

# Symptomatic tachy- and bradyarrhythmias after transcatheter closure of interatrial communications with Amplatzer devices

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### **Abstract**

**Background:** The aim of this paper is to present our own experience related to frequency of symptomatic tachy- and bradyarrhythmias in patients after transcatheter closure of interatrial defects (ASD) and patent foramen ovale (PFO) using Amplatzer plugs.

**Methods:** Transcatheter closure of interatrial communications with Amplatzer devices was carried out on 739 patients in our center. Only patients with new symptomatic arrhythmias (who required pharmacotherapy, cardioversion or pacemaker implantation) were included in to the study. All patients who had had arrhythmias prior to ASD closure, such as supraventricular tachycardias (SVT) or atrial flutter/fibrillation (AF), were excluded.

Results: New tachy- and bradyarrhythmias after implantation of Amplatzer devices were observed in 11 patients (1.5%). There were 9 patients (mean age 36.7 years) with atrial tachyarrhythmias (AF in 8 and SVT in 1 patients), which occurred between the first day and 3 months after implantation. Seven patients were treated initially by pharmacotherapy; in 2 of them sinus rhythm returned just after cardioversion. In other 2 patients cardioversion was performed as an initial therapy. In none of these patients, but one recurrence of tachycardia was observed; however, 7 of them had pharmacotherapy prolonged up to 1 year. In 2 patients, aged 15 and 16, complete atrioventricular (AV) block was observed 4.3 and 1.5 years after Amplatzer implantation, respectively. In the first patient intermittent second-degree AV block (Mobitz II) was observed before ASD closure. In both patients, a DDDR pacemaker was implanted.

**Conclusions:** Transcatheter closure of ASD using Amplatzer devices is associated with a risk of new atrial tachyarrhythmias (usually early after the procedure and in older patients). The risk of conduction disturbances such as complete heart block, which can occur in late follow-up, is low. Thence, close long-term follow-up of these patients is obligatory. (Cardiol J 2008; 15: 510–516)

Key words: atrial septal defect, transcatheter closure, arrhythmias

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## Introduction

Cases of arrhythmia after transcatheter closure of interatrial communications are believed to be a rare problem of minor clinical importance [1]. In general they are mostly observed during the implantation itself, and any deeper analyses of the subject relate mostly to small series of patients.

The aim of this paper is to present our own experience relating to the frequency of symptomatic tachy- and bradyarrhythmias in patients after transcatheter closure of interatrial defects (ASD) and patent foramen ovale (PFO) using Amplatzer plugs.

## **Methods**

The data of 739 patients, in whom an attempt of transcatheter closure of interatrial communications using Amplatzer plugs (AGA Med., Plymouth, MN, USA) had been made, were analyzed. The procedures were performed between October 1997 and June 2008. In 683 of them, ASDs were closed with an Amplatzer Atrial Septal Occluder (ASO) or Cribriformis ASO (Crib ASO). The latter was used in cases of multi-perforated defects. In 56 patients with cryptogenous central nervous system strokes the PFO was closed using the Amplatzer PFO Occluder (APFO). The intervention was successfully accomplished in 712 (96.3%) patients. The reason for the abandoned intervention in 27 patients with ASD was improper ASO position (mostly in cases of missing anterior-superior and floppy posteriorinferior rim of the septum, but in no case because of cardiac arrhythmia). The interventions were done in a routine way using the methods described previously [2]. Data of ambulatory and eventual hospitalization files were analyzed for all patients. Follow-up observation included ambulatory controls within 1, 3, 6 and 12 months of intervention and then yearly. The scope of interest covered any symptomatic arrhythmias: bradyarrhythmias (history of weakness, syncope, unconsciousness) or atrial tachyarrhythmias. The latter included events causing substantial and lasting acute heart action acceleration which required pharmaco- or electrotherapy (cardioversion). These included acute supraventricular tachycardias (SVT) as well as atrial flutter/fibrillation (AF) with fast ventricular action occurring after implantation of the Amplatzer. Patients having durable or intermittent atrial arrhythmia (SVT, AF) prior to ASD closure and those with symptomless arrhythmia or rhythm disturbances such as right His bundle branch block, grade I atrio-ventricular (AV) block, supraventricular extrasystole, etc. were excluded from the study.

In each case of symptomatic tachy- and bradyarrhythmia the patient's data records prior to transcatheter closure of ASD/PFO were analyzed: occurrence of arrhythmia and/or conduction disturbances, demographic data (age, body mass, height, gender), grade of right ventricle enlargement, ASD diameter or length of PFO channel in the transesophageal echocardiographic examination (TEE) and stretched diameter. The course of procedure (including the presence or absence of arrhythmia and/or conduction disturbances during implantation), size and type of applied Amplatzer device, and device/height ratio (D/H) were also analyzed. The latter parameter was not calculated in cases of Crib ASO or APFO (for technical reasons due to the narrow waist of the implant). When arrhythmias or conduction disturbances occurred, it was established how they were revealed, their nature, how they were treated (efficiency of pharmacotherapy and cardioversion in case of tachyarrhythmia) and whether re-occurrences took place.

The study was approved by the local bioethical committee and all patients gave their informed consent.

## Results

Some new and symptomatic arrhythmias and conduction disturbances not observed prior to the intervention were found in 11 patients aged from 3.5 to 56 (mean 36.7) years. In 9 cases they were acute and durable atrial tachycardias. In 2 girls (aged 15 and 16 years) symptomatic complete atrio-ventricular block (CAVB) occurred. Clinical details of all patients are shown in Table 1.

The analyzed material included 10 patients with ASD and 1 with PFO. Among the patients with ASD, 9 were treated with ASO (sized 9–40 mm) and 1 with 35 mm Crib ASO. In a single patient with PFO a 35 mm APFO was applied. In all patients with ASD the enlargement of the right ventricular cavity (100–160% of body mass norm) was found. The D/H ratio exceeded 0.2 in 1 case. There were no cases without any arrhythmia or conduction disturbances found during Amplatzer implantation.

Of the 9 patients with symptomatic atrial tachyarrhythmias, 8 of them were found to have AF and 1 was found to have SVT. Tachyarrhythmia occurred in 4 patients on the first day, in 3 patients in the third week, in 1 patient during the second month and in another 1 in the third month after the intervention. In all of those patients stimulation of the vagus nerve (Valsalva test etc.) was applied initially. As no

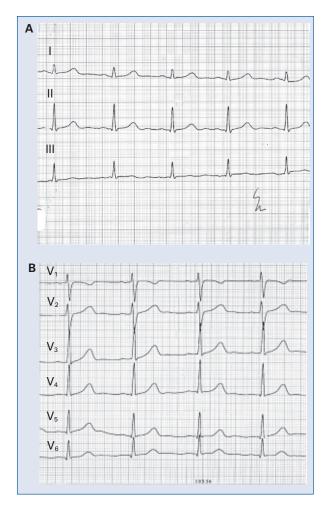
**Table 1**. Clinical findings of patients who developed symptomatic tachy- or bradyarrhythmias.

Pts. (No.)	Age [years]	Implant date	TEE/balloon diam. [mm]	Device [mm]	D/H ratio	RVD (%)	Rhythm disturb.	Time of appear.	Therapy	Comment
<del>-</del>	19	IX 2000	ASD 9/11	15 ASO	0.1	110	AF	3 weeks	Propafenon, CV	Atenolol
2.	10	XII 2003	ASD 8+3/15	15 ASO	0.11	160	CAVB	4.3 years	DDDR	Mobitz II (before)
	47	VIII 2004	ASD 13/22	22 ASO	0.14	130	AF	3 weeks	Propafenon	Propafenon
4.	48	VI 2005	ASD 11/21	22 ASO	0.13	140	AF	1st day	Propafenon, CV	Propafenon
2.	14	VII 2005	ASD 10/16	16 ASO	0.1	120	CAVB	1.5 year	DDDR	Normal ECG
9.	<b>∞</b>	VII 2006	ASD 7/-	9 ASO	0.1	130	SVT	1st day, 4 months	Adenecor, atenolol	Atenolol after second event
7.	26	1 2007	ASD 30/37	40 ASO	0.25	140	AF	3 weeks	Propafenon	Propafenon
ω.	42	X 2007	ASD 12/17	17 ASO	0.1	110	AF	3 months	CV	Propafenon
9.	54	IV 2008	ASD A/M	35 Crib.	ı	100	AF	2 months	Betaloc	Betaloc
10.	3.5	V 2008	ASD 15/19	19 ASO	0.19	130	AF	1st day	Amiodaron	Without medication
17	53	VI 2008	PFO + A	35 APF	1	06	AF	1st day	CV	Without medication
Age —	age during im	plantation; Impl	ant date — date of imp	lantation of the d	evice; TEE/k	balloon diam	. — diameter of	ASD in transesophageal ech	ocardiographic examination and	Age — age during implantation; Implant date — date of implantation of the device; TEE/balloon diam. — diameter of ASD in transesophageal echocardiographic examination and stretched; Device — implanted device

Age—age during impartate micro and progress of the part of the upper limit for the age). Bythm disturbances observed; Therapy — therapy of rhythm disturbances observed; Therapy — therapy of rhythm disturbances after the procedure; Therapy — therapy of rhythm disturbances; Softal Occluder; Crib. — cribriformis ASO, APF — Amplazer PFO Occluder; CAVB — complete atrio-ventricular block; AF— atrial flutter/fibrillation; SVT— supraventricular tachycardia; CV — cardioversion; A— aneurysm; M— multiple perforations

effect was noticed, pharmacotherapy (adenosine, propafenon or amiodarone, dosed intravenously) was applied. In 2 patients the return of sinus rhythm was not observed before cardioversion was applied. In the next 2 patients cardioversion was applied as a primary treatment. No recurrence of tachyarrhythmia was observed in these patients (except for one). In the case of an 8-year-old girl (Table 1 patient No. 6), within the first day after ASO implantation SVT occurred and had to be managed by an intravenous adenosine infusion. SVT repeated within the fourth month and was treated by an intravenous dosage of propranolol. In this case the beta-blocker therapy was continued for 8 months, and within the subsequent period of observation (covering 1 year after the last attack) no SVT recurrences were observed. An extended (up to 1 year after intervention) anti-arrhythmic treatment was also applied in 6 other patients (Table 1 — patient No. 1, 3, 4, 7, 8, 9).

Atrio-ventricular block occurred in 2 female patients aged 15 and 16 years. It occurred 4.3 and 1.5 years after ASO implantation, respectively (Table 1 — patient No. 2, 5). In both cases it revealed by the presence of repeated weakness and syncope reported in the clinical history. In the first patient, a 10-year-old girl, conduction disturbances were already found prior to implantation — an intermitting second-degree AV block of Mobitz type II. She had 2 defects at a small distance (in TEE with diameters of 8 and 3 mm). The size of the stretched defect (a larger one) was 15 mm and such ASO was used to obtain a complete closure of both ASDs (D/H ratio = 0.11). During subsequent observation no previously diagnosed AV block progression was observed until clinical symptoms presented (mentioned above) when intermittent AV block of grade III was found. That was the reason for pacemaker implantation (DDDR). In another girl, neither arrhythmia nor conduction abnormalities were found before or after ASO implantation (Fig. 1A, B). Her ASD size in the TEE was 10 mm (Fig. 2A) and the stretched diameter was 16 mm (Fig. 2B). For this patient a 16 mm ASO was applied (Fig. 2C) and her H/R ratio was 0.1. During the first 3 months after implantation the girl complained of periodical headaches and scotoma. In the control tests the sinus rhythm was maintained; 1.5 years after ASO implantation the patient was admitted to our department with a symptomatic third-degree intermitting AV block. The electrophysiological test was normal. In 24-hour ECG (Holter) recording CAVB with breaks of up to 7 s (Fig. 3) was confirmed. After implantation of a DDDR pacemaker the child has remained asymptomatic for 1 year.

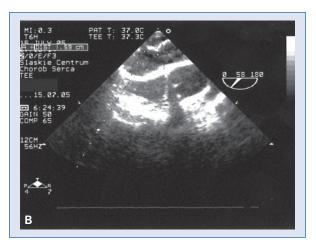


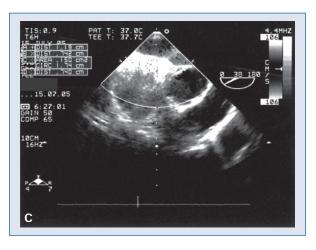
**Figure 1.** Normal ECG of 14-year-old girl after percutaneous procedure closure of atrial septal defect (**A**) limb leads (**B**) precordial leads.

## **Discussion**

Until now, the presented series of patients in whom ASD was closed using ASO and in whom the frequency of rhythm disturbances was analysed consisted of from 23 to 423 patients [1–6]. Chessa et al. [7] observed arrhythmia within the early postprocedural period in 2.6% of patients (11/417). In our material the occurrence of symptomatic tachyarrhythmias was lower at 1.3% (9/708), but cases of benign temporary heart palpitation without clinical manifestation were excluded. It must be emphasized that reported symptomatic tachyarrhythmias occurred within an early postprocedural period: in 4 patients within the first day and in the next 5 after hospital discharge (from 2 weeks to 4 months after intervention). Their incidental nature is also important (there was only one case of recurrence), although in 7 patients antiarrhythmic pharmacotherapy up to 1 year after intervention was continued. They

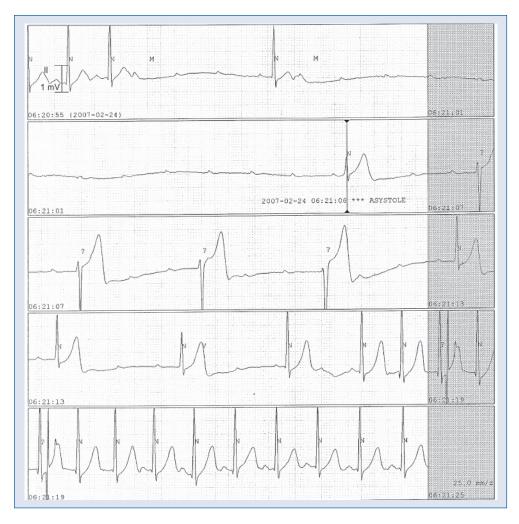






**Figure 2. A.** Atrial septal defect of 14-year-old girl — transesophageal echocardiographic examination: 10 mm of diameter; **B.** Stretched diameter of interatrial defects — 16 mm (balloon calibration); **C.** Amplatzer Atrial Septal Occluder of 16 mm after implantation.

occurred only in 2 cases in children (aged 3.5 and 8 years) and in 6 adults over 40 years old (in our material 40% of the treated patients were adults). According to our observations the appearance of



**Figure 3.** The same patient — Holter ECG at the age of 16 years: intermittent third-degree atrioventricular block with 7 s pauses.

tachyarrhythmias could be connected with the application of large implants only in 2 patients. This was related to a 56-year-old man in whom 40 mm ASO was applied (D/H ratio 0.25). Atrial flutter/fibrillation with tachycardia appeared 3 weeks after intervention. In a 3.5-year-old boy, a 19 mm ASO was used (D/H ratio 0.19) and supraventricular tachyarrhythmia appeared during the first day after the procedure. No other factors (such as localization of ASD, magnitude of right ventricular dilation, etc.) predisposing to such complication were found.

Our material also included 2 patients with symptomatic bradycardia caused by CAVB. In both, rhythm disturbances occurred in a late period after intervention — 4.3 and 1.5 years after implantation, respectively. In the first case of a 10-year-old girl, a second-degree AV block occurred even before the intervention, so it can be supposed that the occurrence of a CAVB was caused by the progressive

nature of conduction abnormalities and there was no dependence on whether or not the Amplatzer was implanted. In the second case, involving a 14-year-old girl, the closure was performed with a 16 mm ASO and no rhythm disturbances were found prior to implantation. It is generally found that AV blocks, when observed during or after ASO implantation, are temporary and reversible. Suda et al. [5] found that their more frequent occurrence is associated with implantation of ASO > 19 mm, a device/ /height ratio over 0.2 and a larger left-right shunt through the ASD (Qp/Qs > 2.5). Such observations were not confirmed in either of our 2 cases. We did not determine the pulmonary/systemic flow ratio because its credibility has recently been under question [8], and in our opinion the right ventricle overload is more substantial (results are presented in Table 1). Potential mechanisms of conduction abnormality after ASO implantation may be caused

by pressure and/or rubbing of the implant's disk on the artrio-ventricular node, a local oedema or progressive scar formation. The available literature includes only single cases demonstrating the necessity to implant a pacemaker due to a CAVB after ASD closure with an Amplatzer. Hill et al. [6] reported the case of a 6-year-old boy who needed a pacemaker implantation after closure of a large ASD using a 24 mm ASO. In that case, even prior to intervention, the Holter test showed short nodal rhythm episodes. The next patient in whom a pacemaker had to be implanted was a 40-year-old woman who experienced a permanent left His bundle branch block and acute atrial fibrillation prior to intervention. Transcatheter ASD closure was performed with a 24 mm ASO according to stretched diameter. After 2 months, acute atrial fibrillation occurred with the necessity of intensive anti-arrhythmic pharmacotherapy. Within the third month after implantation the patient was admitted to the hospital because of syncope and subsequent shock caused by advanced grade II AV block. An endocavitary electrode was implanted in an urgent mode and it caused efficient stimulation. As the block was progressing (up to CAVB), after a week a permanent pacemaker DDD was implanted [9]. In the literature some situations are reported when a CAVB after ASO implantation has resolved after a long corticotherapy [10] or spontaneously soon after implantation [5, 11]. Chessa et al. [7], after implantation of an 18 mm ASO in a 7-year-old boy, observed a complete AV block. The implant was withdrawn and sinus rhythm restored. After a year, a 14 mm ASO was implanted successfully without any conduction disturbances. The same author, in his comprehensive analysis of complications after transcatheter ASD closure, reports a 29-year-old patient who died suddenly 1.5 years after implantation of an 18 mm ASO. This case might have been caused by a late AV block, as happened to our patient. Unfortunately, there is no evidence in the form of ECG records, and no autopsy was performed.

It must be mentioned that there are reports showing a relationship between surgical closure of ASD and conduction abnormalities [12]. Recently, a Mexican group presented a report covering 460 patients of various ages, after surgical ASD closure. They found arrhythmia and conduction disturbances in 6.3% of patients prior to intervention and up to 14.31% afterwards. In 5 of them (1.2%), a CAVB occurred, but only in 1 case with a previous second-degree AV block [13]. In our material presented here (739 patients after transcatheter closure of interatrial communications using Amplat-

zer devices), serious conduction disturbances occurred only in 2 patients (0.3%). Recently a mutation in the NKX2-5 gene was found which causes CAVB. Half of such autosomally-inheriting cases refer to patients with ASD. Symptoms of this complex (ASD – CAVB) are usually revealed in early youth [14]. In the case of our patients, no such genetic tests were carried out.

### Conclusions

In the early period after transcatheter closure of interatrial communications using Amplatzer devices there is a risk of new atrial tachyarrhythmias, of a somewhat temporary nature and in older persons. The probability of late complete AV block after such treatment is low but exists, so precise follow-up of all patients is recommended.

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## References

- 1. Hessling G, Hyca S, Brockmeier K, Ulmer HE. Cardiac dysrhythmias in pediatric patients before and 1 year after transcatheter closure of atrial septal defects using the Amplatzer septal occluder. Ped Cardiol, 2003; 24: 259–262.
- Chan KC, Godman MJ, Walsh K, Wilson N, Redington A, Gibbs JL. Transcatheter closure of atrial septal defect and interatrial communications with a new self nitinol double disc device (Amplatzer septal occluder): Multicentre UK experience. Heart, 1999; 82: 300–306.
- Demkow M, Rużyłło W, Konka M et al. Transvenous closure of moderate and large secundum atrial septal defects in adults using the Amplatzer septal occluder. Catheter Cardiovasc Interv, 2001; 52: 188–193.
- Du Z-D, Hijazi ZM, Keinman CS, Silverman NH, Larntz K; for the Amplatzer Investigators. Comparison between transcatheter and surgical closure of secundum atrial septal in children and adults. J Am Coll Cardiol, 2002; 39: 1836–1844.
- Suda K, Raboisson M-J, Piette E, Dahdah NS, Miro J. Reversible atrioventricular block associated with closure of atrial septal defects using the Amplatzer device. J Am Coll Cardiol, 2004; 43: 1677–1682.
- Hill SL, Berul CI, Patel HT, Rhodes J, Supran SE, Cao Q-L, Hijazi Z. Early ECG abnormalities associated with transcatheter closure of atrial septal defects using the Amplatzer septal occluder. J Interv Card Electrophisiol, 2000; 4: 469–474.
- Chessa M, Carminati M, Buttera G et al. Early and late complications associated with transcatheter occlusion of secundum atrial septal defect. J Am Coll Cardiol, 2002; 39: 1061

  –1065
- Ahmed S, Lange RA, Hills LD. Inaccuracies of oximetry in identifying the location of intercardiac left-to-right shunts in adults. Am J Cardiol, 2008; 101: 245–247.

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- Kołodziej M, Sierżant M, Cieciorowski A, Janion M. Advanced symptomatic atrioventricular block as a complication after transcatheter occlusion of secundum atria septal defect in a 40-year-old woman with prior bundle branch block. Kardiol Pol, 2008; 66: 175–178.
- Lin SM, Hwang HK, Chen MR. Amplatzer septal occluder-induced transient complete atrioventricular block. J Formos Med Assoc, 2007; 106: 1052–1056.
- Celiker A, Ozkutlu S, Karakurt C, Karagoz T. Cardiac dysrhythmias after transcatheter closure of ASD with Amplatzer device. Turk J Pediatr, 2005; 47: 323–326.
- Sobrino JA, De Lombero F, Del Rio A et al. Atrioventricular nodal dysfunction in patients with atrial septal defect. Chest, 1982; 81: 447–482.
- Medinos A, Iturralde P, Marquez M et al. A permanent rhythm and conduction disturbances in patient surgically corrected atrial septal defect. Arch Inst Cardiol Mex, 2000; 70: 47–55.
- Smits JP, Veldkamp MW, Wilde AM, Mechanism of inherited cardiac conduction diseases. Europace, 2005; 7: 122– -123.