Percutaneous closure of atrial septal defect

A consensus document of the joint group of experts from the Association of Cardiovascular Interventions and the Grown-Up Congenital Heart Disease Section of the Polish Cardiac Society

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ABSTRACT
Atrial septal defect is the most common congenital heart lesion in adults. Although atrial septal defect closure is recommended in those with right heart enlargement or paradoxical embolism, data informing such indications in adults are quite limited. This population has many unique characteristics and needs. In recent years, significant progress has been made with regard to diagnostic modalities that facilitate the diagnostic workup of these patients. However, the decision-making process, especially in selected adult patients population (advanced age, various concomitant diseases, poor LV function [both systolic and diastolic], pulmonary hypertension, concomitant arrhythmias or multiple defects, or deficient rims) is still not easy. Available data are predominantly focused on imaging endpoints and short-term morbidity and mortality rather than long-term. The evidence base for outcomes with or without defect closure comes from various studies with different observation periods. Moreover, the clinical experience in diagnosing and treating that subgroup of patients is inhomogeneous between individual physicians (cardiologists, imaging specialists, operators) and between small and large experience centers. In the view of the above, the joint group of experts from the Association of Cardiovascular Interventions and the Grown-Up Congenital Heart Disease Section of the Polish Cardiac Society developed the following consensus opinion in order to standardize the principles of diagnosis, indications for treatment, methods of performing procedures, and tenets of postoperative care in Poland.

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Background information on atrial septal defect Atrial septal defect (ASD) is one of the most prevalent congenital heart defects and may frequently remain undiagnosed until adulthood.[1-4] It is a consequence of various embryological disorders that cause interatrial communication. There are 5 types of anatomic defects known:

- Secundum ASD (ASD II): 80% to even more than 90% of ASDs in the adult population; located in the fossa ovalis region and its surrounding tissue.

- Primum ASD: 10% to 15% of ASDs; also known as partial atrioventricular septal defect or partial atrioventricular canal; located near the crux of the heart, nearly always associated with a cleft in the anterior mitral valve leaflet.
- Superior sinus venous defect: up to 5% of ASDs; located near the superior vena cava entry and often associated with an abnormal connection of the right upper pulmonary vein to the superior vena cava or the right atrium (RA).
- Inferior sinus venosus defect: less than 1% of ASDs; located in the inferior portion of the atrial septum, leading to an overriding of the inferior vena cava (IVC), often associated with anomalous connection of the right lower pulmonary vein to the IVC.
- Unroofed coronary sinus defect or coronary sinus septal defect: less than 1% of ASDs; partially or completely missing roof of coronary sinus with lack of separation from the left atrium (LA), often associated with a persistent left superior vena cava that drains into the coronary sinus.

Atrial septal defect may also occasionally be diagnosed together with other congenital cardiac abnormalities such as anomalous pulmonary venous return, persistent left superior vena cava, pulmonary valve stenosis, or mitral valve prolapse. In some of these circumstances, such as in patients with Ebstein anomaly and pulmonary stenosis, the pathophysiology related to ASD is much more complex, and closure of the defect could cause clinical deterioration.[4-6] Therefore, these recommendations address only isolated ASD and not ASD associated with complex cardiac defects.

The shunt volume through ASD depends on the defect size, right and left ventricular (LV) compliance, and left and right atrial pressures. A simple ASD causes a left-to-right shunt (significant when the defect size is relevant, usually more than 10 mm) secondary to a higher compliance of the right ventricle (RV) compared with the LV, and results in RV volume overload and an increased pulmonary flow. This increase results in late right heart failure with diminished functional capacity and fatigue. Atrial enlargement, which develops with time, leads to frequent atrial arrhythmias (atrial fibrillation and atrial flutter) and higher risk of thromboembolic complications.[6-8] A clinically significant ASD may lead to late pulmonary hypertension (PH). ASDs, like patent foramen ovale (PFO), may also permit paradoxical embolism resulting in stroke or transient ischemic attack.

A reduction in LV compliance, or any conditions (significant ischemic heart disease or hypertension, cardiomyopathies, valve diseases) which are associated with elevation of left atrial pressure, increase left-to-right shunt. Reduced RV compliance (pulmonary arterial hypertension [PAH], RV outflow tract obstruction), pulmonary arteries stenosis or other RV diseases) or tricuspid valve stenosis and / or regurgitation may decrease left-to-right shunt or in some cases cause shunt reversal which results in cyanosis.

Prevalence and genetics Although ASDs are usually sporadic, some are inherited as autosomal dominant syndromes, and heart lesions could be associated with other congenital defects such as Holt–Oram syndrome in which preaxial limb defects are caused by mutations in the *TBX5* gene on chromosome 12q24.1. Family forms of ASD with progressive atrioventricular blocks may suggest mutations or haploinsufficiency of the *Nkx2.5* gene on chromosome 5, while familial ASD without conduction abnormalities may be associated with *GATA4* mutations. Also, defects in genes responsible for the formation of the atrial septum (eg, *MYH6*, *TBX20*) can lead to defects of the intra-atrial septum. Exposure to teratogenes during pregnancy is another potential cause of ASD as observed in fetal alcohol syndrome.[9-12]

Natural history, clinical presentation, and management of unoperated patients

Patients with ASD usually remain asymptomatic until adulthood. Majority of them may develop symptoms after the fourth or fifth decade of life, including: shortness of breath, poorer exercise tolerance (dyspnea and fatigue), and supraventricular arrhythmias (atrial fibrillation, atrial flutter, atrial tachycardia, or sick sinus syndrome). Sometimes paradoxical emboli, heart failure or RV failure, and pulmonary vascular disease can also be observed (5% to 9% of patients).

Studies describing unoperated patients with large ASD (before 1980) revealed that nearly 25% of these patients died before the age of 27 years and 90% before the age of 60 years. Life expectancy is still reduced if closure is performed after the age of 25 years, but survival is much better than previously assumed. Pulmonary artery pressure (PAP) can be normal, but usually increases with age. Severe increase of resistant PH is rare (less than 5%) and its development is related to additional multifactorial factors similar to idiopathic PH.[13] After the fifth decade of life, the severity of PH increases and tachyarrhythmias are more common. Symptoms vary widely with age at presentation and shunt volume, which means that symptoms alone cannot guide therapy.[1]

Physical examination can reveal systolic silent murmur, fixed split second heart sound, or diastolic flow rumble across the tricuspid valve, but the introduction of cardiac ultrasonography has enabled early diagnosis. Low mortality rates after surgical repair led to rapid increase

in the number of ASD closure procedures over the past 2 decades, but the availability of percutaneous closure procedures facilitated the decision to proceed with the intervention especially in children.[13] Currently, only few patients remain treated conservatively without percutaneous or surgical repair due to severe vascular disease leading to irreversible PH and Eisenmenger syndrome.

In patients with atrial fibrillation and / or after thromboembolic event, oral anticoagulation may be considered. There are no data supporting the use of calcium channel blockers in patients with Eisenmenger syndrome. In these patients, according to the European Society of Cardiology (ESC) guidelines, 3 classes of drugs have been recently approved for treatment of PAH: endothelin receptor antagonists, phosphodiesterase type-5 inhibitors, and prostanoids. Efficacy and safety of these drugs have been demonstrated in PH associated with congenital heart disease and in Eisenmenger syndrome. Treatment of Eisenmenger syndrome should include also very late complications associated with hyperviscosity symptoms, bleeding and thrombotic diathesis, thromboembolic events (paradoxical emboli), microcytosis, iron deficiency, arrhythmias (both supraventricular and ventricular), infectious complications (endocarditis, cerebral abscess, pneumonia), and many others.[13]

Noninvasive diagnostics

Echocardiography

Echocardiography is the main imaging modality in the diagnostic workup of ASD. It is necessary in qualification and monitoring of interventional procedures as well as in follow-up, with particular emphasis on peri- and postoperative complications.[14-17] Transthoracic echocardiography (TTE) enables not only the recognition of the defect, but also the assessment of shunt hemodynamic significance. Transesophageal echocardiography (TEE) is required in terms of inconclusive TTE or during qualification for percutaneous interventions.

Transthoracic echocardiography Transthoracic echocardiography is the preferred initial diagnostic modality for the detection and diagnosis of ASD. To properly evaluate the size and location of a communication, multiple views should be used: different cuts of parasternal and apical but also subxiphoid views are very useful, also in adults with poor acoustic window. A detailed overview of the recommended projections is beyond the scope of this document, but should be consulted in the relevant guidelines.[1]

Hemodynamically significant ASD causes dilatation of the RV with paradoxical intraventricular septum movement, RA, and pulmonary trunk which can be assessed on TTE.[14-16] Apart from the RV dimension, it is also important to assess its function.

Symptoms of RV overload are one of the indications for defect closure. Another parameter evaluated in the TTE is the approximate measurement of systolic or mean PAP. In case of any doubt, right heart catheterization (RHC) is required to verify the significance of PH. Transthoracic echocardiography is usually efficient enough to determine not only the ASD type of but also septum morphology and the presence of septum aneurysm defined as a redundancy of tissue with the excursion of 10 mm from the atrial septum plane into the RA or the LA. Transthoracic echocardiography should also include evaluation of mitral and tricuspid valve regurgitation since ASD may coexist with mitral valve prolapse and dilatation of tricuspid ring, which should be considered when referring a patient for percutaneous or surgical treatment. It is mandatory to exclude other congenital malformations, for example, ventricular septal defect or pulmonary stenosis.

Another crucial issue is LV systolic and diastolic dysfunction. Closure of ASD may cause LV overload and acute heart failure with pulmonary edema. It should be emphasized that false-positive detection of ASD on TTE may occur as a result of misinterpretation of, for example, the inflow from the IVC directed towards the atrial septum. Intravenous first-generation contrast infusion or TEE may be conclusive in such condition. Secundum ASD can be misdiagnosed in the presence of a coronary sinus defect—dilated coronary sinus drains to the RA and may imitate lack of continuity of the septum. In this particular condition, contrast injection but only through the left ulnar vein may confirm the diagnosis.

The image quality of TTE, especially in adult patients, does not always permit a correct and detailed evaluation of the shunt; however, right heart overload should prompt a thorough search for its causes, including TEE.

Transesophageal echocardiography Transesophageal echocardiography examination is performed to confirm the diagnosis of the shunt, define its type and, in the case of ASD II, assess the possibility of performing percutaneous closure. It is necessary to determine and verify the number, size, and shape of defects and their spatial relation to the surrounding structures.[14-17] It is pivotal to evaluate rims—the aortic rim is the only one not obligatory for percutaneous closure. All other rims should be at least 5 mm, although in addition to length, their quality is also important. Three-dimensional (3D) imaging is of special importance in assessing shape, size, and spatial relations of especially multifenestrated defects. It is also crucial to assess the length of the pulmonary veins to the LA and dimension of the Eustachian valve and Chiari network which may interfere with catheters

in case of large size or excessive mobility. Such comprehensive assessment is possible in 2D imaging; however, it requires systematic analysis of projections obtained at different esophagus depths and at different angles. A detailed description of the TEE assessment during ASD evaluation and closure procedure (including 3D imaging) is included in Supplementary material.

Cardiac magnetic resonance Echocardiography still remains the gold standard in everyday routine diagnostic work-up of patients with suspected or otherwise confirmed ASD II. This is further supported by increasing accuracy of the assessment, with the introduction of real-time 3D echocardiography on a broader scale. Nevertheless, cardiac magnetic resonance (CMR) has recently become increasingly important in the diagnosis and evaluation of different congenital entities in adults, including ASD II.[18,19]

In general, 3 different scenarios for CMR application can be distinguished. First, it can be applied when, for a number of reasons, the quality of echocardiographic examination is not sufficient. In this regard, CMR can provide information in all aspects usually covered on echocardiography including systemic blood flow [Qs] and pulmonary blood flow [Qp]; however, with inferior ability to assess gradients and / or pressures (eg, PAP) or accompanying small mobile tissue structures. Second, when borderline or conflicting results are obtained on echocardiography, CMR can serve as a second-line method that helps to decide on further clinical steps, especially when it comes to volumes and ejection fraction issues. Third, CMR may be considered as a reference imaging modality in areas where it exceeds echocardiographic performance. Typically, in the suspicion of left-to-right shunt, CMR is considered as an ideal modality to delineate pulmonary venous connections, especially anomalous (eg, innominate or vertical vein).[20,21] Another area in which CMR excels is RV volumes, mass, and ejection fraction estimation (RV overload) which can be difficult to accurately assess by echocardiography.[22,23] It is also worth noting that CMR, due to lack of ionizing radiation, is safe for serial examinations.

Cardiac computed tomography In the setting of ASD II, indications for cardiac computed tomography are similar to those fore CMR.[1,24] Cardiac computed tomography is usually more accessible than CMR and is not contraindicated after permanent pacemaker implantation. In terms of diagnostic abilities for adults with congenital heart disease or after surgery / repair due to superior spatial resolution, it is best suited for extracardiac vascular anatomy assessment and less useful for ventricular volumes / function or valvular structure / function estimation (compared to echocardiography and CMR). Its major drawback is potentially higher radiation dose, therefore limiting its use for serial applications.[25] When used for cardiac assessment, ECG-gated acquisition is recommended. Additionally, in older patients (usually >40 years of age) with confirmed ASD II and referred for either device or surgical closure and with concomitant risk for coronary artery disease, coronary computed tomography angiography might be performed instead of conventional angiography.

Exercise test Adults with congenital heart disease, including ASD, are a rather specific group in whom symptoms are likely to be underestimated and limitations underreported. Moreover, considering the fact that their disease is lifelong, they might have never experienced "normal" function. Cardiopulmonary exercise testing (CPET) with objective assessment of exercise capacity is believed to be useful in both decision-making in unrepaired ASD II and in follow-up after device or surgical closure.[1,24] Usually, in unrepaired ASD, one can expect decreased exercise capacity that is demonstrated by maximum oxygen uptake reduction (peak VO_2 or VO_{2max}), especially seen in older patients.[26] It is not well established whether this decrease is correlated with hemodynamic parameters at rest (eg, mean pulmonary arterial pressure, ratio of Qp to Qs). Interestingly, but in agreement with specificity of the population, decreased capacity in CPET can be found in the so-called asymptomatic patients. Additionally, other parameters that can be affected in baseline CPET are ventilator efficiency (increased VE / VCO_2 slope due to RV dysfunction and / or pulmonary pressure elevation and / or lung disease) and ventilatory anaerobic threshold (decreased due to muscular deconditioning).[26] Given the objective, reproducible, and repeatable assessment and correlation with mortality and morbidity, CPET may play a role in the decision-making process and timing of intervention, and afterwards, may be part of serial follow-up checks after device or surgical closure.

When CPET is not available or the patient cannot complete it for different reasons, the simple 6-minute walk test can be applied instead. It provides more limited data; however, its prognostic value is still higher than relying on medical history alone.[24]

Pulse oximetry Pulse oximetry is a noninvasive, quick, simple, safe, and painless test that measures oxygen saturation (SpO_2). It does not require calibration and is able to provide instantaneous data that correlate well with blood gas measurements. Its use has recently been advocated by guidelines for screening of patients with ASD and accompanying pulmonary vasculopathy.[24] In this subset of patients, despite left-to-right shunt present at rest (SpO_2>90%),

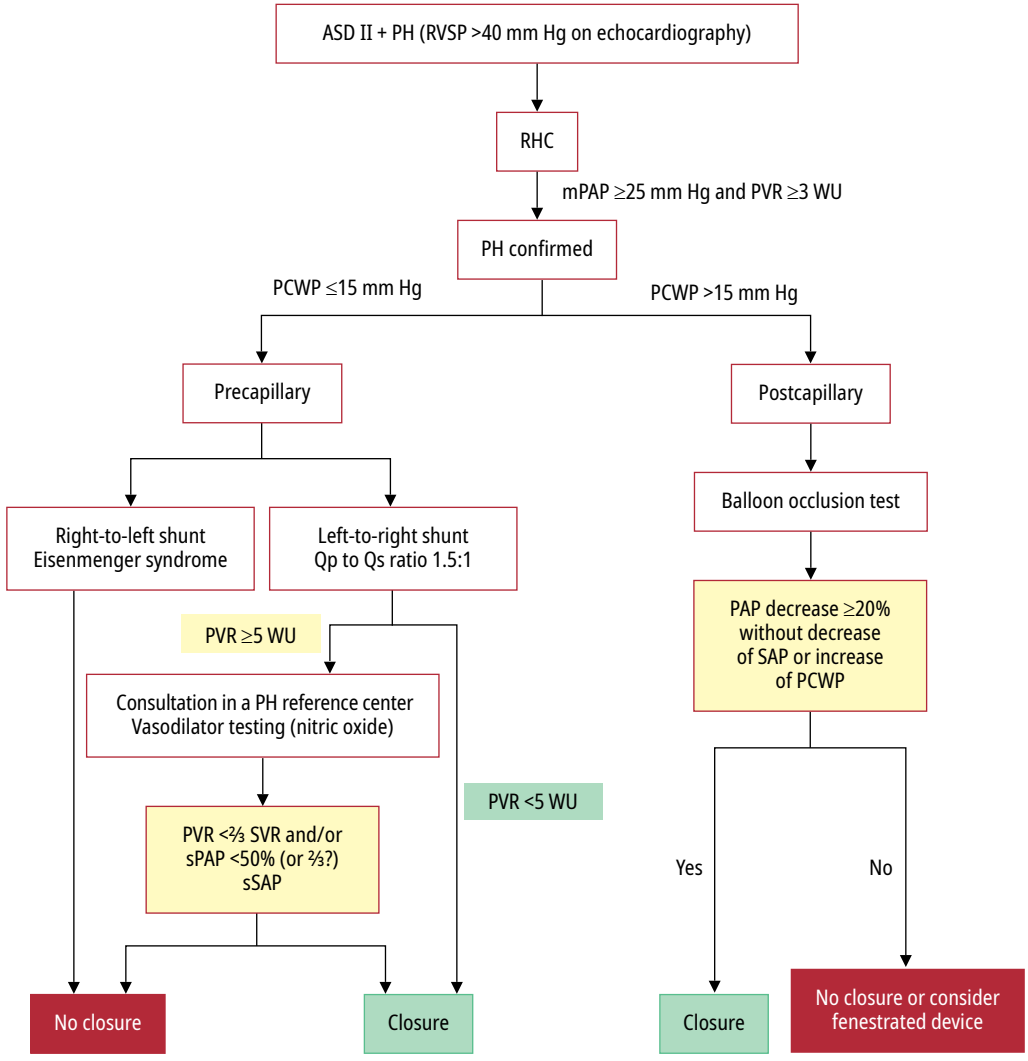

FIGURE 1 Diagnostic algorithm for patients with atrial septal defect and pulmonary hypertension
Abbreviations: ASD II, atrial septal defect type 2; mPAP, mean pulmonary artery pressure; Qp, pulmonary blood flow; Qs, systemic blood flow; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RHC, right heart catheterization; RVSP, right ventricular systolic pressure; SAP, systemic atrial pressure; sPAP, systolic pulmonary artery pressure; sSAP, systolic systemic atrial pressure

flow mediated dilatation of the pulmonary artery cannot increase appropriately and pulmonary arterial resistances go up during exercise resulting in shunt reversal and subsequent SpO_2 drop (<90%). To that end, exercise pulse oximetry can eventually result (eg, after confirming on CPET) in either a decision not to close the defect or to postpone closure with prior preparation and administration of specific pharmacological agents (eg, sildenafil or bosentan) known to reduce pressures and desaturation.[26]

Invasive diagnostics: heart catheterization

Right heart catheterization is the gold standard for the diagnosis of PH in patients with ASD and is used to select patients for defect closure. To determine detailed hemodynamics for decision-making or to clarify discrepant or inconclusive data from noninvasive imaging, diagnostic catheterization may be necessary (FIGURE 1). Right heart catheterization is necessary to assess the severity of hemodynamic impairment and undertake vasoreactivity testing in selected patients.[27]

Selection of patients for RHC is based on the results of echocardiography. If peak tricuspid regurgitation velocity exceeds 2.9 m/s (or RV systolic pressure exceeds 40 mm Hg), PH occurrence is highly suspected.[27] Right heart catheterization should be performed in centers with an expert catheterization laboratory in accordance with current guidelines.[27-29] Right heart catheterization is a technically demanding procedure that requires attention to detail to obtain clinically useful information. Proper patient hydration before the examination is of great importance. The procedure is performed from a central vein puncture using a Swan-Ganz catheter

terminated with a balloon, to measure the pulmonary capillary wedge pressure (PCWP). Direct measurements of blood pressure in the RA, RV, pulmonary artery, and in some cases also in the LA are also performed. Pulmonary hypertension is diagnosed if the mean PAP is 25 mm Hg or higher (more than 20 mm Hg according to the newest guidelines) and simultaneously pulmonary vascular resistance (PVR) is 3 WU or higher.[30] Cardiac output and pulmonary and systemic flow are assessed using the Fick method.[27-29] For this purpose, blood samples for oximetry should be taken from the superior and the IVC (to calculate the oxygen saturation of mixed venous blood in the RA), then in the pulmonary artery. Systemic arterial blood oxygen saturation (in the aorta or peripheral artery) should be determined separately. Subsequently calculations using standard formulas should be performed. The direct oxygen uptake test should be performed before RHC (direct Fick method). This technique is, however, not widely available. It is also possible to estimate oxygen consumption on the basis of the patient's age, sex, and body surface area according to the formula proposed by Bergsta et al,[31] or on the basis of body surface area: body surface area × 125 ml/min or body surface area × 110 ml/min in elderly patients. The indirect Fick method, which uses estimated values of oxygen consumption, is acceptable (20% measurement error is possible).[29] The minute cardiac output by the Fick method is calculated based on the following formula:

$$CO = \frac{\text{oxygen consumption [ml/min]}}{\text{(arterial oxygen content – venous oxygen content) / 10 [ml/l]}}$$

where arterial oxygen content = 1.36 × hemoglobin concentration [g/dl] × arterial oxygen saturation, and venous oxygen content = 1.36 × hemoglobin concentration [g/dl] × venous oxygen saturation.

Based on that, Qp, Qs, as well as cardiac index, and pulmonary and systemic vascular resistance (SVR) values may be calculated. The Qp to Qs ratio exceeding 1.5 in the absence of concomitant irreversible PH is an indication for defect closure.

Table 1 Classic recommendations for closure of atrial septal defect according to the new 2020 European Society of Cardiology guidelines for the management of adult congenital heart disease

Indications for atrial septal defect closure	Class	Level of evidence
Patients with ASD and evidence of right ventricle volume overload and no pulmonary hypertension or left ventricular disease	I	B
Balloon testing is recommended before the decision to close in patients with ASD and left ventricle disease	I	C
Patients with suspicion of paradoxical embolism regardless of size of the defect.	IIa	C
Patients with elevated PVR (3–5 WU) when significant left to right shunt is present (Qp to Qs ratio >1.5)	IIa	C
Fenestrated ASD closure may be considered in patients with PVR ≥5 WU, when significant left-to-right shunt (Qp to Qs ratio >1.5) is present and PVR falls below 5 WU after PH treatment	IIb	C

Abbreviations: see FIGURE 1

Classic indications for intervention: catheter/surgical First surgical ASD repair was reported in 1948,[32] and over the next years, it developed into a procedure with minimal mortality and morbidity. Nowadays, long-term results of surgical repair of ASD II are excellent, especially in young patients under 25 years of age.[13] Safe methods of minimally invasive surgery improved cosmetic results and shortened recovery.[33] The percutaneous transcatheter closure of ASD II was first published in 1974,[34] and it has become widespread after developing the Amplatzer septal occluder (AGA Medical, Plymouth, Minnesota, United States), followed by other devices.

Closure of ASD is indicated in the presence of substantial left-to-right shunt leading to a significant right heart enlargement due to volume overload.[1,24] The significance of shunting could be also determined by the ratio of Qp to Qs higher than 1.5, although this index is of secondary importance comparing to the signs of the above-mentioned RV remodeling. Such indication is supported by strongest evidence. The presence of PH, as it has been mentioned before, requires a more thorough workup. Right heart catheterization may be useful if the echocardiography is nonconclusive. As PAH is potentially one of the consequences of a left-to-right shunt, the indication for an interventional treatment is additionally constrained by the values of PAP / PVR. Closure of ASD should not be performed in patients with Eisenmenger physiology, those with PAH and PVR ≥5 WU despite targeted PAH treatment or desaturation on exercise. New 2020 ESC recommendations for ASD closure are listed in TABLE 1.

Anatomic factors Transcatheter closure has emerged as the leading method of closure in ostium secundum defects. It is now dedicated for patients with feasible morphology (defect diameter ≤38 mm, sufficient rims of 5 mm except towards the aorta—class of recommendation I). Specific indications for surgical approach include: ASD other than secundum, lack of sufficient rims (<5 mm) other than towards aorta, need for other cardiosurgical interventions.

The insufficient retroaortic rim is not an absolute contraindication for transcatheter closure, but was recognized as one of the factors determining the increased risk of device-related aortic or atrial erosion.[35] The qualification for

interventional treatment in case of such anatomy should be proceeded within the Heart Team and the potential risk of a transcatheter approach and a surgical alternative should be carefully discussed with the patient. For transcatheter treatment, the devices without restrictions for the insufficient aortic rim in instruction for use are preferred.

Patients with other types of ASD are usually referred for surgical closure. Those with borderline anatomy should be discussed within the Heart Team.

Special populations Special populations include patients with small ASD (not fulfilling above criteria) and:

- History of cerebrovascular or other embolic events that may be explained by right-to-left shunt and paradoxical emboli. Such patients may be considered for ASD closure regardless of the defect size. The hemodynamic significance of left-to-right shunting is not necessary to be proven in patients with suspicion of paradoxical embolism. In such patients, the workup should include opinion of a neurologist (ESC class of recommendation IIa, level of evidence C).
- Patients with orthodeoxia-platypnea syndrome may be considered for transcatheter closure.
- Patients with small defects but engaging in professional or recreational activities which can increase the risk of paradoxical embolism (eg, divers).
- Patients with small ASD who are planning pregnancy.
- Patients with ASD and functional tricuspid regurgitation related to right heart remodeling are a specific subgroup that not precisely addressed in the current guidelines. The results of a prospective registry[36] demonstrated a widespread improvement in TR severity after percutaneous ASD closure, which may support the choice of this treatment over surgical ASD closure with tricuspid repair. The precise identification of TR mechanism remains of critical importance in this setting.

Interventions in special situations **Patients with left ventricular dysfunction and older adults / elderly patients** There are some differences between teenagers / young adults and older or elderly patients with ASD. Elderly patients or older adults (>60 years of age) with ASD more frequently present with elevated systolic PAP, significant tricuspid regurgitation, and atrial fibrillation. Persistent atrial fibrillation has been observed in one-third of patients with ASD older than 60 years. The majority develop symptoms of reduced functional capacity, shortness of breath during exercise, and palpitations. Symptoms were observed in 83% of patients older than 60 years.[37]

In general, patients with significant shunt and PVR of less than 5 WU should undergo ASD closure regardless of symptoms and age.[1] It should be emphasized that elderly patients also benefit from closure.[1,37,38] Patients older than 60 years improved markedly, with almost 70% of them being asymptomatic after the intervention compared with about 16% before.[1]

Since ASD closure is significantly less invasive than operation and associated with fewer complications,[39] it became an attractive therapy for the older adults / elderly. Percutaneous closure of ASD can be performed safely and successfully in those patients. Symptoms reduction, improvement of functional exercise capacity, and recovery of both RV and LV functions have been reported.[40]

Thus, interventional closure of ASD II with a significant left-to-right shunt can be recommended in elderly patients (>60 years), with good results in the majority of them.[41] In patients of advanced age with ASD who are not feasible for device closure, individual surgical risk associated with comorbidities should be carefully weighed against the potential benefits of ASD closure. Surgical closure of ASD should also be considered in patients who are undergoing tricuspid valve repair or replacement.[1]

Atrial septal defect closure may not be associated with electromechanical improvement in elderly patients despite improved ventricular dimensions and reduced symptoms, and appears not to affect the arrhythmias they had before, or frequency of new which develop during follow-up.[4,7] Those patients should be informed about that before the intervention.

In patients with angina pectoris or even risk factors for coronary disease, a coronary angiogram is advised before closure.[1,42] It is reasonable to use computed tomography coronary angiography to exclude significant obstructive lesions in patients with a low / intermediate risk of coronary artery disease.[24] Patients with significant stenosis can be treated by percutaneous coronary intervention during the same procedure.[42]

In general, the shunt volume depends on RV / LV compliance and defect size. Closure of ASD with abolishment of left-to-right shunt leads to augmented LV filling by increased LV preload and therefore improved LV stroke volume and increase of functional capacity. In older patients, aging, comorbidities such as hypertension (LV hypertrophy), vascular disease, myocardial infarction, cardiomyopathy may cause decreased LV compliance.[43] A restrictive diastolic ventricular function is generally observed more frequently in older adults, and it may lead to secondary PH and, as a consequence, to pressure-overload RV failure. In these cases, reduced LV compliance may also increase left-to-right shunt through the defect, and may secondarily lead to volume-overload RV failure and worsening

of symptoms.[37] This may justify closure of defects considered to be anatomically borderline.

On the other hand, in patients with impaired LV function (both systolic and diastolic) and increased LV end-diastolic and LA pressures, ASD can have a decompressive effect on the LV. The abrupt closure of ASD in this setting may lead to rapid volume and pressure overload of the left heart, and may result in acute LV failure and pulmonary edema requiring mechanical ventilation, catecholamines support, and increased doses of diuretics.[41] Atrial septal defect closure in such situations may also lead to reduced exercise tolerance, supraventricular arrythmias and biventricular congestive heart failure.[37,44]

Patients with evidence of LV dysfunction should undergo careful additional evaluation before ASD closure to establish the optimal treatment.[24,43] Plasma biomarkers (brain natriuretic peptides) may also be helpful in identifying patients with symptoms of subclinical heart failure.[45] If ASD closure is planned in those patients, preinterventional assessment with echocardiography and evaluation of mitral inflow pattern and ASD balloon occlusion with reassessment of hemodynamics is recommended.[37] The balloon occlusion test includes temporary occlusion of the ASD with a sizing balloon and maintaining catheters in the LA and LV to monitor pressure changes. Special care should be taken not to compromise pulmonary veins and mitral valve inflow with the inflated balloon.

In patients with a left atrial pressure increase of more than 10 mm Hg, or systemic pressure drop, or pulmonary pressure increase (positive balloon occlusion test), the closure should be postponed.[37,46] Pulsed Doppler measurements of the mitral valve inflow during balloon inflation may also help to estimate the ASD closure results. The high-risk patients can be recognized if a pathological increase of the E to A ratio (ratio of early to late ventricular filling velocity) of the mitral inflow pattern is observed.[37]

Further treatment is requested before closing the defect in those patients to reduce the risk of worsening symptoms of left heart failure after the intervention. Reversible causes of LV dysfunction such as myocardial ischemia or uncontrolled hypertension should be treated first.[46] If this is not possible, a pharmacological treatment with diuretics, vasodilators, or inotropes is advised. This LV "preconditioning" before ASD closure, applied for 48 hours to 4 weeks, was successful in all reported high-risk patients older than 60 years.[47,48]

Self-fenestrated devices (eg, Amplatzer ASD occluders) have been described to successfully minimize the risk of heart failure and pulmonary edema following successful device closure. A self-made fenestration of 4 to 6 mm can be made in the occluder to permit residual shunting and can be closed later with a vascular plug if residual shunting is hemodynamically significant and balloon occlusion does not show any significant increase in LV filling pressure.[49]

In summary, irrespective of the differences with younger patients, the symptomatic older adults / ederly (>60 years) with secundum ASD benefit from transcatheter closure. Coexisting systolic / diastolic LV dysfunction should be taken into account in these patients. The abrupt closure of ASD in this setting leads to rapid volume and pressure overload of the left heart, and may result in acute LV failure. Patients with evidence of LV dysfunction should undergo additional preinterventional echocardiographic and catheter evaluation. In some patients, ASD closure has to be deferred and performed after successful „preconditioning" of the LV.

Patients with pulmonary hypertension Pulmonary hypertension with an increased systolic PAP of 40 mm Hg or higher, assessed by echocardiography, has been observed in 6% to 35% of patients with ASD II.[50] Moderate-to-severe PH in ASD is seen in 9% to 22% of cases.[51] Pulmonary hypertension in patients with ASD may be associated with functional capacity limitations, heart failure, atrial tachyarrhythmias, and increased mortality. Preprocedural PH remains a predictor of heart failure, arrhythmias, and mortality even after defect closure.[50-52]

If PH is suspected on echocardiography, RHC should be performed. Pulmonary hypertension is diagnosed if mean PAP is higher than 25 mm Hg and PVR is 3 WU or higher. In addition, precapillary PH is diagnosed if PCWP is 15 mm Hg or below, and postcapillary PH is diagnosed if PCWP exceeds 15 mm Hg.[30]

Pulmonary hypertension in the setting of ASD can be secondary to various etiologies. Postcapillary PH may be secondary to elevated LV end-diastolic pressure as seen in patients with ischemic heart disease, arterial hypertension, diabetes mellitus, and chronic kidney disease or due to mitral valve disease.[53] Precapillary PH associated with ASD may be a result of increased blood flow through a large shunt and increased pressure causing pulmonary arteriopathy, but in some cases, PH may be disproportionate to the shunt volume. Patients with reversible PH who will clearly benefit from shunt closure are at one end of the spectrum. At the other end, however, are those with irreversible PH with shunt reversal (Eisenmenger syndrome) in whom closure of ASD is not recommended and should be managed pharmacologically (TABLE 1).[27,53]

The main challenge is the precise identification of patients with ASD and reversible PH who may benefit from shunt closure.[53] A therapeutic strategy in patients with ASD II and significant PH remains controversial due to lack of evidence-based trials. In case of post-capillary PH and LV dysfunction, ASD closure

may precipitate acute LV insufficiency. For this reason, the balloon occlusion test (balloon inflation sealing the defect until the shunt completely disappears, lasting 10–15 minutes along with PCWP and PAP monitoring) should be performed. If the PAP decreases by more than 25% with no decrease in systemic pressure and PCWP at the same time, the test is deemed positive and closure may be considered.[54] Otherwise, implantation of a fenestrated device may be an option.[55]

There is no precise cutoff parameter that would preclude ASD II closure in the presence of precapillary PH. Currently, according to the ESC guidelines, the closure is recommended, if the defect is significant and PVR is less than 5 WU.[1] However, guidelines indicate that PH may be corrected by closure of the defect when PVR is 5 WU or higher, but the shunt is still left to right, PAP is less than two-thirds of systemic levels, and the ratio of PVR to SVR index is below 2/3, without a specific PVR cutoff value. If values exceed 5 WU and PVR to SVR index is above 2/3, the decision should be made in an expert center based on a hemodynamic assessment with the reversibility test, optimally with nitric oxide usage.[1] According to the 2018 guidelines if the American College of Cardiology and American Heart Association for the management of adults with congenital heart disease, closure of ASD, either percutaneously or surgically, may be considered if left to right shunting (Qp:Qs) is 1.5:1 or greater, systolic PAP is 50% or more of systemic arterial systolic pressure, and/or PVR is greater than one-third of the systemic resistance.[2] Nevertheless, ASD closure should not be performed in adults with systolic PAP higher than 2/3 systolic systemic arterial pressure and/or PVR higher than 2/3 of SVR.[2] On the other hand, Galiè et al[27] considered closure to be contraindicated at a PVR of 4.6 WU or higher (PVR index – PVRI, more than 8 WU × m^2), but the recommendation was based on an expert opinion rather than randomized trials. Despite the lack of reliable data, acute pulmonary vasodilator testing in reference PH centers is widely recommended in cases with a baseline PVRI of 4 to 8 WU × m^2 to assess the residual dilatatory capacity of the pulmonary vascular bed. A decrease of 20% in PVR, and decrease of 20% in the ratio of PVR to SVR, resulting in a final PVRI of less than 6 WU × m^2 and a final ratio of PVR to SVR of below 1/3 are considered to indicate a favorable outcome after shunt closure.[56,57] Re-evaluation can optionally be made after a short attempt at treatment with specific PH therapy, for example, endothelin receptor antagonists (bosentan), prostacyclines, or phosphodiesterase-5 inhibitors. The major limitation of these recommendations is that they are based on expert opinions or data derived only from small case series.

Patients with multiple defects or deficient rims **Interventions in patients with multiple defects**

Patients with multiple interatrial defects account for about 10% of all patients with secundum ASD. There are many challenges to consider when planning percutaneous closure of multiple interatrial defects. One of the basic ones is to accurately determine the number and size of individual defects, the distance between them and the topography of the defects in relation to each other and to surrounding structures of the heart. It is also important to determine whether the septum structure is stable or aneurysmatic, since the latter often have multiple defects.

Meticulous diagnostic imaging is extremely important for the percutaneous closure of multiple interatrial defects. Real-time 3D echocardiography should always be considered in addition to standard transthoracic and TEE. It facilitates the understanding of spatial relationships and helps to plan the transcatheter procedure. In addition, which is very important, it allows recognizing whether one irregular defect is not incorrectly interpreted as multiple defects. In some cases, balloon calibration may also be useful when planning the procedure. With its help, we can determine the compliance of the atrial septum tissue, usefulness of the rims, and likelihood of closing the surrounding defects at the same time.

Usually, small defects in the close proximity of a large defect can be closed with a single device. This mainly applies if the defects are not located further than 7 mm away from the large defect (the protruding part of the left atrial disc). If a small residual shunt remains close to the implant, it usually closes during follow-up due to endothelization process.[58] If it does not happen after 6 months, subsequent device implantation may be considered. Using 1 device is cost-effective, but most importantly, may help avoid bulky profiles.

Alternatively, if the defects are close together and the tissue separating them is thin, it is possible to implant an oversized device in the hope that this tissue band will burst creating one larger defect.[59]

If the defects are located more than 5 to 7 mm away from each other, 2 implants should be used. There are various techniques for such implantation. If the defects are far away, the order of release does not matter. However, if the implants interfere with each other, it is best to use the interleaving technique. The implants are released simultaneously in the following order: the left atrial disc of the first implant, then the left and the right atrial disc of the second implant, and finally the right atrial disc of the first implant. This ensures the least bulky profile of the atrial septum.[60] Alternatively, occluders can be implanted sequentially, but there is no agreement on this topic in the available literature on which occluder should be implanted first: larger or smaller.[59-61]

For a multifenestrated large aneurismatic septum, a non–self-centering device placement may be a good option.[62] Sometimes in such cases, in order to close all the defects while stabilizing the floppy septum, one may consider puncturing the central part of the septum with a transseptic needle and implanting through this hole a non–self-centering implant.

Percutaneous closure of multiple (or multifenestrated) interatrial defects is possible and effective; however, there are reports of a higher frequency of residual shunts.[63]

Interventions in patients with deficient rims The ideal type of interatrial defect for percutaneous closure is the defect located in the central part of the septum with minimum rims of 5 mm or larger. A deficient rim is defined as less than 5 mm.[64]

The deficient retroaortic rim is present in 36% to 57% of patients with secundum ASD.[65] It was shown that percutaneous closure of ASD with the absent or deficient retroaortic rim is the cause of an increased risk of erosion, but most leading centers treat these patients percutaneously. The deficient retroaortic rim is associated with an increased risk of impingement of the device on the aorta, but this is not associated with aortic regurgitation. More recent studies do not confirm the relationship of the deficient retroaortic rim with an increased risk of erosion or other adverse events.[66] However, in patients with large defects, the absence of the retroaortic rim can cause difficulties in the percutaneous closure of ASD. In this situation, the left atrial disc of the implant trends to slip over the anterior wall of the atrium and prolapse into the RA.

There are a number of technical modifications helpful to close these challenging defects. The most common is the clockwise rotation of the delivery system, thus the left disc of the device attempts to position itself towards the roof of the LA before the right disc is implanted. If this technique is ineffective, the left disc of the device can be initially implanted into the right or left upper pulmonary vein. After implantation of the right disk, introducer sheath is gently pushed so the left disk can prolapse from the pulmonary vein into the correct position. Finally, the balloon-assisted closure technique might be considered. By the additional femoral venous sheath, the sizing balloon is introduced to the septum and partly inflated during the device implantation. After the complete implantation of the device, the balloon is deflated and carefully removed. In some cases, the use of steerable introducer sheath or the sets with a more flexible connection between the implant and the insertion cable can also facilitate the closure of a large defect with a deficient retroaortic rim. It should be emphasized that it is not recommended to close defects with a deficient or absent retroaortic rim with no superior rim.

The deficient posteroinferior rim occurs in 3.3% of patients with secundum ASD. Percutaneous closure of this defect is feasible but it is associated with an increased risk of device embolization. Even if the device is well implanted and initially stable, it can slip to the IVC, which usually takes place a few hours after the procedure. In rare situations, cyanosis can appear after the procedure, despite stable position of the implant. This happens because the implant's straddling over the IVC may lead to a right-to-left shunt to the LA. Due to the difficult visualization of posteroinferior rim with the transesophageal echocardiographic guidance, defect assessment and monitoring of the procedure with intracardiac echocardiography is recommended. Since the percentage of complications may be significant, the percutaneous closure of secundum ASD associated with posteroinferior rim deficiency should rather be avoided.[61,67]

In the case of a posterior rim deficiency, the feasibility of percutaneous closure of ASD II depends on the extent of the defect. If it reaches the border with the IVC, device closure should be avoided due to the significant risk of embolization. It is extremely important to distinguish the deficiency of posteroinferior rim from the deficiency of posterior rim. While there is a significant risk of embolization with the device closure of the former, percutaneous treatment is appropriate for the latter.

The deficient posterosuperior rim rarely accompanies ASD. Percutaneous closure of such defect is feasible, but one should be extremely careful, because sometimes it is directed towards the deficient retroaortic rim, and this poses a significant risk of atrial wall erosion.

Only secundum ASD should be closed percutaneously; however, in recent years, there have been few reports of transcatheter closure of superior sinus venosus-type ASDs using covered stents, however such procedure is still controversial. The use of 3-dimensional reconstruction of the heart with subsequent 3D printing may be extremely helpful in planning such procedure.[68]

Some authors believe that finding one deficient rim is generally not a significant problem if the opposing rim is well developed.[69] Although percutaneous closure of ASD II with deficient rims is feasible in many cases, this technique should not be recommended in most cases except in cases of deficient retroaortic rim.

Short review of the devices available in Poland Transcatheter occlusion of ASD by the double umbrella system was first described by King et al in 1974.[34,70] Various devices have been used in transcatheter occlusion of these defects with different outcomes. The differences in

TABLE 2 Comparison of commonly used devices for atrial septal defect closure available in Poland

Device	Company	Construction	Connection system	Available sizes	Delivery system	Number of sizes	Comments
Amplatzer Septal Occluder	Abbott, United States	Nitinol double discs	Micro thread	4–38 mm	6–12 F	26	–
Figulla Flex II	Occlutech GmbH, Germany	Nitinol double discs	Unique flexible ball connection	4–40 mm	7–12 F	20	Titanium oxide--covered nitinol wire
Cera, CeraFlex	LifeTech Scientific Co, China	Nitinol double discs	Micro thread / premonted flexible connection	6–42 mm	7–14 F	19	Titanium nitride--coated nitinol wire
Cocoon Septal Occluder	Vascular Innovations Co, Thailand	Nitinol double discs	Micro thread	8–40 mm	7–14 F	17	Nitinol wire nanocoated with platinum
Hyperion	Shanghai Shape Memory Alloy Co, China	Nitinol double discs	Micro thread	6–42 mm	8–14 F	19	Preoxidized nitinol wires
MemoPart ASO	Lepu Medical Co, China	Nitinol double discs	Micro thread	6–42 mm	8–14 F	26	–
Nit-Occlud ASD-R	PFM Medica, Germany	Double-disc "reverse configuration" of the single--nitinol-layer on the LA disc	"Snare-like" central locking wire and a pusher with a distal wire noose; premonted	8–30 mm	8–14 F	12	1 piece of nitinol wire without any connecting elements
Ultrasept II ASD Occluder	Cardia, Minneapolis, United States	Nitinol wire frame forms 2 sails with a self-centering mechanism	Flexible bioptom–like connection	6–34 mm	9–11 F	15	Covered by a polyvinyl alcohol membrane

Abbreviations: LA, left atrial; others, see FIGURE 1

results concerned complication types and rates. Their main problems were low closure efficacy (residual leaks), unstable device position (migrations), and fracture of its elements. A breakthrough came with using nitinol (a superelastic metal alloy of nickel and titanium) to build the skeleton of the Cardio-SEAL device (a modification to the Clamshell device). The next alteration was using a self-centering device (Star--Flex). Thus, transcatheter device retrieval before its release and reposition of the device become possible.

The perfect ASD closure device should be the one that completely closes the defect with the minimal risk of complications, the device is easily implantable and can be repositioned and retrieved. As of yet, no device on the market meets all of these criteria. Most of currently available devices show excellent closure efficacy and quite comparable outcomes; however, they have their own advantages and disadvantages.[71-80]

There have been a couple of ASD closure devices available on the Polish market over the past years (TABLE 2; see Supplementary material for a detailed description).

Potential complications of atrial septal defect closure

A successful closure of ASD is reported in about 98% of patients.[76,81,82] The prevalence of residual shunt is decreasing over time after the procedure, and it is present in majority of patients immediately after the implantation. Residual shunt is present in only 1% and 2% of patients 2 years after ASD closure.[76,82,83] Total complication rates vary and they are estimated to be between 2.2% and 8.6%.[71,84-86] Periprocedural death is reported incidentally, mostly secondary to other procedure-related major complications.

Complications of percutaneous ASD closure include: device dislodgement, cardiac structure damage or erosion, device impingement on valves, veins, or other vessels such as the aorta, new onset atrial arrhythmia, atrioventricular block, and thromboembolism arising from thrombosis on the device, air embolism, access--site hematoma and bleeding (TABLE 3). The frequency of complications depends on the experience of the center, number of performed procedures, patient selection, and—less importantly—type of the implanted device.

Device dislodgement Device dislodgement, with subsequent embolization, occurs usually within 24 to 48 hours after the procedure with the incidence rate of 0.2% to 1.67%.[71,82,84,86-89] However, it can also occur sporadically within several months or later after ASD closure.[71,89] The most common reasons for occluder dislodgement are a thin and aneurysmal septum, an inadequate

TABLE 3 Complications of transcatheter closure of atrial septal defect

Complication	Incidence, %	Onset	Clinical significance	Management
Device-related cardiac perforation	0.1	Mostly within 24–48 h, possible late occurrence (up to 3 y)	• Potentially lethal cardiac tamponade • Hemopericardium	• Heparin neutralization • Pericardial effusion drainage • Surgical intervention if required
Cardiac erosion	0.05–0.46	Majority within 96 h, probable during the first 6–8 mo, incidentally up to 9 y	• Potentially lethal cardiac tamponade • Hemopericardium • Aortic fistula	• Pericardiocentesis for pericardial effusion and tamponade • Surgical intervention
Device embolization	0.2–1.67	Mostly within 24–48 h, sporadically within several months	Depends on the place of dislodgement	• Anticoagulation • Percutaneous device retrieval • Surgical retrieval if required
Device impingement	Extremely rare	Up to several weeks, usually due to device shift or wire fracture	• Severe mitral valve regurgitation • Pulmonary edema	• Replacement of the device with a smaller device if possible • Surgical intervention
Device thrombus	1.2	Mostly during 1–6 mo	Cardiovascular embolic events	• Anticoagulation • In rare cases, surgical intervention
Atrial tachyarrhythmias	1.3–5	Increased in a periprocedural period, subsides over time	• Hemodynamic compromise • Thromboembolic events	According to event-specific guidelines
• Conduction abnormalities • Advanced heart block	Below 1	Typically in the first 24 h, or hours and days later, then resolve over time	Hemodynamic compromise	• In case of durable advanced block, device removal (early phase) • Pacemaker placement if late-onset advanced heart block
• Access-site complications • Groin hematoma, bleeding	1–3	Up to 24 h after the procedure	Anemia, local pain	Usually conservative
Nickel allergy	Unknown	24 h–1 mo	Headache, rash / urticaria, fewer, palpitations, difficulty breathing	Medical therapy with steroids, antiallergic agents, in rare cases, device explanation

or floppy rim, ellipsoidal shape of the defect, greater defect size (>30 mm), device mobility postimplantation, and operator-related technical issues, especially the use of an undersized ASD device.[90] The most of dislodgements take place into the main pulmonary artery, LA or RA, ascending aorta, and RV; however, devices can be located also at the LV, descending aorta, abdominal aorta, iliac bifurcation, and iliac arteries.[89,91,92] To prevent device embolization, it seems reasonable to use a device 1 to 2 mm greater than a "stretched diameter" of the defect. To prevent embolization of the occluder, it is very important to ensure a proper and stable position of the device by the so-called push–pull maneuverer (Minnesota maneuver) before device detachment. However, if the device dislodges (especially to the atria, pulmonary artery, or aorta), endovascular retrieval is possible with a goose neck snare or loop snare, biopsy forceps, or other retrieval-dedicated devices. A sheath size at least 2 to 4 F larger than the sheath that was used to deliver the device should be used. In an Amplatzer or Amplatzer-like device, pulling out is usually feasible by grabbing a female screw site of the right atrial disc.[92] The primary objective is to bring the device out of the heart into the IVC and then out from the body through the femoral vein. A large stiff-tip braided sheath works best.[92] A second venous access may be used to hold the device before snaring the occluder. If the device is in one of the ventricles, especially trapped in the chorda, retrieval may be difficult and it is reasonable to refer the patient to surgery, where both device retrieval and the defect closure could be performed.[92] According to available data, surgical intervention is required in about one-fourth of device embolization cases.[89] It should be emphasized that all operators who perform percutaneous ASD closure should be prepared for percutaneous device retrieval in the event of device embolization.[88]

Cardiac perforation and erosion Cardiac perforation, which is mostly a catheter-related injury during the procedure, is a very rare complication (0.1%) manifesting usually as cardiac tamponade.[88] It requires immediate heparin neutralization and decompression by pericardial puncture. If intrapericardial bleeding persists, surgical intervention is required. Small,

negligible pericardial effusion is sometimes visible during or after the procedure, especially in prolonged implantations. The mechanism is unknown and a complete resolution of the effusion is observed within 3 weeks.[71]

Cardiac erosion seems to be a complication that is Amplatzer-device specific following transcatheter closure of ASD. The rates of erosion ranged from 0.05% to 0.46%.[76,86-88] Cardiac erosions might appear in less than 24 hours after the device implantation; however, most of them appeared within 6 months but they could be observed as late as even 9 years after the procedure. Cardiac erosion could lead to cardiac tamponade or aortic fistula. It was observed that 90% of patients with erosions had deficient aortic rims. The motions of the device relative to the aortic root were the main possible mechanisms of cardiac erosion. Avoiding oversized devices seems to be a crucial factor to prevent cardiac erosions after defect closure.[88,92,93]

Air embolism Air embolism may be responsible for transient electrocardiogram changes, including ST segment depression or ST segment elevation, sometimes accompanied by chest pain and rhythm disturbances. Usually, it is transient and do not require intervention. In very rare cases with persistent ST elevation, coronary angiography may be necessary. Prevention of air embolism requires multiple careful saline flushes of the device, the loading system, and the delivery sheath.[66,88,93]

Device impingement Device impingement on valves, veins, or other vessels should be avoided by careful attentive inspection by TEE during the implantation.[71,88] In patients with atrioventricular valve rim deficiency, the edge of the device may interfere with the anterior mitral valve leaflet and result in mitral regurgitation. In case of the impingement with cardiac structures, reposition of the device may be helpful or the exchange to a smaller device size (if the size of the defect allows it), otherwise surgical ASD closure should be considered.[88,94]

Arrhythmias and conduction abnormalities The reported complications from device closure of ASD include development of atrial tachyarrhythmias or advanced atrioventricular (AV) block. Atrial arrhythmias including extrasystole, supraventricular tachycardia, atrial flutter, or atrial fibrillation may occur in up to 5% of procedures.[88,92,95] In majority of cases, they are transient and do not require treatment. In supraventricular tachycardia, atrial flutter, or fibrillation, routine management according to guidelines is recommended.

Advanced or complete AV block can occur sporadically (below 1%) as early as at the time of the procedure and device deployment or hours, days, or even years later.[86-88,92,96] Risk factors include young age and large defect / device size. Generally, it is believed to be caused by compression of the AV node or inflammatory reaction to a foreign body and scarring at the level of the triangle of Koch caused by the device.

Most of the atrioventricular blocks are transient. There were attempts to treat with corticosteroid but there are no controlled trials for AV block caused by device implantation.[92] Durable or late-onset complete AV blocks require pacemaker implantation.[92]

Thrombus on the device Studies showed that the incidence of thrombus on the device is about 1.2%.[97] However, significant differences were noted between different device types. Older generation devices (Amplatzer septal occluder, Starflex, Cardioseal) have higher incidence of thrombus formation as compared with more recent devices.[93] The lowest rate of thrombus was reported with the Amplatzer nitinol wire frame filled with polyester fabric and the Helex nitinol wire covered by an ultra-thin membrane of expanded polytetrafluoroethylene.[93] Significant risk factors for thrombus formation were atrial fibrillation and atrial septal aneurysm, which had remained after device closure. In most patients, the thrombus resolved after medical therapy with heparin or oral anticoagulants without adverse ischemic events.[84,89] Only few cases required surgical removal of thrombus.[88,93] Although antiplatelet therapy has been recommended for 3 to 6 months after the procedure, the time frame of thrombus formation ranged from 4 weeks to 7 years after implantation. Early neurological complications after transcatheter ASD closure can be resolved by heparin infusion.[98] Late incomplete endothelization of the device with subsequent clinical consequences is also possible.[99]

Nickel allergy Nickel is a contact allergen, and an allergic reaction may occur in case of implantation of a nitinol-containing device. The frequency is not known; however, women present more often. The reaction occurs from 2 days to 1 month after the implantation and manifests as headaches, rash / urticaria, difficulty breathing, fewer, palpitations, and rarely pericardial effusion.[88,92,100] Fortunately, all patients respond to medical management by steroids and antiallergenic agents[100,101] and symptoms subside in a few months. In rare cases, if medical management is ineffective, the device should be explanted.[88,100,101] A nickel skin test is available and may be performed in patients with allergy or those "suspected."[74] In case of allergy, platinum-coated device may be considered.

Access-site related complications Final, yet equally important, complications are groin

hematoma and bleeding episodes following ASD closure. Groin hematoma is reported in up to 5% of patients, more frequently in women. It can be avoided by the "Z-shaped" suture technique of the skin, followed by prolonged local compression on the groin. In cases, of severe bleeding episodes, treatment should be applied in accordance to the guidelines on the management of patients referred to invasive cardiology procedures.

The most frequently used occluder in ASD closure is Amplatzer device. In recent years, several Amplatzer-like occluders have been registered and introduced into practice. Recent evaluation of 3 devices (Amplatzer, Figulla, Cera) showed no significant differences in efficacy and safety in short and mid-term follow up.[102] Also other Chinese and Thai nitinol occluders seem to be effective.[73,74]

Percutaneous closure of atrial septal defect in children: procedure details Atrial septal defect leads to volume overload of the right chambers of the heart and an increased Qp. These changes proceed gradually, making it possible to postpone the decision to operate and choose when best to do it (usually when the child is 3–4 years old). Sometimes, however, the symptoms of the defect appear faster, are more severe, and the degree of hemodynamic disturbances justifies operating sooner. Atrial septal defects can change as children grow. Small ASDs can close spontaneously. The main predictor of spontaneous closure is the initial ASD diameter of less than 8 mm.[103] On the other hand, the defects can also increase in size as the child grows. McMahon et al[104] reported a 50% increase in size of ASDs in two-thirds of patients. In some patients the rate of the size increase amounted to up to 0.8 mm per year.[104]

It is of extreme importance to monitor the changes in defect size and its hemodynamic consequences. Choosing the best time of qualification for ASD closure is of particular significance. Treating defects that are not increasing in size as the child grows may be postponed. As time passes, the hemodynamic and anatomical situation becomes increasingly favorable for the safety of the procedure. The decision to close an ASD that increases in size as the child grows should be made without a delay. Postponing the decision may cause an increase in the ratio of ASD size to child body mass. This in turn may make percutaneous procedure impossible. Apart from shortening the heart's exposition to adverse hemodynamic conditions, the additional benefit of deciding on the procedure sooner is the use of a smaller closure device. The eligibility criteria for ASD treatment in young children are difficult and unclear. It is not fully known when treatment is necessary and what its benefits are.

In the pediatric population, the size of the defect should be assessed in relation to patient's body mass when considering the probability of successful device closure. A defect that can easily be closed percutaneously in an adult may disqualify a child from this treatment method due to small heart size (especially the interatrial septum) and the proximity of the surrounding structures. Petit et al[105] demonstrated that good outcomes are obtained in patients with a ratio of defect to body mass of less than 1.2. However, closure of much larger defects where the ratio was equal to 2.3 was also shown to be possible.[106]

There is a natural tendency toward closing ASDs in more challenging clinical scenarios and larger defects in smaller patients. Lim et al[107] conducted an effective percutaneous ASD closure in a 2.8-kg preterm newborn.[107] There are numerous accounts of ASD closure in infants and very small children. Although percutaneous ASD closure is feasible even in very small infants, it requires extensive experience from the interventional team (especially in case of low body mass or large defects).[108] Treatment of large ASDs in small children has a relatively low procedural success rate and carries a significant risk of procedural and delayed complications.[109,110] Percutaneous defect closure in children is difficult because of the small blood vessel diameter that complicates the procedure. Devices with the smallest size of delivery systems are therefore preferred, as they minimize the risk of damage to the vessels.

Before a small child qualifies for treatment, all risks and benefits must be evaluated. A good echocardiographic visualization of children makes it possible to conduct the procedure under TTE control without the need for TEE. For obvious reasons, however, the procedure always requires general anesthesia.

After closing the defect with the device, the size of the enlarged right heart chamber normalizes quickly. As the child grows, there is no problem with the development of heart structures, interatrial septum included. Though the septum is stiffened at the implantation place, it retains its potential to grow in the part not covered by the device.

Long-term follow-up observations of children post percutaneous ASD closure show no difference in the type and number of long-term complications in comparison with the general population.[110]

Follow-up recommendations After percutaneous ASD closure, TTE should be performed at 24 hours after implantation to ensure that there is no significant pericardial effusion.[1,4] Chest pain or syncope may suggest device erosion or migration and should be evaluated without a delay.[71] After percutaneous defect closure,

early (1–3 months) and intermediate (1 year) follow-up is recommended with echocardiography. Periodic follow-up is required thereafter, every 2 to 4 years.[1,111,112] Follow-up evaluation should include TTE with the assessment of a potential residual shunts, RV size and function, tricuspid regurgitation, and PAP.[97,113-115] Patients with residual shunt, elevated PAP or arrhythmias and those repaired at adult age (particularly >40 years) should be followed on a regular basis, preferably in specialized centers.[1,116,117]

After percutaneous ASD closure, careful assessment of arrhythmias by history, ECG, and if necessary, Holter monitoring should be performed.[113,117] Among late post-operative arrhythmias after ASD closure in the younger population (age <40 years) the most frequent are intra-atrial re-entrant tachycardia or atrial flutter, which can be successfully treated with radiofrequency ablation. In the older adult population (age >40 years), atrial fibrillation becomes more common and may require antiarrhythmic therapy or ablation. Access to the LA may be, however, restricted after device closure. Patients with atrial fibrillation should receive oral anticoagulation. Sick sinus syndrome or heart blocks are less common, but can appear even up to 4 years after implantation.[1,4,93,96]

Patients successfully repaired under the age of 25 years (no residual shunt, normal PAP, normal RV, no arrhythmias) do not require regular follow-up.[1,4,93,117] However, they should be informed about the possible late occurrence of tachyarrhythmias.

Medical therapy Dual antiplatelet therapy with acetylsalicylic acid and clopidogrel is recommended for at least 3 months with a single antiplatelet drug until at least 6 months after transcatheter closure of ASD.[1,2,6,12]

Infective endocarditis prophylaxis Infective endocarditis prophylaxis is recommended for 6 months after ASD device closure.[1,4,113,118] Infective endocarditis can be diagnosed in case of incomplete endothelization even late in follow up.[99]

Exercise / sport There are no contraindications or restriction to any kind of sports in asymptomatic patients with ASD without PH, significant arrhythmias, or RV dysfunction. Patients with PH should limit low-intensity recreational sports. In the case of arrhythmias during physical exercise, the intensity of effort should be limited to the level that does not cause symptoms.[119]

Pregnancy and contraception In ASD patients without PH, pregnancy-related risk is low. Closure of ASD performed before pregnancy may prevent worsening of patients clinical status and help to avoid paradoxical embolism. Pregnancy is contraindicated in patients with severe PH or Eisenmenger syndrome.[114-120] The recurrence rate of congenital heart diseases is 3% to 10% (excluding familial ASD and heart–hand syndromes with autosomal dominant inheritance).[120] There are no contraindications to any type of contraception in patients with ASD.[1,119,120]

Recommendations for training physicians to perform atrial septal defect closure To ensure optimal results, ASD closure should be performed in experienced centers that routinely perform other structural heart interventions, as it was advocated in previously published PFO guidelines of the same group.[121] Data from numerous registries regarding ASD and PFO closure procedures show that both individual operators and centers with a small number of procedures performed annually have worse treatment results.[122] Trainee operators should have the theoretical knowledge and technical skills required to safely perform this procedure to ensure a low number of complications.

The group of experts from the Association of Cardiovascular Interventions and the Grown-Up Congenital Heart Disease Section of the Polish Cardiac Society recommends that the training physician should acquire theoretical knowledge regarding the indications for procedure, patient preparation, practical experience with the equipment, proper selection of the device size, conduction of the procedure, and peri- and postprocedural pharmacotherapy. It is also necessary to learn about the possible complications of the procedure, their prevention, and treatment. After theoretical and practical training in a center with extensive experience (at least 3 sessions), the training operator should perform a minimum of 20 ASD closure procedures independently but under the supervision of an experienced operator (proctor).[123,124] However, based on experts' opinion, the presence of cardiac surgery on-site is not required in centers which intend to start ASD / PFO closure procedures program.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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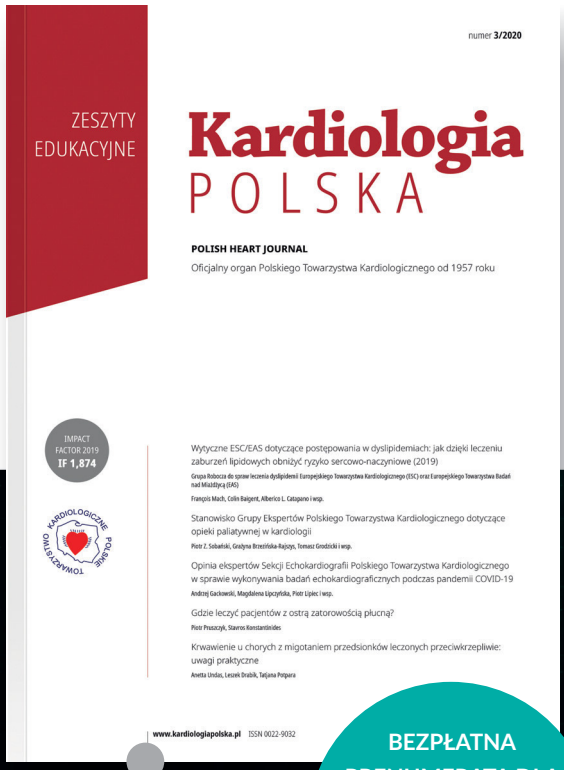

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