Original article

# Percutaneous closure of post-traumatic and congenital muscular ventricular septal defects with the Amplatzer Muscular VSD Occluder

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**Abstract**

**Background:** Muscular ventricular septal defects (VSD) are an important and difficult surgical problem. In the last few years a new alternative has emerged – possibility of VSD closure using percutaneous approach.

**Aim:** To present our experience in percutaneous closure of congenital muscular and one posttraumatic VSD.

**Methods:** We treated 10 patients – 7 children (age 0.8-7 years) and 2 adults (43 and 46 years) with congenital VSD, and one 18-year-old patient with posttraumatic VSD (knife stab). All the patients had a large haemodynamic shunt (Qp:Qs 1.9) and in all cases percutaneous closure attempt with an Amplatzer Muscular VSD Occluder (MVSDO) implant was undertaken. Five of 6 children with multiple muscular VSDs had in infancy previous pulmonary artery banding and one patient had complex heart disease: transposition of great arteries (dTGA), pulmonary stenosis (PS) and perimembranous VSD. All procedures were performed using the standard technique.

**Results:** Eleven procedures were performed in 10 patients (one child had 2 attempts). Seven procedures were successful. In all cases a considerable reduction in flow or complete closure was achived. In one case, despite multiple attempts, VSD caniulation was ineffective and the procedure was abandoned. The patient had oblique VSD – morphology confirmed was later by the operating surgeon. The reason for the other 3 failures was early embolisation to the left ventricle and aorta. This complication was noted in 2 adult patients – one with congenital and one with post-traumatic VSD. In both cases the interventricular septum was thick (10 and 11 mm) and implants were removed with a bioptome or vascular lasso. Another embolisation occurred in a child with TGA – in this case the cardiac surgeon removed the implant from the aortic arch during Rastelli operation.

**Conclusion:** Our experience acquired during muscular VSD closure with MVSDO indicates that the method is useful in children with isolated defects. Adult patients and children with a complex form of congenital defects should have morphology of MVSDO carefully evaluated and width of the interventricular septum measured to avoid potential implant embolisation.

**Key words:** muscular septal defects, interventional cardiology

Kardiol Pol 2008; 66: 715-720

## Introduction

Ventricular septal defect (VSD) is the most common congenital cardiac malformation. It may be located in the muscular (15%), perimembranous (approx. 70%) or supracristal (so-called subarterial VSD; a rare type in the European population) part of the ventricular septum. Sometimes the defects are multiple.

Ventricular septal defects located in its muscular part often tend to close spontaneously. On the other hand, this type of congenital ventricular septum malformation is considered a real challenge for the cardiac surgeon because it is usually difficult to visualise intraoperatively from the right side of the septum with many muscular trabeculae. Unfortunately, it can lead to surgical failure (failed or partial, incomplete closure). Banding of the pulmonary artery is usually carried out in the infancy to prevent development of pulmonary hypertension and to facilitate spontaneous closure of muscular VSD. It also enables definite surgical correction to be postponed.

Post-traumatic VSD is a very rare defect and only sporadic cases are reported in the medical literature.

Procedures of percutaneous VSD closure using many types of device have been attempted since 1987 [1]. However, only with the introduction of the Amplatzer plugs did outcomes become satisfactory [2].

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**Received:** 28 February 2008. **Accepted:** 23 April 2008.

In this report, our own experience with percutaneous occlusion of muscular VSD, either congenital or post-traumatic, carried out in children and in adults, is presented.

## Methods

### Patients

Data on the examined patients are presented in Table I. Eleven procedures were performed in 10 patients. This group involved 7 children aged 0.8-7 years, and 3 adult subjects aged 18, 43 and 46 years, respectively. Congenital muscular VSD type was diagnosed in 9 patients and posttraumatic defect in one 18-year-old patient. In the latter case, a defect in the ventricular septum with significant shunt was caused by a violent knife slash. Pericardial tamponade and injury of the right ventricular free wall were treated successfully on an emergent basis in the regional hospital and then the patient was transferred to the tertiary cardiovascular centre (the procedure was done in this case by M. Szkutnik and T. Przewłocki). In patients with congenital VSD, defects were located in the middle or apical part of the muscular ventricular septum but in the subject with posttraumatic VSD the defect was located in the middle portion and had a slightly oblique course. There were multiple defects in 6 cases and a single one in 4 subjects. Five children (patients 1, 2, 7, 8, 10 in Table I) underwent pulmonary artery banding in the early infancy (at the age of 2 to 5 months). In three children (patients 1, 2 and 7 in Table I) prior to interventional VSD closure (performed between the 10$^{th}$ and 17$^{th}$ months of their life), pulmonary artery banding was removed surgically with subsequent closure of the perimembranous defect. Unfortunately, simultaneous attempts to close muscular defects failed. In one patient (case 4 in Table I) transposition of the great arteries (dTGA) and large outflow perimembranous defect accompanied by pulmonary artery stenosis were detected additionally to the apical muscular VSD. This patient was initially selected for Rastelli operation but due to additional apical VSD an attempt of percutaneous closure prior to surgical correction was undertaken. Detailed echocardiography (transthoracic – TTE) and intraprocedural transoesophageal examination (TEE) were performed in all patients before interventional catheterisation. Echocardiographic parameters, including cardiac chamber dimensions, VSD location, its diameter and ventricular septum thickness were evaluated. Only in one child (with dTGA – patient number 4) was assessment using a 24-mm calibrating balloon (AGA Med) done due to a low quality of echocardiographic visualisation of the defect diameter.

In all patients left ventricular (LV) and left atrial chambers were dilated. The latter was less pronounced in patients with previously placed surgical pulmonary

**Table I.** Demographic, haemodynamic and procedural data on the study patient

| No | Age [years] | BM [kg] | VSD | Qp/Qs | Access | Impl. | VSD /MVSDO | Fluoroscopy [min] | Result | Follow-up [years] | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1.5 | 9.0 | M | 1.5 | R | RFV | 5/6 | 38 | + | 5.6 | residual shunt |
| 2a | 2.7 | 10.6 | M | 2.1 | R | RJV | 9/10 | 33 | + | 0,5 | postprocedural shunt (haemodynamically significant) |
| 2b | 3.2 | 12.0 | M | 1.8 | R | RJV | 4/4 | 34 | + | 5,4 | residual shunt |
| 3 | 0.8 | 9.6 | S | 2.0 | R | RJV | 4/6 | 22 | + | 5.5b | successful occlusion |
| 4 | 7 | 19.0 | M | 2.5 | R | RJV | 6/12 | 20 | embolis | surg (op. m. Rastelli) | TGA, MVSD, pmVSD, PS |
| 5 | 43 | 89.0 | S | 1.5 | L | RFV | 7/8 | 31 | embolis | Refusal of surg | AH |
| 6 | 46 | 81.0 | S | 1.8 | L | RRV | 7/10 | 54 | + | 2.8 | successful occlusion |
| 7 | 1.5 | 8.4 | M | 2.0 | R | RJV | 4/6 | 51 | + | 2.4 | residual shunt |
| 8 | 1.2 | 10.2 | M | 2.1 | R | RJV | 5/6 | 17.4 | + | 0.3 | residual shunt |
| 9 | 18 | 70.0 | S | 1.8 | L | RFV | 6/8 | 25 | embolis | surg | VSD after trauma caused by knife |
| 10 | 1.7 | 11.2 | M | 1.9 | – | – | 6/– | 22 | – | surg | impossible cross over through VSD |

*Abbreviations: BM – body mass; VSD – ventricular septal defects, VSD type: S – single, M – multiple; access – access through ventricular septum: L – from the left side, R – from the right side; impl. – access of device introduction: RFV – right femoral vein, RJV – right internal jugular vein; MVSDO – Amplatzer Muscular VSD Occluder, embolis – early embolic complication to the systemic circulation (up to 24 hours following procedure); TGA – transposition of great arteries, pmVSD – perimembranous ventricular septal defect, residual shunt – haemodynamically non-significant; VSD/MVSDO – VSD diameter in TEE/diameter of Muscular Ventricular Septal Occluder (in mm); surg – surgical closure of VSD; op. m. Rastelli – surgical closure Rastelli method; PS – pulmonary stenosis*

artery banding. In children ventricular septum thickness never exceeded 7 mm and in adult patients was 11 mm, 8 mm and 10 mm, respectively (patients 5, 6 and 9 – Table I). In a 43-year-old man (number 5 in Table I) systemic hypertension requiring medical treatment was diagnosed.

### Procedure

Procedures performed in children were done in general anaesthesia and in adults only in local anaesthesia and with guidance of both TEE and fluoroscopy. After the jugular and/or femoral vein were punctured, the diagnostic part of catheterisation with pressure measurements and calculation of pulmonary to systemic flow rate (Qp/Qs) was carried out. Depending on muscular defect location an attempt to cross the ventricular septum, initially from the right ventricular side, was undertaken, then in the case of failure – from the left aspect of the ventricular septum. In the latter case an arterial-venous loop was used as the procedure was always performed from the right aspect of the ventricular septum via the venous system. An Amplatzer Muscular VSD Occluder (MVSDO) (AGA Medical Corp. Mn) was used and implanted according to the guidelines published elsewhere [3, 4]. This implant device consists of a nitinol net of spool-like shape that has a 7-mm connector and discs wider than 4 mm. Nitinol plugs – MVSDO – are available in sizes ranging from 4 to 18 mm (every 2 mm) and the sizing corresponds to the mean diameter of the connector part (Figure 1).

## Results

The procedures were successful in 7 out of 11 cases. In one patient (number 2 in Table I) with muscular 'Swiss cheese-like' type VSD, 2 procedures were performed (with a 5-month interval) (Figure 2). Prior to intervention, the mean pulmonary to systemic flow rate (Qp/Qs) was 1.9. Fluoroscopy time 32 minutes. In 3 patients presenting with multiple VSD (patients 1, 7 and 8 in Table I) the largest defects were occluded successfully (using a single implant). In all of those subjects, non-significant residual shunts through other small VSDs were detected. In all children, physical development and body mass growth were normal after the procedure, which combined with the findings of TTE examination indicated significant post-procedural shunt reduction. In one case of multiple and congenital VSD (number 10 in Table I), crossing with a guidewire through the ventricular septum and largest VSD failed in spite of many attempts undertaken from both the venous and arterial side. This defect had an oblique and tunnelled course and this finding was confirmed by the cardiac surgeon who operated on this child subsequently. The reason for three consecutive failures was early implant embolisation. It was seen in patients presented in Table I as numbers 4, 5 and 9 (two of them with congenital and one with post-traumatic VSD). In the first and third case the events took place a few minutes after implantation

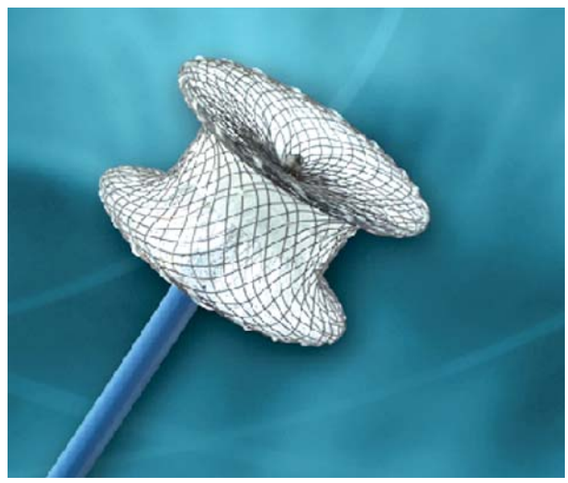

**Figure 1.** Amplatzer Muscular Ventricular Septal Occluder

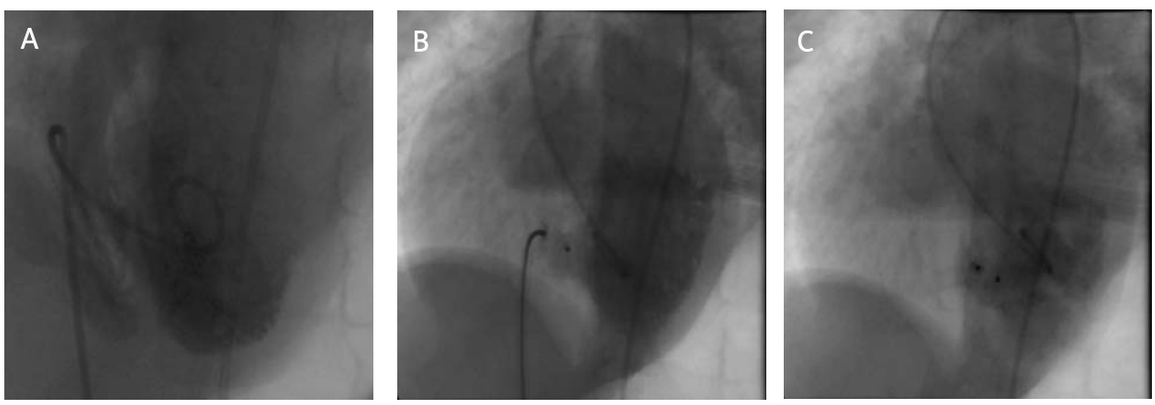


**Figure 2.** Stages of muscular portion of ventricular septum percutaneous closure with Amplatzer Muscular VSD Occluder. **A.** A pig tail catheter crossing VSD from the right ventricular aspect. **B.** MVSDO closing VSD but still connected to the delivery system. **C.** MVSDO released from the delivery system

(periprocedural), and in another one (number 5) on the day after the procedure. All embolic complications were limited to the systemic circulation. In one case (number 4) the cardiac surgeon removed the implant out of the aortic arch lumen during Rastelli operation performed on the same day (this patient had additional dTGA, perimembranous VSD and valvular pulmonary artery stenosis). In another two cases in adults, the implants were removed through the arterial approach using a bioptome or vascular lasso (Goosneck, Microvena).

## Discussion

In recent years Amplatzer implants have increasingly often been used and have gained wide recognition in occlusion of muscular VSD. First, they were used successfully in experimental animal models [5], then clinical application in children was introduced [3, 4, 6, 7]. Our experience supports high usefulness of this occluding device also when used to occlude perimembranous VSD with at least 4 mm wide aortic rim [8] and in selected cases to occlude postinfarction VSD [9]. The observations presented herein indicate generally that MVSDOs are useful to occlude congenital muscular VSD in children. However, some reflections regarding employment of this implant in adults have emerged.

In our series there were three cases of implant embolisation that require detailed discussion. Of note they were the only cases ever observed in our institution. We performed 49 procedures of percutaneous closure of various VSD types, including both postinfarction [10] and perimembranous ones [11]. The first case of embolic complication was a child with TGA, initially referred for Rastelli operation. In this child rotation of the ventricular septum was found that interfered with its visualisation and calculation of defect diameter. It prompted us to calibrate the defect using a balloon catheter (contrary to the usual setting when measurements made in echocardiography are sufficient). Although we used in this case an oversised occluding device (calculated defect 6 mm – used implant 12 mm), embolisation into the LV chamber and then the aortic lumen occurred. The cause of this complication is difficult to define, but it seems that it is related somehow to unusual ventricular anatomy. The implant was removed during a cardiac surgical procedure performed on the same day (earlier planned Rastelli operation).

It is of interest to establish the real cause of implant embolisation in 2 out of 3 adult patients (numbers 5 and 9). We suppose that it resulted from a discrepancy between device length and thickness of the ventricular septum. According to the MVSDO's manufacturer the device may be used in patients weighing 8 kg or more. The upper weight limit however has not been determined. It must be kept in mind that the length of the implant connector is designed to fit the mean thickness of the ventricular septum in children of 7 mm. It seems that in adult patients with a hicker ventricular septum (as was noted in two of our cases – one with congenital and the second with post--traumatic VSD type where this dimension was 10 and 11 mm, respectively) this implant should have not been employed due to high risk of embolisation. In the third adult the ventricular septum thickness was 8 mm and this procedure and the post-operative period were completely free from any adverse events. A better option in patients with a thicker ventricular septum would be to use an occluding device designed by the manufacturer to close postinfarction interventricular ruptures (with the length of 10 mm). Unfortunately, they are commercially available starting from 18 mm diameter, and thus are not suitable in the case presented in this study.

It is also of interest that all MVSDO embolic events involved the LV chamber (although in theory with higher LV pressure we could expect embolisation predominantly to the right ventricle). A reasonable explanation of this fact may be incomplete deployment of the right ventricular disc (resulting from the mentioned discrepancy between ventricular septum thickness and implant connector length) and consequent pushing out by the contracting ventricular septum.

Post-traumatic VSD may result from penetrating stab wounds (as in our case) or blunt trauma. To occlude such defects, Amplatzer devices designed for closure of patent arterial ducts (Duct Occluders), atrial septal defects and muscular postinfarction VSD were employed [12-15]. Our experience indicates that the use of MVSDO may not be an optimal solution due to the aforementioned reasons.

An intriguing alternative for transluminal closure of congenital and post-traumatic VSD are hybrid procedures performed in cooperation with cardiac surgeons at the time of surgery [16, 17].

## Conclusions

Our experience indicates that the occlusion of muscular VSDs using percutaneous access is safe and efficacious in children. In adult patients, particular attention must be paid to precise determination of the ventricular septum thickness to avoid potential complications such as embolic events.

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# Przezskórne zamykanie mięśniowych ubytków międzykomorowych – pourazowego i wrodzonych – z zastosowaniem korków Amplatzer Muscular VSD Occluder

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## Streszczenie

**Wstęp:** Ubytki w mięśniowej części przegrody międzykomorowej (VSD) stanowią dość trudny problem w leczeniu chirurgicznym. W ostatnich latach pojawiła się alternatywna możliwość ich zamykania metodami kardiologii interwencyjnej.

**Cel:** Prezentacja doświadczeń własnych w przezskórnym zamykaniu mięśniowych VSD – wrodzonych oraz jednego pourazowego.

**Metodyka:** W leczonej grupie było 10 chorych – 7 dzieci (w wieku 0,8–7 lat) i 2 dorosłych (43 i 46 lat) z wrodzonymi VSD oraz jeden 18-letni chory z pourazowym VSD (po pchnięciu nożem). U wszystkich stwierdzono istotny hemodynamicznie przeciek (średni Qp:Qs 1,9) i u wszystkich podjęto próbę przezskórnego zamknięcia ubytku za pomocą implantu Amplatzer Muscular VSD Occluder (MVSDO). Spośród 6 dzieci z mnogimi mięśniowymi VSD, u 5 w okresie wczesnoniemowlęcym założono przewiązkę na tętnicę płucną, a jedno dziecko miało złożoną wadę serca – przełożenie wielkich pni tętniczych (d-TGA) ze współistniejącym zwężeniem tętnicy płucnej i odpływowym okołobłoniastym VSD. Zabiegi wykonywano standardową techniką.

**Wyniki:** U 10 chorych wykonano 11 zabiegów (u jednego dziecka dwa zabiegi). Zabieg ukończono skutecznie w 7 przypadkach – u wszystkich obserwowano istotne ograniczenie przecieku bądź szczelne zamknięcie VSD. W jednym przypadku mimo wielokrotnych prób nie udało się przejść przez przegrodę międzykomorową (VSD) prowadnikiem ani od strony żylnej, ani od strony tętniczej. Ten ubytek miał skośny, tunelowaty przebieg, co potwierdził operujący później dziecko kardiochirurg. Przyczyną kolejnych trzech niepowodzeń była wczesna embolizacja implantu do lewej komory i dalej do aorty. Dotyczyło to dwóch dorosłych – jednego z wrodzonym i jednego z pourazowym VSD. W obu przypadkach przegroda międzykomorowa była pogrubiała (odpowiednio 10 i 11 mm), a implant usunięto przeznaczyniowo za pomocą bioptomu lub lassa naczyniowego. Kolejna embolizacja wystąpiła u dziecka z TGA – w tym przypadku kardiochirurg usunął implant z łuku aorty podczas operacji Rastellego.

**Wnioski:** Uzyskane doświadczenia wskazują, że przezskórne zamykanie mięśniowych VSD za pomocą MVSDO jest użyteczną metodą leczenia dzieci z izolowaną postacią wady. U osób dorosłych oraz dzieci ze złożonymi wadami i współistniejącym VSD należy zachować szczególną ostrożność, określając morfologię i grubość przegrody międzykomorowej, aby uniknąć embolizacji implantu.

**Słowa kluczowe:** mięśniowe ubytki międzykomorowe, kardiologia inwazyjna

Kardiol Pol 2008; 66: 715-720

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**Praca wpłynęła:** 28.02.2008. **Zaakceptowana do druku:** 23.04.2008.