Percutaneous Closure of Ventricular Septal Defect With an Amplatzer Device

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We present a series of 4 patients in whom a ventricular septal defect (VSD) was closed with an Amplatzer muscular VSD device during cardiac catheterization. In one patient with type I truncus arteriosus and subarterial VSD, closure of a wide apical defect was done to allow further surgical correction while avoiding left ventricular ventriculotomy. The second patient had congenitally corrected transposition of the great arteries with complete A-V block and a pacemaker implanted from birth. Our intention was unload the systemic anatomical right ventricle. The third patient had had 3 heart surgeries to correct a double outlet right ventricle. She had a residual ventricular septal defect which was large enough to cause heart failure. The last patient had an isolated muscular ventricular septal defect. Percutaneous closure was successful in all patients, and there were no complications. Percutaneous closure of the defect may be used as a primary procedure, before surgery, or as a complementary procedure after surgery.

Key words: *Congenital heart disease. Catheterization. Ventricular septal defect. Transposition of great arteries.* Truncus arteriosus.

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Cierre percutáneo de la comunicación interventricular con dispositivo Amplatzer

Presentamos nuestra experiencia en el cierre percutáneo de la comunicación interventricular (CIV) con dispositivo Amplatzer muscular en 4 pacientes. El primer caso se trataba del cierre de una CIV apical amplia, adicional a la CIV subarterial de un tronco arterioso tipo I, con el objetivo de evitar la ventriculotomía apical en la corrección quirúrgica completa posterior. En el segundo caso, la CIV estaba asociada a una transposición de grandes arterias congénitamente corregida con dextrocardia, *situs inversus* y bloqueo auriculoventricular, portador de marcapasos desde el nacimiento. La CIV se cerró con el fin de descargar al ventrículo derecho (sistémico). En el tercer caso, la CIV era posquirúrgica en un paciente con ventrículo derecho de doble salida en el que con anterioridad se habían realizado 3 intervenciones quirúrgicas. El último paciente tenía una CIV muscular aislada. El cierre fue completo en todos los casos. No hubo complicaciones. El cierre percutáneo de la CIV puede hacerse como procedimiento de elección primaria, previo a la cirugía o como complemento de ésta.

Palabras clave: *Cardiopatías congénitas. Cateterismo cardíaco. Comunicación interventricular. Transposición de grandes arterias.* Truncus arteriosus*.

INTRODUCTION

Ventricular septal defect (VSD) alone or as part of a more complex lesion is seen in 20% of all children with heart defects.[1] Most VSD that occur in isolation close spontaneously. A few cases require treatment with diuretics, digoxin or angiotensin-converting enzyme (ACE) inhibitors, and only a minority require surgical treatment with extracorporeal circulation. In 1988 O'Laughlin and Mullins published the first report of percutaneous closure with a Bard double-umbrella device.[2] The few cases reported since then have involved a variety of devices such as the clamshell,[3] Sideris,[4] and more recently, the new Amplatzer device (AGA Medical Corporation, Golden Valley, MN, USA).[5-8] In Spain this device has also been used to close VSD in patients with myocardial infarction.[9] The safety and efficacy of the device has attracted attention because of its potential for treating VSD, even in very young patients, although this technique is currently recommended for children weighing more than 7 kg.

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CASE STUDIES

Patients

Table 1 summarizes the general characteristics of our patients. Mean age was 11.25 years (range, 0.6-26 years) and mean body weight was 30.9 kg (range, 7.2-57 kg).

Case 1

This young girl had type I truncus arteriosus (TA-I) with a subarterial VSD and a muscular apical VSD measuring 9 mm. At 28 days of age she was treated with pulmonary artery banding for heart failure. At 6 months of age, to avoid extensive right ventriculotomy or left apical ventriculotomy in the course of corrective surgery, she underwent percutaneous closure of the apical VSD with an Amplatzer device (AD) to occlude the 6-mm VSD. The device was implanted via a transjugular approach (Figure 1). Echocardiographic control confirmed that the AD was correctly deployed (Figure 2A). After percutaneous closure of the VSD, clinical improvement was seen, and 1 month later surgical repair of the truncus arteriosus was performed with a bioprosthesis.

Case 2

This boy had a prenatal diagnosis of dextrocardia, situs inversus, congenitally corrected transposition of the great arteries (L-TGA) and complete heart block with a ventricular pulse rate of 60 beats/min during gestation. During the neonatal period a perimembranous VSD was discovered. At 2 weeks of age a permanent internal, dual-chamber pacemaker was implanted. During clinical follow-up in the ensuing years it was judged that the perimembranous septal VSD was small, although during infancy pulmonary overcirculation had increased. Mild to moderate tricuspid valve failure developed (systemic atrioventricular valve), and the boy was treated with ACE inhibitors and diuretics. When the boy was 8 years old it was decided to close the VSD with a 6-mm AD to avoid right (systemic) ventricular volume overload as much as possible, and to indirectly treat tricuspid insufficiency in the context of an anatomically complex heart defect. After percutaneous closure the patient´s course has been satisfactory, and diuretics have been stopped.

Case 3

This girl had double outlet right ventricle (DORV) that required creation of a Blalock-Taussig fistula during the neonatal period because of valvular and infundibular pulmonary stenosis. When she was 2 years old complete correction was performed in another country at the parents' request. The VSD was closed, the right ventricular outflow tract was enlarged, and pulmonary valvulotomy was performed with a transannular patch. After surgery a small, residual VSD was detected adjoining the patch, with 2 small VSD in the ventricular apex and an infundibular gradient of 45 mm Hg. At 5 years of age the infundibular gradient had increased to 80 mm Hg and a residual VSD was confirmed, but considered to be small.

When the girl was 7 years old the same surgical team performed infundibulectomy of the right ventricular outflow tract without closing the VSD from the initial operation because they considered it to be small. After this operation the patient showed signs of biventricular heart failure and was treated with diuretics, digoxin and vasodilators. Six months later, when her Qp/Qs ratio was 2.9, the residual VSD was closed with percutaneous implantation of a 10-mm AD. After

TABLE. **General Characteristics***

Case	Age, years	Sex	Weight, kg	Diagnosis	Qp/Qs	Size of the AD, mm	Type of Closure
1	0.5	F	7.2	TA-I+apical VSD+subarterial VSD	2.5	6	Presurgery
2	9.7	M	37.4	L-TGA+pm VSD+pacemaker	1.8	6	Primary
3	8.7	F	22	DORV+residual VSD	2.9	10	Postsurgery
4	26	M	57	Muscular VSD	1.93	8	Primary

*F indicates female; M, male; TA-I, type I truncus arteriosus; pm VSD, perimembranous ventricular septal defect; AD, Amplatzer device for VSD; DORV, double outlet right ventricle; L-TGA, congenitally corrected transposition of the great arteries.
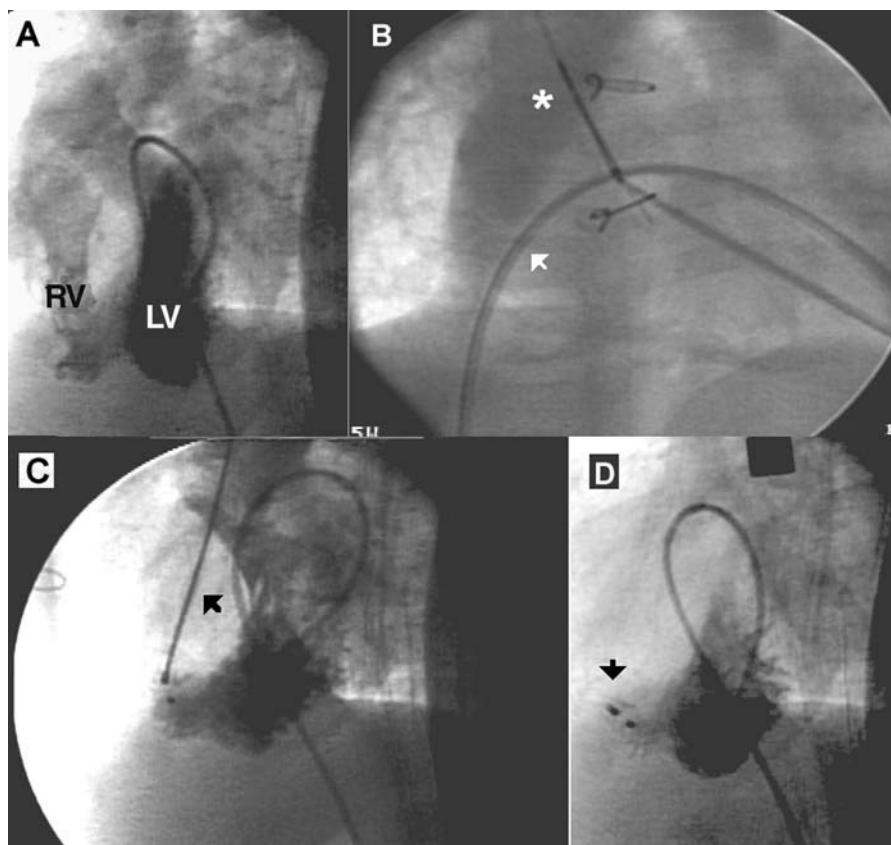
Fig. 1. Case 1. A: left ventriculogram. B: guidewire (arrow) introduced via the femoral vein, snared by the lasso catheter (asterisk) introduced via the right internal jugular vein. C: left ventriculogram showing ventricular septal defect (VSD) occluded by the Amplatzer device, still attached to the delivery catheter (arrow). D: Amplatzer device (arrow) after release, completely occluding the VSD. RV indicates right ventricle; LV, left ventricle.

closure clinical improvement was seen, and medication was suspended.

Case 4

This adult had a muscular VSD in the trabecular septum and was in functional class II, with cardiomegaly, a cardiothoracic index of 63% and pulmonary overcirculation on chest x-ray. The Qp/Qs ratio was 1.93. After closure of the VSD with an 8-mm AD the patient reported feeling better physically.

Technique

In all patients correction of the VSD was undertaken after informed consent was obtained. The procedures were done under general anesthesia. A transesophageal echocardiogram was obtained before therapeutic catheterization, and the probe was left in place for echocardiographic control during the procedure. In all four patients we used an AD for muscular VSD. In general, we used techniques described previously to close muscular[6] and perimembranous VSD,[7] with the following modifications:

– The diameter of the VSD was measured with

transesophageal echocardiography and angiography instead of with the balloon method.

– Case 1: to avoid creating an arteriovenous circuit we modified the technique to create a venovenous circuit. The catheter was advanced via an anterograde approach from the femoral vein through the foramen ovale to the left ventricle. The guidewire was then advanced through the VSD to the right ventricle and then to the right atrium. The guidewire was snared and pulled through with a lasso catheter introduced through the right jugular vein to complete the venovenous circuit. A 180° 6 Fr sheath was inserted via the right jugular vein to implant the 6-mm AD.

– Case 2: an exchange guidewire was advanced from the left ventricle through the perimembranous VSD to the right pulmonary artery, where it was captured with a lasso catheter advanced from the femoral vein (Figure 3). A Mullins sheath was inserted via the femoral vein, and the tip was left in the ascending aorta. The sheath was then withdrawn slowly and cautiously to open the first disk in the left ventricular outflow tract after the aortic valve had been passed, and the disk was placed snugly against the VSD.

– Cases 3 and 4: once the arteriovenous circuit had been established with guidewires via the jugular vein
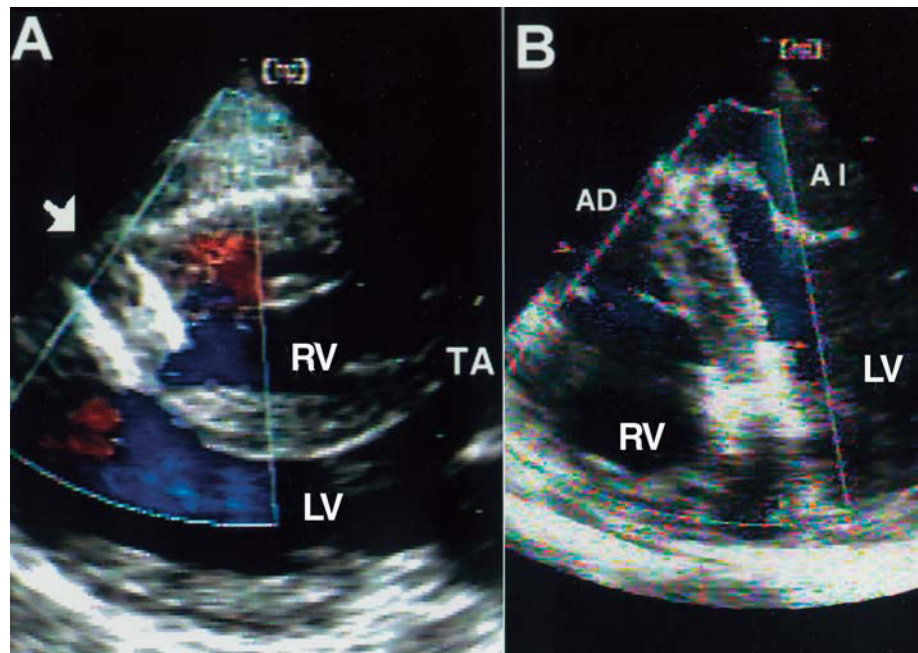
Fig. 2. Case 1. A: transthoracic echocardiograph (parasternal long axis view). Note the Amplatzer device about to be released, occluding the muscular apical ventricular septal defect (VSD). Note the free wall of the hypertrophied right ventricle, the delivery catheter in the right ventricular cavity, and the subaortic VSD adjacent to the truncus arteriosus (TA). B: 4-chamber transesophageal echocardiograph showing the Amplatzer device occluding the muscular apical VSD (case 4).

and femoral artery, the Mullins sheath was advanced to the left ventricular chamber with the tip pointing toward the left ventricular outflow tract, where the first disk of the AD was opened. The AD and sheath were then withdrawn until the other disk could be opened against the septal wall of the VSD.

Intravenous heparin (100 U/kg) was used as an anticoagulant during catheterization in all patients, and aspirin was given at 5 mg/kg per day during 6 months. Three doses of cefazolin were given: 1 during the procedure and 2 more at 8-hour intervals thereafter. Follow-up lasted for a mean of 7 months and was managed on an outpatient basis. Physical examination and echocardiographic studies (Figure 2B) failed to disclose evidence of residual VSD. We observed no complications such as aortic insufficiency, new valvular lesions or worsening or pre-existing lesions, hemolysis, systemic embolism or infective endocarditis.
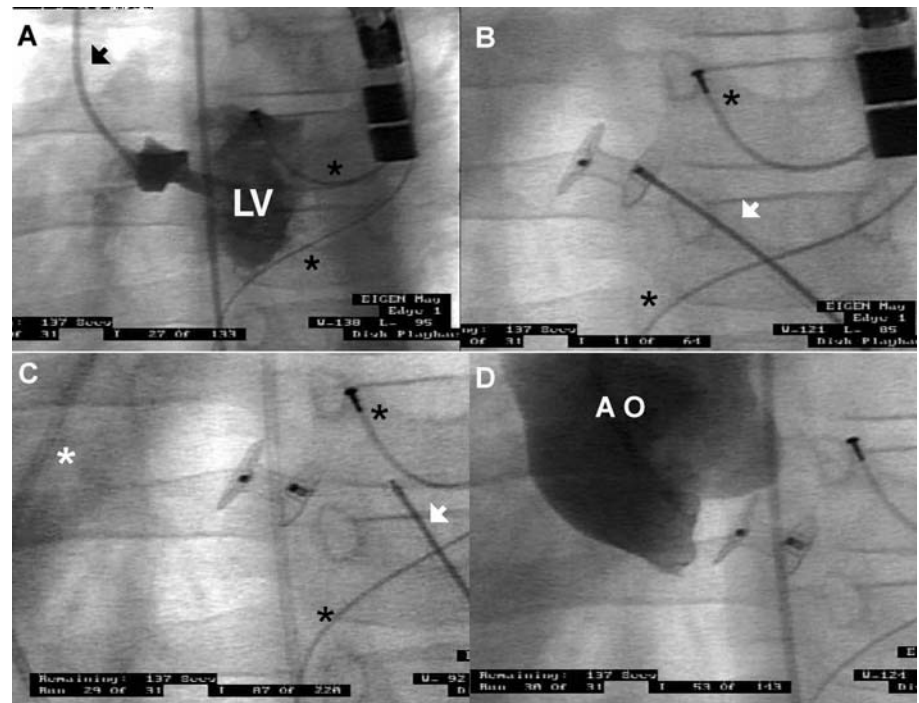
Fig. 3. Case 2. A: angiogram showing the ventricular septal defect (VSD) from the anatomical right ventricle, and the catheter (arrow) introduced via the aorta (AO). The dual-chamber pacemaker electrodes are marked with black asterisks.
B: Amplatzer device deployed in the VSD. C: device in place after release from the delivery catheter (arrow). D: aortogram showing competence of the aortic valve.

DISCUSSION

Perfecting a percutaneous technique to close a VSD with an AD was seen as a qualitative measure representing a logical step in the training of interventional cardiology teams already experienced in closing ostium secundum atrial septal defects. Although closure of VSD is similar to closure of atrial septal defects in conceptual terms, the 2 techniques differ in several important aspects, e.g.: *a)* the difficulty in locating and determining the size of the VSD, and *b)* the need to establish an arteriovenous circuit with the exchange guidewire which makes it possible to advance the guiding catheter safely to the site where the device is to be implanted. Two basic strategies are needed depending on the anatomical location of the VSD. Apical VSD or defects in the trabecular septum are accessed via the aorta through the left ventricle, and once the catheter is in the right ventricle the guidewire is allowed to float freely so that it can be captured with the lasso catheter advanced to the pulmonary artery from the jugular vein. However, for perimembranous septum VSD the arteriovenous circuit is created from the aorta and left ventricle, crossing the VSD toward the right ventricle, and the exchange guidewire is snared in the pulmonary artery and exteriorized through the femoral vein. In case 1 we modified this technique and established a venovenous circuit because in this young girl, we aimed to avoid using a 6 Fr Mullins sheath in the femoral artery. Access was possible via the femoral vein because of the presence of a patent foramen ovale. Crossing the VSD is often easier than expected, and capturing the exchange guidewire with the lasso catheter should be accomplished with as little trauma as possible. We recommend capturing the anterograde guidewire with the lasso catheter in the pulmonary artery, but it can also be captured from the right atrium, as was done in the patient with TA-I.

Sizing the VSD with a balloon catheter is an additional maneuver that may move the guidewire out of position without providing much useful information. We prefer to match the device to the diameter measured by echocardiography or ventriculography to achieve the best possible fit between the device and the diameter of the VSD.

Once the guiding catheter has crossed the VSD, the tip should be advanced as distally as possible to keep the catheter from slipping out of place in the left ventricle while the device is being maneuvered into position. It has been recommended[7] that in perimembranous septal VSD, the guiding catheter should be placed in the apex of the left ventricle to ensure that the device opens correctly and to avoid trapping the aortic valve with the disk that opens in the left ventricle. However, we have noted that this maneuver can also be performed differently, by positioning the guiding catheter in the aorta and withdrawing it slowly to the left ventricular outflow tract under transesophageal echocardiographic guidance. For apical VSD and defects in the trabecular septum, in contrast, the tip of the guiding catheter should be located in the left ventricle as distally as possible to the VSD.

In 2 of 4 patients the procedure was done as a complementary measure to surgical treatment. In one case the presence of two VSD made it advisable to perform pulmonary artery banding first, and then to deploy the device to close the apical VSD. This avoided the need for left apical ventriculotomy, which would have been needed in addition to complete reparative surgery for TA-I and the subarterial VSD. In case 3, we avoided a fourth heart surgery.

Primary closure in case 2, with a Qp/Qs ratio of 1.8, was chosen because in this patient the L-TGA had begun to cause right (systemic) ventricular failure, manifested as tricuspid valve insufficiency.

The patient described as case 4 had enjoyed a normal life except for intolerance of strenuous effort. There were radiological signs of pulmonary overcirculation, and his Qp/Qs ratio was 1.93. After percutaneous closure the patient´s clinical condition improved, as did his ability to tolerate strenuous exercise. Although this is only a single case, percutaneous closure may be indicated when spontaneous closure of the VSD is unlikely (e.g., in adolescents and adults). One of our patients had complete atrioventricular heart block and had needed a permanent pacemaker since infancy. No conduction defects were observed in the other patients after the AD was implanted, although transitory atrioventricular block has been seen[8] in 2.8% of the patients, and complete block has been reported in 1.9%.

Only one of our patients had a perimembranous VSD, and we saw no signs of interference with the aortic valve. This complication has been reported with other devices,[1] although it has not been seen with the Amplatzer device.

COMMENTS

Although our initial experience is still limited, we found the primary closure of the VSD, if done with care, led to no complications. Closure of the VSD can be used for patients in whom the high risk associated with further surgery should be avoided. In other cases the risks associated with complex surgical procedures can be reduced. In patients with few symptoms in whom spontaneous closure of the VSD is unlikely, percutaneous closure may become a useful alternative.

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