

Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal



Journal of Clinical and Basic Cardiology 2001; 4 (1), 35-38

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Nonsurgical Closure of Secundum Atrial Septal Defect and Patent Foramen Ovale

T. Chatterjee, St. Windecker, Ch. Seiler, B. Meier

In the past surgery has been the established therapy to treat a significant atrial septal defect. In the presence of patent foramen ovale with otherwise unexplained (cryptogenic) cerebral embolism, the therapy was oral anticoagulation or antiplatelet therapy. Surgery was considered only in case of recurrence. Percutaneous transcatheter occlusion of atrial septal defect and patent foramen ovale is a new valuable alternative to surgical closure. This report presents the current knowledge and our own data concerning nonsurgical closure of atrial septal defects and patent foramen ovale. *J Clin Basic Cardiol* 2001;4: 35–38.

Key words: patent foramen ovale, atrial septal defect, catheterization, paradoxical embolism

The first nonsurgical closure of a secundum type atrial septal defect (ASD) with a Dacron device was performed in 1973 in dogs, and in 1974 in a 14 year old girl by King and Mills [1, 2]. But closure of ASD by catheter techniques fell into disfavour until the mid Eighties, when the development of various closure systems was revived. Today, several systems are in clinical or investigational use in different countries: “Rashkind device” [3, 4], “CardioSeal device” [5–7], “Sideris buttoned occluder” (Fig. 1a, 1b) [8–15], “Angel-Wings device” (Fig. 1c) [16], “Monodisk device” [17], “ASDOS device” [18–20], “PFO-Star” (Fig. 1d) and “Amplatzer device” (Fig. 1e) [21–27].

The purpose of the present article is to review the present status of nonsurgical closure of ASD and patent foramen ovale (PFO).

Atrial Septal Defect

Incidence

An ASD is the most frequent congenital heart defect (10 %). The incidence in women is three times higher than in men.

Among the various ASD types, the ostium secundum defect (secundum type ASD), located in the area of the fossa ovalis, is the most common (70 %) isolated inter-atrial connection [28].

Pathophysiology and clinical symptomatology

The severity of an ASD depends on its size, the diastolic function of both ventricles and the resistance of the pulmonary and systemic circulation. Left-to-right shunt at the atrial level may result in volume overload of the right ventricle and increased pulmonary blood flow. Long-term volume overload of the pulmonary vascular bed results in increased pulmonary vascular resistance and subsequently pulmonary hypertension.

Most patients remain asymptomatic until the age of 20 or 30 years. Beyond the age of 30 to 40, many patients develop atrial fibrillation, heart failure, pulmonary arterial hypertension or even the Eisenmenger-Syndrom with peripheral cyanosis and risk of paradoxical embolism. Without shunt closure the average life expectancy of symptomatic patients amounts to 35–40 years [28–31].

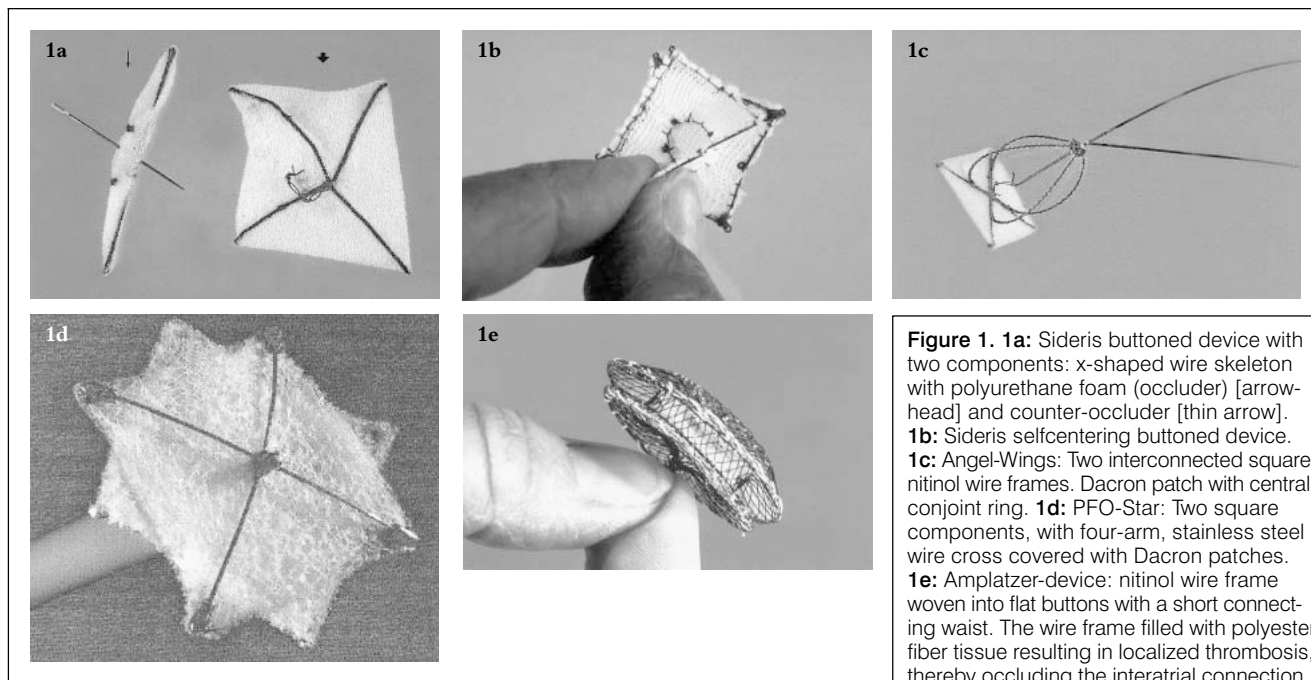


Figure 1. 1a: Sideris buttoned device with two components: x-shaped wire skeleton with polyurethane foam (occluder) [arrow-head] and counter-occluder [thin arrow]. 1b: Sideris selfcentering buttoned device. 1c: Angel-Wings: Two interconnected square nitinol wire frames. Dacron patch with central conjoint ring. 1d: PFO-Star: Two square components, with four-arm, stainless steel wire cross covered with Dacron patches. 1e: Amplatzer-device: nitinol wire frame woven into flat buttons with a short connecting waist. The wire frame filled with polyester fiber tissue resulting in localized thrombosis, thereby occluding the interatrial connection.

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Therapy

Closure of secundum type ASD is recommended in the presence of symptoms and a relevant left-to-right shunt ($Q_p:Q_s > 1.5$). Whether a small ASD should be corrected is controversial. While mortality of surgical ASD closure is very low ($< 1\%$), thoracotomy results in considerable discomfort, morbidity and unsatisfactory cosmetic results. King and Mills successfully implanted a Dacron double device into an ASD via a femoral venous access through a 23 Fr sheath (8 mm diameter) [1].

Despite good initial results, nonsurgical closure of secundum type ASDs was not further pursued for a considerable time. However follow up of the five initially treated patients, showed good results up to 10 years after device closure [32]. In 1983, Rashkind reported on the successful implantation of a self-developed occluder, which was implanted through a 16 Fr sheath (5 mm diameter) [3]. In the following years, the "hooked" device by Rashkind was modified, first to a Rashkind "double umbrella", then to a "lock-clamshell" device, and finally to the CardioSeal device, which can be delivered to the ASD site through a 11 Fr sheath (4 mm diameter) [33]. Sideris developed a "buttoned occluder" which can be delivered through an 8–11 Fr sheath (3–4 mm diameter). The latter system clearly represents an advantage especially in children, in which large sheath sizes cause vascular problems [8–11]. In the largest study so far, a "Sideris buttoned device" (Fig. 1a) or a "selfcentering buttoned device" (Fig. 1b) were implanted in 180 patients (predominantly children) for ASD-closure. In 160 of 180 patients (89%), an effective closure was obtained, defined as residual shunt in transthoracic colour Doppler echocardiography of less than 1 min jet diameter and disappearance of right ventricular volume overload [13].

We implanted a total of 34 devices in 35 patients presenting with a secundum type ASD. The following devices were utilized: Sideris buttoned device ($n = 20$), Sideris selfcentering device ($n = 6$), Angel-Wings ($n = 3$), Amplatzer septal occluder ($n = 5$) (Fig. 1) [33]. In three patients, the implantation of a device was not possible because of an oversized ASD and missing atrial septal rim (defect diameter 30, 32 and 34 mm). In two patients, a second device was implanted in a second session, because of a significant residual shunt. In twelve patients, the ASD could be effectively closed. In nine patients, there was a residual shunt < 5 mm, while in eleven patients a residual shunt > 5 mm was still present several months after the procedure (Fig. 2). In nearly all patients clinical symptoms had improved at follow-up (Tab. 1). Compared to the study of Rao et al. [13] our results were less encouraging. The main reason may be the fact, that our study included predominantly large ASDs (including patients with ASD size > 25 mm), which are less likely to be suited for percutaneous ASD closure (Tab. 2). According to our and other experience, the defect size should be not larger than 20 mm at echocardiography or 25 mm during invasive balloon sizing for successful implantation of currently available devices. In larger defects, the success rate

is considerably lower because of insufficient anchoring of the device on the atrial septal rims [14, 34]. Rao and coworkers have shown that a residual shunt may continuously decrease until twelve months after closure [13]. If there is a significant residual shunt after implantation, an attempt to reduce the remaining defect by implantation of a second device may be considered [35].

Depending on the closure system various complications may occur mostly during or immediately after implantation. Fractures of device arms have been reported [13–16]. Device embolization during implantation occur in 3–6%. Atrial perforation with tamponade is rare, as is thrombotic material adherent to the device surface ($< 1\%$) [13–16, 36–41]. In studies with the percutaneous device closure of ASD the mortality rate was 0% [13, 15, 39]. In our experience, embolization of the buttoned device into the right ventricle was followed by catheter perforation of the free right ventricular wall, which occurred in a 70 year old patient with a large ASD. The patient died after repeated surgical interventions for bleeding complications. In another patient, transoesophageal echocardiography revealed a thrombus in the left atrium at follow-up, which was treated successfully by oral anticoagulation. Low-dose antiplatelet therapy (eg, aspirin) is recommended for six months. After this time the device surface is covered by endothelium. We recommend endocarditis prophylaxis for six months until additional data are available [15].

Based on these results, transcatheter closure of secundum type ASD with the various closure systems available represents a less invasive alternative to surgical closure in selected patients. Transoesophageal echocardiography prior to the intervention aids in selecting patients suited for transcatheter closure and allows a specific device for individual defects to be selected.

Patent Foramen Ovale

Incidence

A patent foramen ovale (PFO) is present in 20–30% of the normal population, independent of sex [42].

Pathophysiology and clinical symptomatology

The relation between PFO and paradoxical embolism was first described by Julius Cohnheim in 1877 [43]. Venous thrombosis as a trigger of paradoxical systemic embolism frequently presents with no or minimal clinical signs, often escapes phlebological diagnostics, or has resolved, by the time the embolism occurs and diagnostic tests are performed [44, 45]. A direct proof of thrombus in the PFO can rarely be

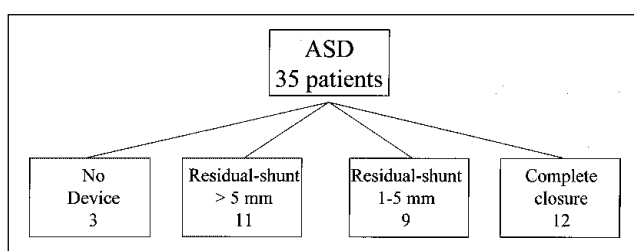


Figure 2. Success rate of atrial septal defect closure

Table 1. Clinical and echocardiographic variables before and after closure of secundum type atrial septum defect

	Before closure	After closure
NYHA-classification	2.7 $\pm$ 0.5	1.3 $\pm$ 0.5
ASD-diameter (echocardiography)	17 $\pm$ 5 mm	4 $\pm$ 3 mm

NYHA: New York Heart Association; ASD: atrial septal defect

Table 2. Success rate of ASD closure according to pre-interventional atrial septum defect size

	Number	ASD size*
No device	3	32 (30–34) mm
Residual-shunt > 5 mm	11	25 (15–33) mm
Residual-shunt < 5 mm	9	21 (10–25) mm
Complete closure	12	17 (08–28) mm

* ASD size in mean and (range)

found *post mortem* [46–50]. A small number of case studies, however, have documented a thrombus lodged in the PFO at transoesophageal echocardiography [51–68].

Atrial fibrillation, rheumatic and degenerative valvular heart diseases and left ventricular aneurysm are the classic potential cardiac sources for systemic embolism, in addition to plaques of the ascending aorta, the aortic arch and the cerebral arteries. In patients with cerebral embolism presenting with neither a cerebrovascular nor a cardiac source of embolism, the prevalence of a PFO is increased as compared to the normal population. The presence of a PFO is associated with a four times higher risk of cerebral embolism than in a population without PFO. In the presence of a concomitant atrial septal aneurysm the risk is increased 30-fold [53, 69, 70]. In a study published by Knauth and coworkers it has been shown that the presence of a large PFO in divers was related to significantly more frequent paradoxical cerebral gas embolization by gas bubbles [71]. These results confirm the notion that a right-to-left shunt mediated through a PFO may lead to paradoxical embolization with ischaemic brain damage. A paradoxical embolism implies a pressure reversal on the atrial level as is typical at the end of a Valsalva maneuver, resulting in a volume overload in the right atrium and consecutively a right atrial pressure rise over the left atrial pressure. A right-to-left shunt through an open PFO also occurs while coughing or during continuous increase of the right atrial pressure such as in chronic obstructive lung disease and after pulmonary embolism. The shunt volume depends on PFO size, duration of atrial systole and the pressure difference between the two atria.

Left-to-right shunting is usually prevented by complete closure of the PFO while pressure is higher in the left compared to the right atrium. In large atria, however, left-to-right shunting can also occur, as has been shown during transoesophageal echocardiography. Although generally negligible, right-to-left shunt may cause serious ventilation problems under certain conditions, such as in patients with major pulmonary embolism.

Therapy

An accidental diagnosis of a PFO or atrial septum aneurysm during echocardiography remains without therapeutic consequences. After a cerebrovascular embolic event, the existence of a PFO in the absence of other sources of embolism generally results in the institution of oral anticoagulation or antiplatelet therapy. There is no general consensus on which of these therapies is preferable. Additionally, no data exist on the success rate and duration of such drug therapy. In this ambiguous therapeutic situation, transcatheter closure of PFO represents a valuable alternative to surgical closure or medical therapy. The closure of a PFO is performed with similar devices as for ASD. At best, the possible source of embolism is eliminated, when an additionally present atrial septal aneurysm is splinted by the device [72].

Favourable primary results with complete PFO closure have been shown in 28 of 36 patients (78 %) [73, 74]. During an average follow-up of three years, in 97 % of the 36 treated patients there was no recurrent embolism. We treated 45 patients with a history of presumed paradoxical embolism, 41 suffered from cerebral, and 4 patients from peripheral embolism. The significance of the PFO was quantified by the number of microbubbles in the left atrium on a still frame image during transoesophageal contrast echocardiography ("Haemacel") with Valsalva-maneuver [15, 75, 76]. Complete closure was observed in 30 of 44 patients (68 %) (Tab. 3). During 708 patient-months, a second cerebral event occurred in 2 patients. In one of them, the PFO was subsequently closed surgically although no residual shunt had been

Table 3. Success rate of patent foramen ovale closure measured by contrast transoesophageal echocardiography

PFO shunt grade	Before implantation (n = 45)	After implantation (n = 44)
0	0	30
I	0	7
II	2	2
III	42	5

PFO-grading by transoesophageal contrast echocardiography: Grade 0: complete closure; Grade I: 1–5 blubbles into the left atrium in a still frame image; Grade II: 6–25 bubbles; Grade III: > 25 bubbles

found by TEE. A few months later a third cerebral embolism occurred. Indeed, in this patient, the initial theory of a paradoxical embolism was incorrect. A recurrent transient ischaemic attack occurred 4 weeks after PFO closure in a female patient. At echocardiography, a residual shunt in the device area was found and closed by a second device [77].

As in ASD, platelet inhibition for six months is recommended. For safety reasons, an endocarditis prophylaxis should be performed for six months [15].

Although randomized studies are not yet available, the present results appear promising with respect to secondary prevention. Indeed, percutaneous device closure represents an alternative to life long oral anticoagulation or open heart surgery.

Summary

Current devices permit transcatheter closure of secundum type atrial septal defect and of patent foramen ovale. The intervention is performed via a transvenous access under local anaesthesia. Most operators use peri-interventional transoesophageal echocardiography, but this is not obligatory for PFOs or small ASDs. The indication of secundum type atrial septal defect closure differs from that of a patent foramen ovale: In the former, left-to-right shunt volume has to be reduced primarily, while in the latter, a haemodynamically right-to-left reverse shunt as a source of embolism has to be eliminated. First results for both indications are promising. Further technical development is within reach and should take into consideration the different intents of application. Studies have to be conducted comparing the atrial septal defect-closure with surgical results. It is, however, likely that an increasing number of haemodynamically borderline defects will also be treated by catheterization technique because of the less severe stress for patients (and lower costs) compared to surgery.

Randomized studies are needed to compare transcatheter closure of patent foramen ovale to anticoagulation (or surgery). Even if the recurrence rate of embolism were identical, transcatheter closure would be preferable because of the annual risk of bleeding complications of 2–3 % per year, and the long-term financial expenses.

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