków przegrody międzykomorowej za pomocą korka Amplatzer. *Folia Kardiologia* 2003; 10: 481-5.
26. Bass JL, Karla GS, Arora R, et al. Initial human experience with the Amplatzer perimembranous ventricular septal occluder device. *Catheter Cardiovasc Interv* 2003; 58 (2): 238-45.
27. Pedra C, Pedra S, Esteves C, et al. Transcatheter closure of perimembranous ventricular septal defects. *Expert Rev Cardiovasc Ther* 2004; 2 (2): 253-64.
28. Van der Velde ME, Sanders SP, Keane J, et al. Transesophageal echocardiographic guidance of transcatheter ventricular septal defect closure. *J Am Coll Cardiol* 1994; 23: 1660-5.
29. Hijazi Z. Device closure of ventricular septal defects. *Cathet Cardiovasc Interv* 2003; 60: 107-14.

## Leczenie okołobłoniastego ubytku przegrody międzykomorowej – porównanie metody inwazyjnej i operacyjnej

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### Streszczenie

**Wstęp:** Od czasu wprowadzenia do leczenia zatyczek Membr VSDO jest możliwe porównanie dwóch metod leczenia – operacyjnej i inwazyjnej kardiologicznej.

**Cel pracy:** Ocena skuteczności leczenia oraz ryzyka występowania powikłań u chorych leczonych z powodu okołobłoniastych ubytków przegrody międzykomorowej (VSD) zatyczką Membr VSDO i operacyjnie oraz porównanie czynności skurczowej i rozkurczowej lewej komory po zabiegu.

**Metodyka:** Zbadano 11 dzieci, u których okołobłoniaste VSD leczono za pomocą zatyczki (Grupa A) oraz 12 dzieci leczonych operacyjnie (Grupa C). Grupy pacjentów A i C różniły się nieco wiekiem ( $p=0,026$ ), lecz miały porównywalną masę ciała (0,103), wielkości stosunku przepływu płucnego do systemowego (Qp:Qs) ( $p=0,929$ ) i podobny czas obserwacji po zabiegu. Przed zamknięciem VSD nie stwierdzano w echokardiograficznym badaniu przekłatkowym (TTE) istotnych statystycznie różnic w funkcji skurczowej – frakcja wyrzutowa lewej komory (EF) ( $p=0,567$ ) i rozkurczowej – stosunek prędkości przepływu mitralnego fali E do A (E/A) ( $p=0,975$ ), czas deceleracji przepływu fali E (DCT) ( $p=0,346$ ), czas rozkurczu izowolumetycznego (IVRT) ( $p=0,606$ ) oraz częstości akcji serca (HR) ( $p=0,133$ ) i wielkości lewej komory (LV) obu grup ( $p=0,09$ ). W obu grupach wykonano badanie TTE i zmierzono powyższe parametry w okresie od 3 do 16 miesięcy po zabiegu i porównano wewnątrz i pomiędzy grupami. Oceniano również niedomykalności zastawek mitralnej (MV), trójdzielnej (TV) i aortalnej (Ao).

**Wyniki:** Niedomykalność zastawki trójdzielnej (TI) ( $p=0,028$ ) i niedomykalność zastawki mitralnej (MI) ( $p=0,043$ ) istotnie zmniejszyły się po zamknięciu VSD Membr VSDO. Stwierdzono istotnie statystycznie mniejszą MI po leczeniu zatyczką niż po leczeniu operacyjnym ( $p=0,026$ ). TI ( $p=0,486$ ) i niedomykalność zastawki aortalnej (AI) ( $p=0,607$ ) w okresie po zabiegu pomiędzy grupami A i C nie różniły się istotnie statystycznie. Po operacji EF lewej komory ( $p=0,004$ ) była istotnie statystycznie niższa, a HR ( $p=0,043$ ) wyższa w porównaniu do dzieci leczonych zatyczką. Nie stwierdzano istotnych różnic funkcji rozkurczowej – E/A ( $p=0,88$ ), DCT ( $p=0,413$ ), IVRT ( $p=0,09$ ).

**Wnioski:** 1) W obserwacji średnioterminowej EF lewej komory jest wyższa po leczeniu okołobłoniastych VSD zatyczką Membr VSDO niż po korekcji chirurgicznej. 2) Wyższa EF i redukcja wielkości lewej komory po leczeniu okołobłoniastych VSD zatyczką mogą wpływać na rzadsze występowanie i mniejszy stopień MI w porównaniu do pacjentów leczonych chirurgicznie.

**Słowa kluczowe:** Amplatzer Membr VSDO, okołobłoniasty ubytek międzykomorowy

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